2014: The Year of Change, p. 22

January 15, 2014

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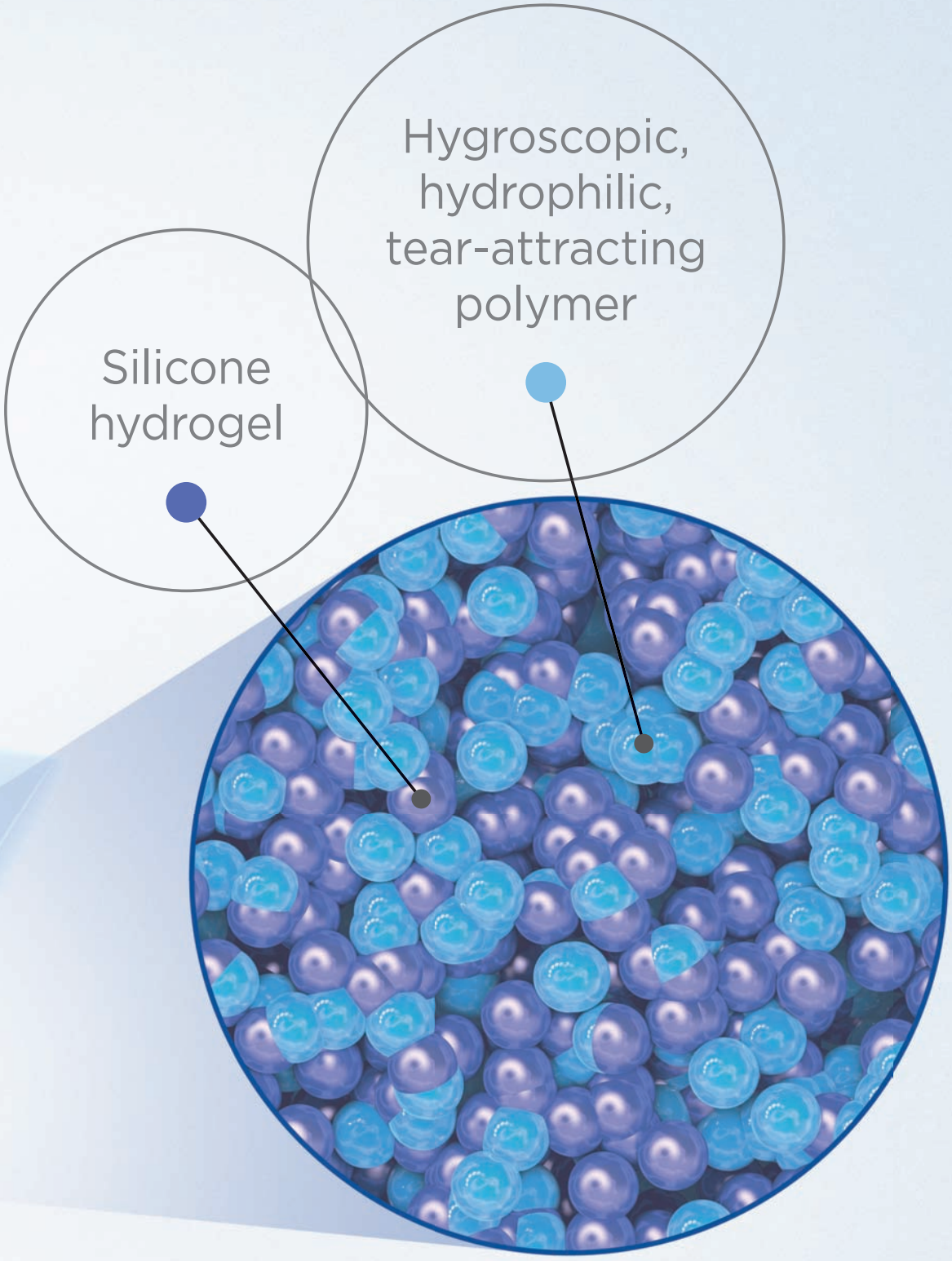

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VOL. 151 NO. 1 ■ JANUARY 15, 2014

IN THE NEWS

WellPoint Inc., the second-largest US health insurer, is selling **1-800 Contacts** to private equity firm Thomas H. Lee Partners LP. WellPoint also plans to sell **Glasses.com**, a division of 1-800 Contacts, to **Luxottica Group SpA.** Financial terms were not disclosed.

Says WellPoint CEO Joseph R. Swedish, "1-800 Contacts has strong brand recognition and a leading direct-to-consumer model. However, as we prepare for the coming changes to the health care system, we are focused on our core growth opportunities across both our commercial and government business segments. Proceeds from this transaction will support our continued capital deployment strategies."

InSite Vision announced positive Phase III results for the non-steroidal anti-inflammatory drug **BromSite** (bromfenac 0.075% in DuraSite vehicle) for reduction of inflammation and pain after cataract surgery. In one of two trials, 57% of patients on BromSite were completely free of inflammation at day 15, compared to 21% on the vehicle. The company plans to file a New Drug Application with the **FDA** later this year for the approval of BromSite.

An **FDA** advisory committee recommended the approval of **Hetlioz** (tasimelteon, **Vanda Pharmaceuticals**) as the first treatment of non-24-hour-disorder (**"Non-24"**) in the totally blind. Non-24 is a serious, rare and chronic circadian rhythm disorder that affects the majority of totally blind individuals and is characterized by the inability to synchronize the master body clock with the 24-hour day-night cycle, the company says.

NBEO Begins its Own Board Certification

NBEO board certification aims to recognize lifelong learning beyond entry level. **By John Murphy, Executive Editor**

The National Board of Examiners in Optometry (NBEO) has announced that a new optometric board certification program is now available.

NBEO-Board Certification Inc. is a private, nonprofit partner organization of NBEO that "recognizes and promotes professional lifelong learning to practicing optometrists through the joint Board Certification and Maintenance of Certification programs, thereby encouraging and supporting excellent optometric care for the general health, welfare, and benefit of the public," according to its website (www.optometry.org/nbeo-bc).

NBEO Board Certification "is used to recognize those optometrists who go beyond the entry-level requirements," the organization says. These basic requirements are the NBEO exams parts I, II, III and the TMOD exams.

To qualify for board certification, applicants must:

- Have a specified amount of experience in active practice or in a residency.
- Agree to take the CPDO exam after applying.
- Participate in the NBEO-BC Maintenance of Certification (MOC) program.

The MOC program requires participants to complete 50 COPE-accredited hours of CE (including 10 hours of self-assessment modules) as well as two "performance in practice modules" every two years.

The costs of NBEO-Board Certification include an initial application fee of $150, the CPDO exam fee of $500 and an ongoing MOC fee of $150 every two years.

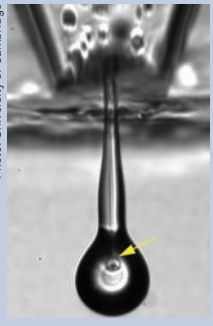

Photo: University of Cambridge

First Retinal Cells from an Inkjet Printer

Scientists at Cambridge University have shown proof-of-principle that an inkjet printer can be used to print retinal ganglion cells (yellow arrow in photo) and glial cells from adult rats.

"Our study has shown, for the first time, that cells derived from the mature central nervous system, the eye, can be printed using a piezoelectric inkjet printer," the researchers wrote. "Although our results are preliminary and much more work is still required, the aim is to develop this technology for use in retinal repair in the future."

Lorber B, Hsiao WK, Hutchings IM, Martin KR. Adult rat retinal ganglion cells and glia can be printed by piezoelectric inkjet printing. Biofabrication. 2013 Dec 17;6(1):015001. [Epub ahead of print]

Crizal® Prevencia™ and the Blue Light Hazard

Blue light plays a paradoxical role in health and vision. Not only is blue light essential for color perception, recent research has found that light in this band triggers critical physiological responses that include pupil constriction reflex and synchronization of the human biological clock. However, blue light may also be damaging to the eye, and the term "blue light hazard" has been coined to describe the danger this light presents to critical structures within the eye. Blue light can induce formation of damaging phototoxins, leading first to the death of critical retinal pigment epithelium (RPE) cells and then to photoreceptors. This damage is cumulative, and has been implicated in the development of retinal degenerative diseases such as age-related macular degeneration (AMD). The fact that blue light is both beneficial and harmful raises a critical question: Can we protect the eye from harmful blue light without simultaneously denying it the beneficial blue light? One way to accomplish this would be the creation of a lens that would selectively filter out the harmful wavelengths while transmitting the beneficial ones.

To determine if specific bands within the blue light spectrum were responsible for blue light's phototoxic effects, researchers from Essilor's Paris R&D laboratories joined forces with scientists from the Paris Vision Institute - one of the most important research centers in Europe on eye diseases—to develop a unique illumination system that allowed cultured swine retinal cells to be exposed to narrow bands of light. Using this test system, it was discovered that RPE phototoxicity was concentrated in a relatively narrow band, separate from the wavelengths necessary for the beneficial physiological effects of blue light. This finding paved the way for **selective photofiltration**: the creation of lenses that reduce the level of exposure to the harmful portion of the blue light spectrum, ranging from 415-455 nanometers (known as Blue-Violet light) while permitting the rest of the visible spectrum including beneficial blue light (known as Blue-Turquoise light), to enter the eye at a normal level. Thus, the eye's necessary visual and non-visual functions can be maintained while exposure to hazardous wavelengths is reduced.

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Crizal® Prevencia™ No-Glare lenses with Light Scan™ represent the first application of new patented technology[1], that enables selective filtration of harmful light – both Blue-Violet (BV) and Ultraviolet (UV) – while allowing beneficial light to pass through and maintaining exceptional transparency at all other visible-light wavelengths. In fact, Crizal Prevencia No-Glare lenses reduce the quantity of harmful Blue-Violet light reaching the eye by 20%. Unlike common yellow-tinted "blue blocking lenses," Crizal Prevencia No-Glare lenses cause minimal color distortion—indeed, these lenses are almost perfectly clear. Designed to selectively block harmful blue light while preserving transmittance of beneficial blue light, Crizal Prevencia No-Glare lenses offer the most selective eye protection on the market today.

Additionally, Crizal Prevencia No-Glare lenses also feature an Eye-Sun Protection Factor (E-SPF®) of 25, which means they provide 25 times more UV protection for the eye than wearing no lens at all. Integrating Essilor's superior No-Glare technology, Crizal® lenses are easy to clean, resistant to smudges, scratches, dust, and water, and protect against distracting glare and reflections. Maintaining excellent transparency, Crizal Prevencia No-Glare lenses offer optimal vision at all times.

INSTITUT DE LA VISION PARIS

UPMC SORBONNE UNIVERSITÉS

1. Covered under U.S. Patent No. 8,360,574. Additional U.S. and foreign patents pending.

Are Vitamins Just a Waste of Money?

Daily vitamin and mineral supplements do not prevent or limit the progression of chronic disease, according to an editorial in the December issue of *Annals of Internal Medicine*.

"We believe that the case is closed," the editors wrote. "Supplementing the diet of well-nourished adults with (most) mineral or vitamin supplements has no clear benefit and might even be harmful."

The editorial summarized the results of three meta-analyses published in the same issue, and garnered national media attention.

The first study analyzed the health benefits of multivitamin or single supplement use in more than 400,000 participants across 27 separate trials. The authors found no clear evidence to suggest that any form of nutritional supplementation reduced subjects' risk of all-cause mortality, cardiovascular disease or cancer.

The second study evaluated the protective effect of daily multivitamin supplementation on 5,947 men aged 65 years or older who were at an increased risk for cognitive decline. After 12 years of follow-up, the researchers observed no differences in cognitive performance or verbal memory between subjects who used multivitamins and those who received a placebo.

Mulitivitamins may not be so helpful, and may even be harmful, a new article says.

In the third study, researchers examined the potential benefits of a high-dose, 28-component multivitamin in 1,708 men and women with a history of myocardial infarction. After 4.6 years of follow-up, no significant reduction in recurrent cardiovascular events was observed in those who received the supplement.

After reviewing these data, as well as the results from several other multivitamin trials, the editorial's authors concluded: "Most supplements do not prevent chronic disease or death, their use is not justified, and they should be avoided."

So, should eye care professionals be seriously concerned by these findings? Probably not.

Ocular Nutrition Society Science Committee Director A. Paul Chous, MS, OD, of Tacoma, Wash., suggests that health care providers and the lay public should be very wary whenever such blanket claims are made. "Many supplement studies are relatively short term and use high doses of a single micronutrient that often depend on a host of additional cofactors," he says. "This means that the benefits and harms of supplementation may not be observed properly, and that any nutritional imbalance may exert pro-oxidant, disease-promoting effects."

In specific regard to eye care, Dr. Chous believes that optometrists who currently recommend nutritional supplements to their patients should continue to do so with confidence. "The AREDS studies prove that a combination of antioxidants and zinc—plus xanthophylls in AREDS2—reduces the risk of advanced AMD in high-risk patients," he says. "Further, there are now numerous interventional trials showing improved visual function in early AMD and diabetic eye disease patients following vitamin supplementation."

Guallar E, Stranges S, Mulrow C, et al. Enough is enough: Stop wasting money on vitamin and mineral supplements. Ann Intern Med. 2013 Dec;159(12):850-1.

New Handheld OCT Prototype Revealed

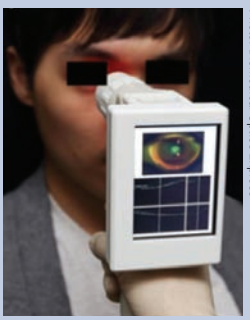

Photo: Biomedical Optics Express

Researchers at Massachusetts Institute of Technology described a new handheld OCT that can quickly scan for diabetic retinopathy, glaucoma and macular degeneration—and improve public access to eye care.

The researchers say this design is the first to combine ultrahigh-speed 3-D imaging, a tiny micro-electro-mechanical systems (MEMS) mirror for scanning, and a technique to correct for unintentional movement by the patient. These innovations, the MIT researchers say, should allow clinicians to collect comprehensive data with just one measurement.

The investigators only studied retinal imaging, but they say this device could be modified for anterior segment imaging, too. It's still a prototype, so no price has been determined yet.

Lu CD, Kraus MF, Potsaid B, et al. Handheld ultrahigh speed swept source optical coherence tomography instrument using a MEMS scanning mirror. Biomedical Optics Express. 2014 Jan;5(1):293-311.

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Ophthalmology and Optometry Academies to Work Together

For decades, ophthalmologists and optometrists have worked compatibly alongside one another on a clinical level, yet contentiously against one another on an organizational level. Now, the American Academy of Ophthalmology and the American Academy of Optometry have announced a joint initiative to put many of their differences aside to act together.

"There are many clinical settings in which optometrists and ophthalmologists work together very productively throughout the country," says Bernard J. Dolan, OD, MS, president of the American Academy of Optometry. "We thought if we could come together to develop mutually acceptable educational programs within those areas, it would be a benefit to both our members and the patients that we serve."

This effort marks the first-ever large-scale, organized effort within the optometry and ophthalmology professions in support of joint educational initiatives.

Bernard J. Dolan, OD, MS, president of the American Academy of Optometry.

David W. Parke II, MD, chief executive officer of the American Academy of Ophthalmology.

"Our ultimate goal here is to help our members deliver the best quality, safest, most resource-effective eye care that we can," says David W. Parke II, MD, chief executive officer of the American Academy of Ophthalmology. "We do work together individually in a very positive and collegial fashion and it is the responsibility of both organizations to try to not only meet member needs in this area, but to anticipate what those needs are going to be in the future."

No details have been decided yet, the organizations' executives say. The next step is to gather feedback from each group's members and stakeholders, and begin to explore common areas that will have the most significant impact on members and their patients.

But they aim to take their time and do it right. "Everyone appreciates that the devil's in the details," Dr. Parke says. "I think one of the reasons that Dr. Dolan and the Academy of Optometry and myself and the Academy of Ophthalmology are moving very slowly is we want to take the right steps to make this a sustainable relationship."

The organizations anticipate that these programs will be developed over the next 12 to 18 months, with a formal launch in 2015.

Since the initial announcement, the feedback has been guardedly positive, the executives say.

Induced Myopia and Hyperopia Can Be Reversed in Chicks—and Maybe in Children, Too

Researchers in Singapore have found that newly hatched baby chicks raised in red light for up to six weeks developed progressive myopia, while those raised in blue light had progressive hyperopia.

More interestingly, the researchers were able to reverse the light-induced myopia to hyperopia in the chicks by exposing them to three weeks of blue light. Likewise, hyperopic chicks developed myopia after the same time under red light.

The researchers speculate that this manipulation of chromaticity could apply to the management or prevention of myopia in children.

But that remains to be determined, says Frances Rucker, MCOptom, PhD, whose research at New England College of Optometry also explores post-natal myopia development.

"Recent experiments have shown that blue light is important in preventing myopia development in chicks, but other experiments in monkeys have indicated that red light is important, too," Dr. Rucker says. "Taking this into consideration, it is my opinion that optometrists should recommend the use of white light illuminants with a strong blue (but not UV) component—in other words, 'daylight' bulbs—to provide protection against myopia progression."

This study builds on previous research on refractive error under blue and red light exposure, Dr. Rucker says. An interesting modification in this study is that the researchers didn't use pure blue or pure red light, yet still acheived the refractive effect.

Foulds WS, Barathi VA, Luu CD. Progressive myopia or hyperopia can be induced in chicks and reversed by manipulation of the chromaticity of ambient light. Invest Ophthalmol Vis Sci. 2013 Dec 9;54(13):8004-12.

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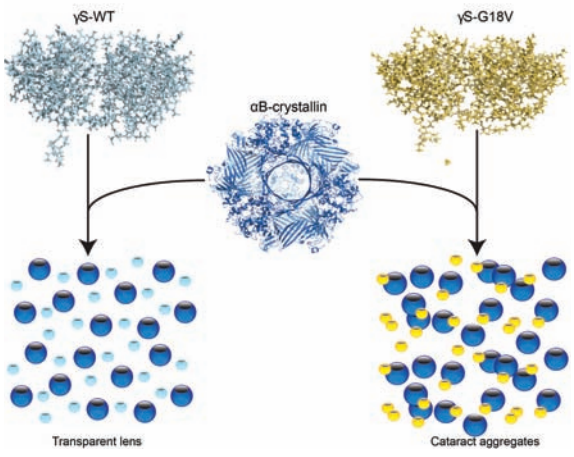
Researchers Decode Cataract Creation

Researchers have mapped the molecular inner-workings of the human eye and may have cracked the code of cataract creation. The chemists' groundbreaking new findings could be used to help prevent a condition that currently affects nearly 20 million people worldwide.



The lens stays clear thanks to the "chaperone" protein alpha B-crystallin, which binds to gamma S-crystallin (above right) to prevent a cataract from forming.

Researchers from University of California Irvine and the Leibniz Institute for Molecular Pharmacology in Germany identified the structures of normal proteins in the human eye and a genetic mutation known to cause cataracts in children. According to their study, the "chaperone" protein in the eye—which prevents the other two structural proteins from clumping into cataracts—binds far more strongly to mutated or damaged proteins in an effort to keep the lens clear.

Unfortunately, the human eye is equipped with only a finite number of these chaperone proteins, called αB-crystallins. When they're tapped out, the now-weakened eye proteins are able to aggregate, which forms a cataract.

However, "understanding the molecular mechanism of what goes wrong in the eye that leads to a cataract could lead to the development of better treatment options, including more sophisticated artificial lenses and drugs," says the study's co-author Rachel Martin, PhD, associate professor of chemistry at UC Irvine.

For instance, the researchers are hopeful that organic chemists can use their findings to create sight-saving treatments to prevent initial aggregation.

Kingsley CN, Brubaker WD, Markovic S, et al. Preferential and specific binding of human alpha b-crystallin to a cataract-related variant of gamma s-crystallin. Structure. 2013 Dec 3;21(12):2221-7.

In-Office, Disposable Dry Eye Test Now Available

The FDA has cleared RPS's InflammaDry—a rapid, disposable, in-office test for the diagnosis of dry eye disease—for sale in the United States. The InflammaDry test is reimbursable under CPT Code 83516. ■

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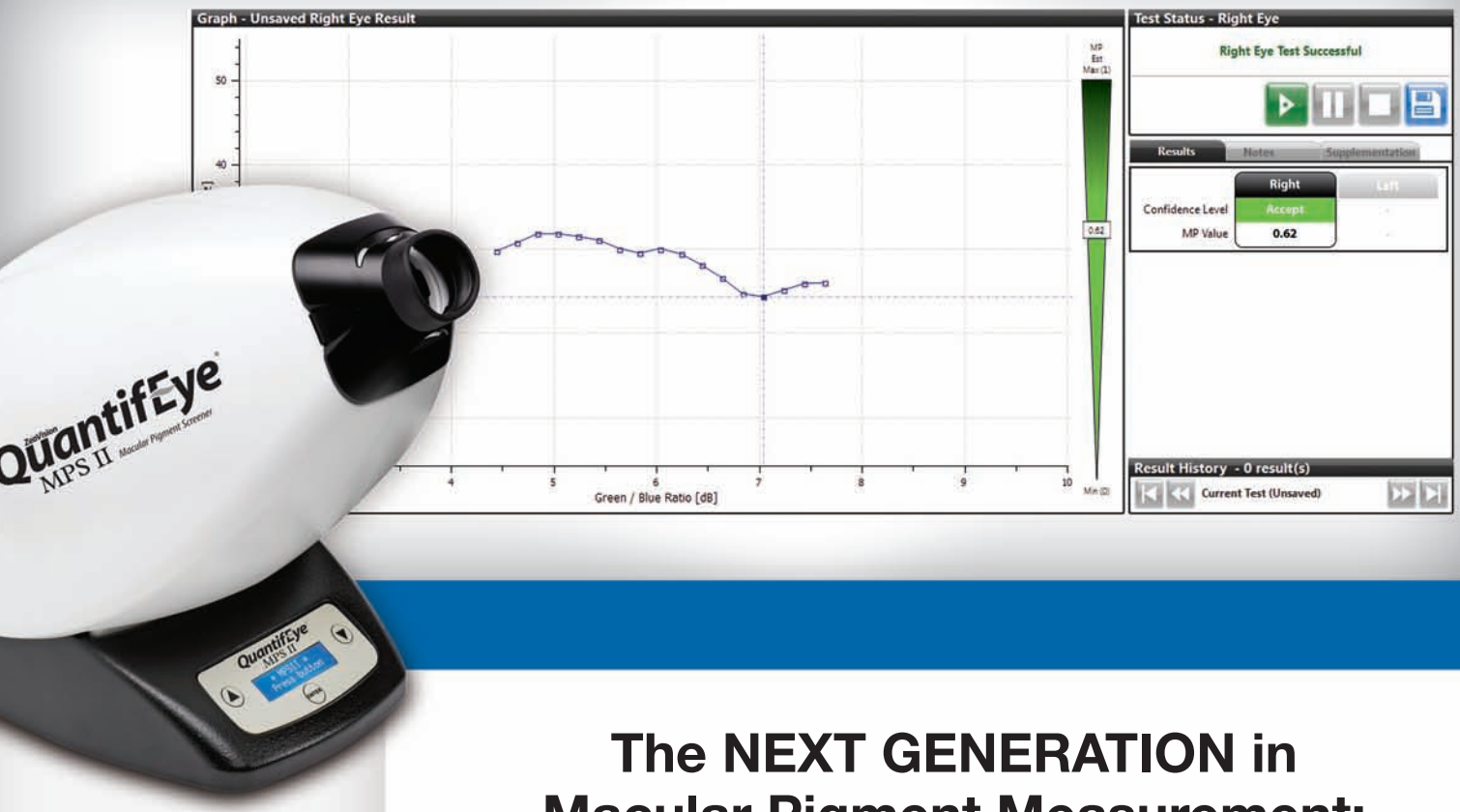

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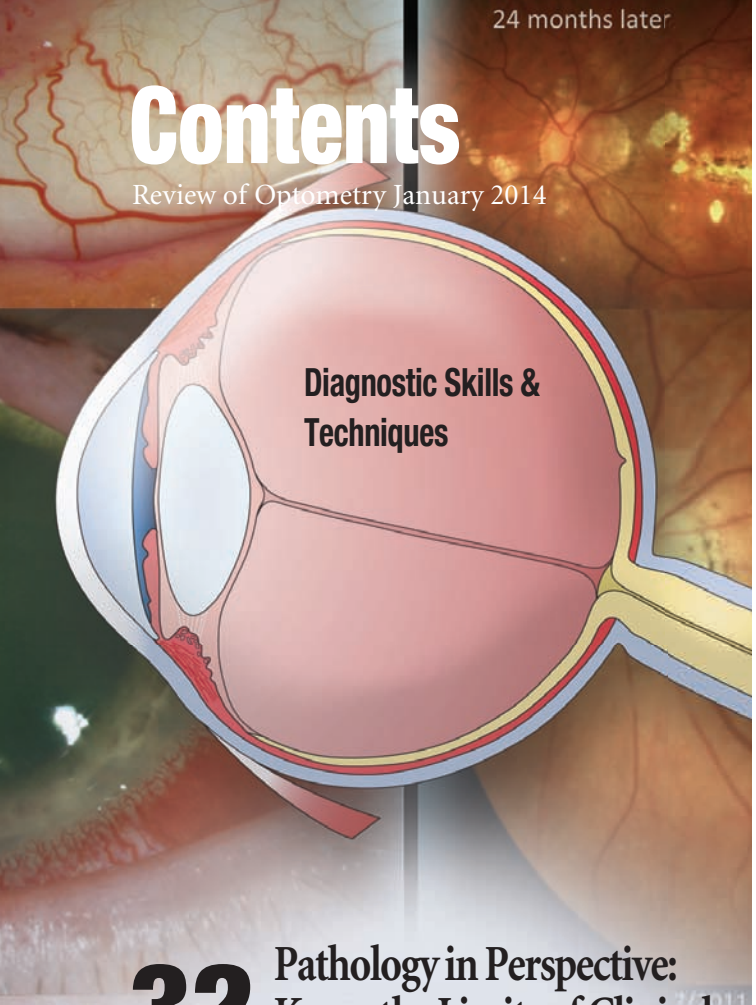

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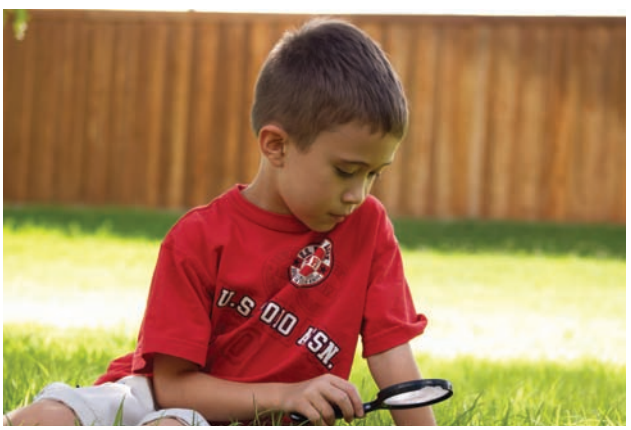
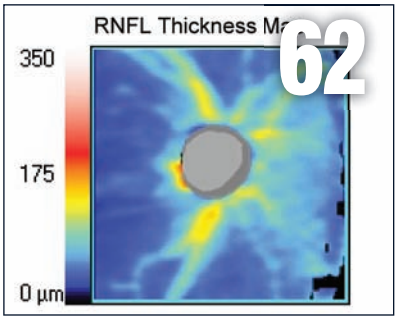
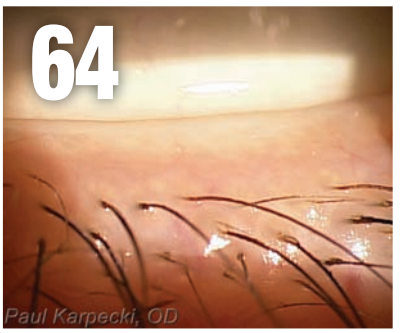


Departments

Review of Optometry January 2014





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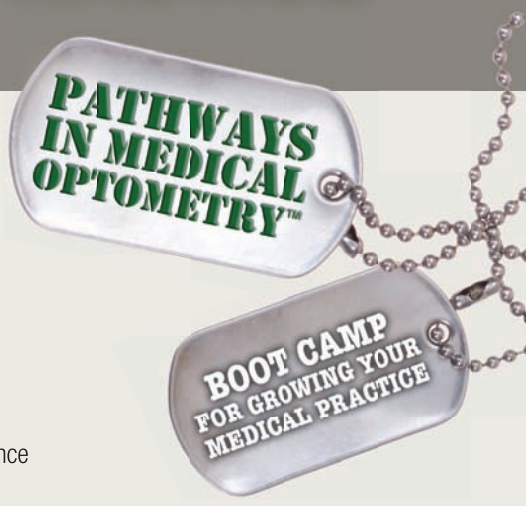

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ALLERGAN

Elementary, My Dear?

Get out your magnifying glass and put on your deerstalker hat. The game is afoot!

By Jack Persico, Editor-in-Chief

One of the great pleasures of reading or watching Sherlock Holmes (in any incarnation, but especially Benedict Cumberbatch's steely portrayal in the new BBC series) assess a crime scene is his legendary capacity for deductive reasoning. A seemingly innocuous clue—a scratch on a cell phone, for instance, or the position of a chair in the room—reveals previously hidden patterns of cause and effect obvious only to him.

Sherlock's great skill is his ability to differentiate meaningful facts from humdrum happenstance. He can effortlessly make connections between disparate clues and, in so doing, construct with utter certainty a narrative of events he did not witness. It's entertaining, inspiring—and obviously fictional.

Doctors who spend their days attempting to pull off similar feats of deductive acumen in the exam room surely know just how tenuous those connections, and how provisional the conclusions, really are. It's the bane, and I imagine also the joy, of clinical practice. When those flashes of brilliance do happen, they make it all worthwhile.

A Visit to Baker Street

This month, we're pleased to highlight several articles to help you hone those Sherlockian skills.

On page 32, associate clinical editors Al Kabat, OD, and Chris Sindt, OD, present pairs of similar photos that might lead one to the wrong conclusion based on appearance alone. Take the 20-question self-test to match wits with these clinicians by collecting the clinical evidence, sussing out the red herrings and nailing the diagnosis.

Retina specialists Mohammad Rafieetary, OD, and Eric Sigler, MD, address similar challenges in the posterior segment on page 46. Take a look at these retinal masqueraders and see if, as Sherlock said, when you eliminate the impossible, whatever remains, however improbable, must be the truth.

And in our Optometric Study Center on page 52, Albert David Woods, MS, OD, and Michelle K. Caputo, OD, offer guidance on when to call in the diagnostic heavy artillery—radiologic testing.

Killer Whales, Eskimo Tales

Those of us in the lower 48 probably think it's overkill that Eskimos are reputed to have so many different words for *snow*. Surely some are superfluous, right? Yet optometrists must be familiar with umpteen different types of red eye, along with the appropriate treatment for each.

Greater nuance in classification can be liberating, but also a little maddening. Doctors are already griping about the new obligations that will befall them when the 10th edition of the ICD takes effect this October. The number of diagnostic codes one must contend with will jump from 14,000 to a whopping 70,000—that's a lotta suspects, even for a master sleuth. So many, in fact, that it's become a source of parody. A publication called *Struck by Orca* pokes fun at the more eccentric maladies in ICD-10. (FYI, should you encounter such a patient, the code is W56.22xA, "struck by orca, initial encounter.")

The new ICD will frustrate and challenge you. So will your next patient who presents with subtle and conflicting findings. But will all your hard work to hone your skills feel worth it, when inspiration comes and you've cracked the case?

Why, that's elementary. ■

The Heart of the Matter

This month in our companion publication *Review of Cornea & Contact Lenses*, which just got a terrific new design courtesy of talented art director Matt Egger, longtime columnist Gary Gerber, OD, discusses how he answers the question, "What do you do?" Instead of defining himself matter-of-factly by his job title, he gets at the essence of what he truly enjoys the most about optometry: "Ten to 20 times per day, I get a chance to change someone's life for the better, and let them experience the world in ways they never thought they could." It's a refreshing way to view your place in the world and recognize that you're part of something special. Be sure to look for Dr. Gerber's column in the new and improved *RCCL*. And tell us what you think of it. Drop me a line at jpersico@jobson.com with any feedback or suggestions. Because that's what *I* do—help doctors share their expertise with each other, for the common good. Thanks for being a part of it.

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References: 1. Kassan SS, Moutsopoulos HM. Clinical manifestations and early diagnosis of Sjögren syndrome. *Arch Intern Med*. 2004;164:1275-1284. **2.** Sjögren's Syndrome Foundation. Sjögren's Syndrome Foundation. 2001. Available at http://www.sjogrens.org. Accessed September 5, 2013. **3.** Liew M, Zhang M, Kim E, et al. Prevalence and predictors of Sjögren's syndrome in a prospective cohort of patients with aqueous-deficient dry eye. *Br J Ophthalmol*. 2012;96:1498-1503. **4.** Martin LS, Massafra U, Migliore A. Sjögren's syndrome: an under-diagnosed disorder. *CLI*. May 2004.

Phenomenon-sense

Omens are everywhere, ruling our lives. For example, if a patient hears strange voices in his head, it's a sign that he will never leave your practice. **By Montgomery Vickers, OD**

Logic, education and intelligence have become so yesterday in their value. Now, we're in a new era of charms, potions and, especially, omens.

Of course, I've always known that some mysterious power must be summoned in order to succeed in life. After all, how else can one explain how I made it through Physiological Optics in 1975? Had to be a miracle.

When my daughter was in labor with her first son, Max, her husband's father, Amir, was seen wandering back and forth across a busy intersection about a hundred yards from where I was listening to a huge flock of birds chattering in a tree above my head. Amir's pacing, combined with my own fascination with the aforementioned birds (none of which pooped on my head, despite their best efforts) must have been a good omen, right? Right! And grandson Max is amazing, although he does sit on tree branches and tweet while pacing from branch to branch a lot.

Mysterious Ways

There are, on the whole, good omens and bad ones. Their bottom line is Cause and Effect. I want to share some of these omens I've noted in my practice:

1. ***If*** a patient blocks out the sun when he enters the room, ***then*** he will likely break the footrest off of your exam chair.

2. ***If*** a squirrel chases you into your office (this has happened to me), ***then*** you are probably nuts.

3. ***If*** a patient tells you, "My insurance always pays my whole bill," ***then*** you should, as professionally as possible, invoke the old French phrase "merde de taureau," or something very similar.

4. ***If*** your new presbyopic patient picks up her glasses and then drives her car through your office landscaping while texting, ***then*** you are the wonderful doctor who helped her see her cell phone again.

5. ***If*** a patient appears to be hearing strange voices in his head, ***then*** he will never leave your practice.

6. ***If*** you see an increase in the number of woolly caterpillars outside, ***then*** you can expect the next week's worth of patients will be tracking woolly caterpillar guts onto your office carpet. (FYI: The best way to remove woolly caterpillar guts from your office is to set it on fire and start over.)

7. ***If*** you spill fluorescein on your patient's new dress, ***then*** she was heading from your office to the most important job interview in her life—which she will tell you right after you hit her with phenylephrine and mydriacyl drops, too.

8. ***If*** Hell freezes over, ***then*** you will see an increase in your Medicare reimbursement.

9. ***If*** you decide to cheerily tell the next patient "Merry Christmas," ***then*** he or she will be a Jehovah's Witness.

10. ***If*** a patient says he "can't afford" your recommendation, ***then*** he is a smoker.

So, doctors, what do you see happening in your office that seems witchy or perhaps from a higher power? Have you learned there are no accidents and your life is perhaps controlled from without, not within? What are your omens? What causes lead to what effects? If you see more patients, do you make more money? Ain't the vision plans fault there. Are you in control of anything at all? Should you hire consultants, or just buy a Ouija Board? Strategize by tea leaves or fortune cookies, perhaps?

Works for me—tea and cookies have never let me down. ■

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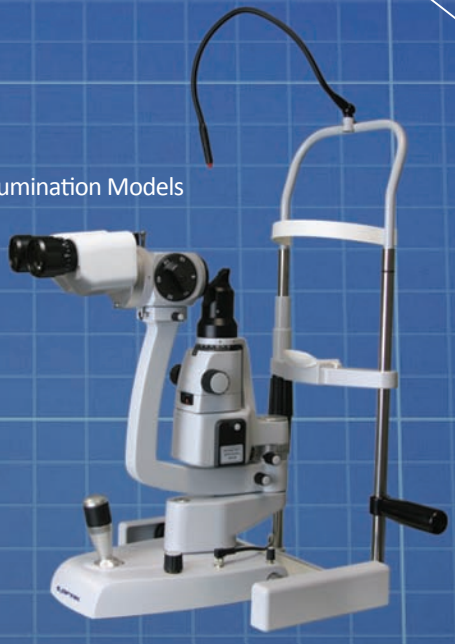

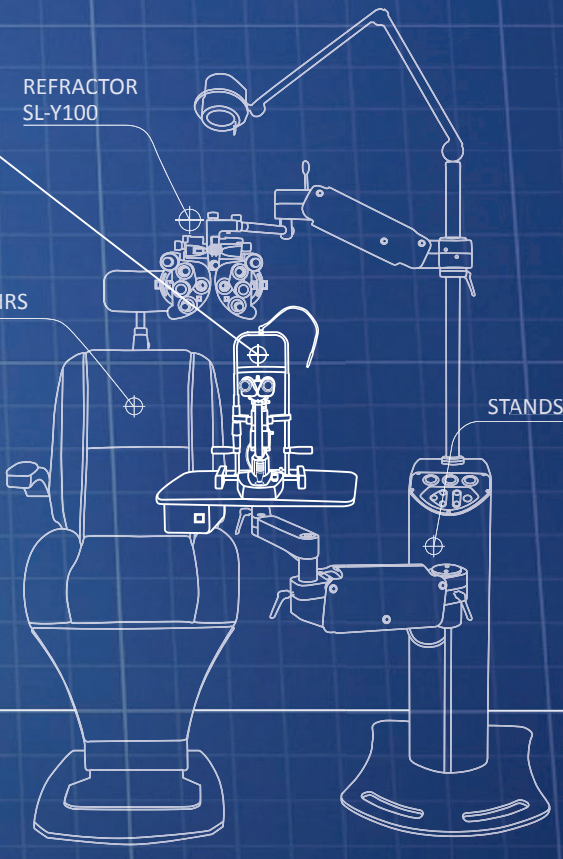

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2014: The Year of Change

Plan now to get ready for many changes this year—like Obamacare, the Medicare fee fix and ICD-10, just to name a few. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

Here we go—this is the year of change: The Affordable Care Act implementation has started; Medicare's flawed Sustainable Growth Rate formula for its physician fee schedule is finally being fixed; and the ICD-10 rolls out in October. Add to that: changes in professional responsibilities tied to Meaningful Use Stage 1 and Stage 2 EHR Incentive Programs; increased scrutiny of professional services via prepayment audits (meaning your practice is targeted for review *prior* to getting paid)—and many of us are just now implementing many of the new rules that were put into place *last* year.

Holy $#@%! What's a health care provider to do?

Plan for Change

First of all, don't panic. All of these changes are manageable. But, you have to have a plan to track, prioritize, integrate and implement these changes into your practice.

Let's use the ICD-10 as an example.

The implementation date is October 1. So, knowing that you have about nine months to go, what does your roadmap for implementation look like? CMS has published a number of training schedules and checklists that you can reference (www.cms.gov/Medicare/Coding/ICD10/ProviderResources.html). Keep in mind that eye care providers have a very focused implementation of the ICD-10, so not everything on the CMS list applies to you. Here's how you could approach it.

1. Obtain the new CMS-1500 forms (if you still print out a paper form for submission) and/or start a dialogue with both your EHR provider and billing gateway to ensure that they are using the new format, which was required as of January 1, 2014.

2. Talk with your EHR provider to find out how it's rolling out its transition from ICD-9 to ICD-10. (Keep in mind that you'll need both code sets to run simultaneously for one full year: October 1, 2014 to September 30, 2015).

3. Check with your billing clearinghouse to find out how it's implementing its transition from ICD-9 to ICD-10. How will it track claim rejections, reprocess claims that have dates of service prior to the October 1 start date, etc.?

4. Mine your EHR database for your top 25 diagnoses that were billed in 2013, based upon frequency.

5. Categorize these diagnoses. For example, refractive/contact lens related diagnoses vs. medical diagnoses. And further subdivide these into anterior segment (allergy, infectious, inflammatory) vs. posterior segment (glaucoma, macular degeneration, retinal vascular disease), and so on.

6. Get current information. Access resources (such as www.ReimbursementPLUS.com* or www.icd10data.com/Convert) to assist you with the crosswalks from the ICD-9 to the ICD-10.

7. Hold monthly training sessions with your entire staff to work on a specific group of diagnoses. For example, learn the refractive changes in February, anterior segment (infectious) in March, anterior segment (allergic/inflammatory) in April, and so on. By the end of summer, everyone in the office will be well versed in the ICD-10 format of the diagnoses that you work with 95% of the time.

8. Confirm with insurers that their claims processing will not be delayed with ICD-10 submissions.

9. Have your entire office staff attend CE sessions on ICD-10.

While this is just an illustration of how to manage a change event in the practice—from discovery to integration—the concept can be applied to any of the arising issues. The key is to assign a single source within the practice who will track and disseminate information about changes that could impact the practice. Create opportunities for training, and commit yourself to following through to successful implementation.

Keep in mind that change isn't always bad. It's how you approach it and manage it that makes the difference. So, learn how to anticipate change, embrace change, direct change and enjoy the benefits of what successfully integrating change can bring. Maybe 2014 isn't just The Year of Change—maybe it's also The Year of Success! ■

* Disclaimer: As the owner of this company, I have a financial interest in this product.

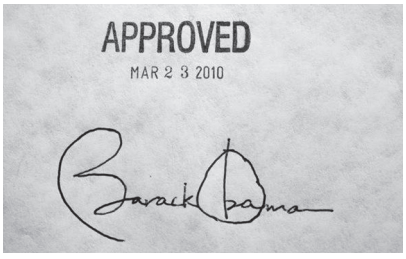
How to Adopt the Affordable Care Act in Your Practice

The new health care law holds tremendous opportunity for ODs. Here are three ways to get your office on board with Obamacare. **By Bryan M. Rogoff, OD, MBA, CPHM**

The US government's effort to increase the quality and affordability of health care, while decreasing the costs and protecting consumers, has been extremely political and highly criticized. Even after March 23, 2010, when President Obama signed the Patient Protection and Affordable Care Act (ACA) into law, the matter was hardly settled.

Indeed, the questions and confusion had only just begun. Lawmakers, providers, patients and managed care companies have been scrambling to prepare for the biggest overhaul in health care since 1965, when Medicare and Medicaid were introduced. As recently as a couple months ago, President Obama and the Department of Health and Human Services came under fire for the initiative's poorly functioning website, HealthCare.gov.

Despite a rocky start, the program is picking up steam—as of January 1, more than 2.1 million Americans had enrolled for coverage. And, before HealthCare.gov even launched, Americans were already benefiting from the early



Almost four years after President Obama signed the Affordable Care Act into law, its implementation remains a challenge.

provisions of the ACA:

- Young adults under 26 years of age can remain on their parents' health care plan.
- Children with pre-existing conditions can't be denied coverage.
- Small businesses can receive tax credits for contributing to their employees' health insurance.
- Patients have the right to appeal health care plan decisions.
- Insurance companies are prohibited from imposing lifetime dollar limits on essential benefits, like hospital stays.
- Preventive services—such as immunizations, annual physicals and screenings—are covered on all new plans.

Still, 2014 poses the biggest, most anticipated, and most controversial changes from the ACA yet. These changes have tremendous opportunity for optometrists, but they must be proactive with the upcoming changes. For instance, 17 different vision plans enrolled 48% of adults in 2012, but only one out of three members had an eye exam. With the expected influx of even more patients, there's plenty of opportunity to further educate them about the importance of comprehensive eye examinations.

Now, to compete in today's changing health care arena, practitioners must be able to react, educate and market to continue growing their practices.

1. Essential Health Benefits

The ACA requires that health care plans provide 10 essential health benefits (EHB) to standardize the minimum coverage offered to patients. These essential health benefits were written to protect patients from insurers that sold plans of sub-par coverage, which could leave patients in financial

ruin if something catastrophic happened to their health. These benefits must be included for a plan to be certified to compete on the health care exchanges.

The 10 EHBs are: ambulatory patient services; emergency services; hospitalization; maternity and newborn care; mental health and substance use disorder services (including behavioral health treatment); rehabilitative services and devices; prescription drugs; laboratory services; preventive/wellness services and chronic disease management; and pediatric services, including dental and vision care.

• ***How to plan:*** For optometrists, the most important of these is the pediatric vision care benefit. The Department of Health and Human Services predicts that more than 8 million children could gain from this essential health benefit.

The pediatric vision care benefit includes an annual comprehensive eye exam, treatment and materials from birth through age 18.

However, the ACA language is vague about exactly what this benefit should entail; individual states can define the actual coverage using current vision plans as a benchmark, so coverage will vary from state to state. Some plans may include devices and materials; some may only include services. (Because of this law's vague language, there is still a good deal of confusion and many unknowns.) Eyeglasses and contact lenses may be at least partially covered and exempt from the 2.3% excise medical device tax.

Also, the ACA mandates that there should be no annual limit on any EHB. Plans that have an allowance—in which employees are given a specific amount to apply toward eyewear products of their choice and have the option to pick high-end materials with add-ons—may run up against this mandate. So, expect different interpretations of the no out-of-pocket limit rule.

Although the ACA does not specify exactly which services—let alone tests and treatments that will be reimbursed—it is expected to follow the FEDVIP (Federal Employees Dental and Vision Insurance Program) plans. FEDVIP programs vary by state, along with the coverage and allowances for eyewear and contact lenses. Different insurance carriers offer different allowances for products. For example, Aetna, VSP, United Healthcare and Blue Cross Blue Shield offer FEDVIP plans; consult their reimbursement schedules for products and services to help you prepare for the EHB changes.

Understanding and planning for EHB is just the start of how you can prepare your practice for 2014. It's more important than ever to market internally to your existing patients about these new benefits. Educate parents about the importance of yearly pediatric eye exams and the services your practice offers.

Also, market externally by building partnerships with your local PCPs and pediatricians to demonstrate your expertise and concern with this demographic. Partner with local philanthropic

How the ACA Affects You, the Small Business Owner

For years, small business owners—such as optometrists who own private practices—didn't have the leverage that large corporations had to negotiate better health insurance coverage and pricing for employees, leaving their staff members either underinsured or uninsured.

The ACA definition of a small business is one that has the equivalent of 50 or fewer full-time employees. These businesses are not required to offer or contribute to their employees' health insurance—but do qualify for tax credits when they contribute to their employees' health coverage. The tax credit is worth up to 50% of the contribution toward employees' premium costs.

The smaller the business, the bigger the credit. The tax credit is highest for companies with fewer than 10 employees who are paid an average of $25,000 or less.

To qualify for the Small Business Health Care Tax Credit, you must:

• Have fewer than 25 full-time equivalent employees making an average of about $50,000 a year or less.

• Pay at least 50% of your full-time employees' premium costs. (You don't need to offer coverage to your part-time employees or to dependents.)

• Purchase your employees' health insurance through the Small Business Health Options Program (SHOP, www.healthcare.gov/marketplace/shop), a marketplace much like the health care exchanges offered to individuals. Plans offered on SHOP have the same 10 essential health benefits as those on the individual exchange.

(If you're self-employed and have no employees, you don't use SHOP. You'd get insurance through the individual health exchange marketplace. Also, larger businesses that have more than 50 full-time equivalent employees with average wages above $250,000 must provide full-time employees with health insurance in 2015.)

From a business perspective, the cost of employee turnover can never be recouped in any business model, so offering quality, affordable health care to your employees can help reduce turnover to a minimum.

For more information, visit www.healthcare.gov/small-businesses.

clubs and schools, and volunteer to perform vision screenings to uncover hidden refractive and binocular disorders, helping your practice capture this emerging market share.

2. Health Care Exchanges

The health care exchanges were set up as a marketplace for consumers to shop, select and purchase certified health insurance. The key word is *certified* so that, as mentioned before, all plans available on the exchanges have the 10 EHBs. Now, because all plans must have the same basic coverage, the exchanges should increase competition, streamline administrative costs and eliminate discrimination of individuals with pre-existing conditions. This methodology was designed to improve efficiencies and reduce waste, with the goal of lowering health care costs.

How will exchanges lower health care costs for consumers? Traditionally, large corporations have negotiated with insurers for the type and amount of coverage they would provide for their employees. Depending on their risk pool, average age and the number of employees, the corporations and insurers negotiated a price. Usually, the employer would pay a percentage of the premium and the employee would be responsible for the rest.

If you did not receive health insurance from an employer or were self-employed, you had to purchase health insurance on your own. Individuals and small businesses didn't have the opportunity to negotiate pricing, have a portion subsidized from an employer, or be placed in certain risk pools. Instead, premiums were determined by health, age and pre-existing conditions.

With the implementation of the ACA, the exchanges now treat individuals and small businesses like corporations by grouping them together to receive certain discounts. Having the public exchanges allows individuals to compare plans and choose according to coverage and prices (www.healthcare.gov/marketplace/individual).

Plans are categorized in tiers: bronze, silver, gold and platinum. As you go up in tier, the pricing reflects additional coverage and lower deductibles. Once an individual has selected a plan, additional insurance (such as dental and vision) can be added. Remember, pediatric vision coverage is included with plans on the exchanges, so individuals older than 18 have to add vision insurance separately if they want additional coverage.

If you've visited the exchanges, you may have noticed that the traditional vision plans—such as VSP, EyeMed, Davis Vision, etc.—are not listed. There is a reason for this: Vision plans do not provide ALL 10 EHBs, so they're not considered certified health care plans. Instead, vision insurance providers had to partner with other insurers to provide the vision portion of their plans on the exchanges.

• ***How to plan:*** Even though it has been difficult to access the exchanges and purchase health insurance, practitioners should be aware of which insurers are participating in their respective states. Some exchanges are state-run, and may be easier to access than those that are federally administered. (*See "State-Run Health Insurance Exchanges," left.*) Not all insurers are listed on the exchanges, but the ones that are listed have agreed to participate and have their plans certified.

If you are not currently a provider under the health care plans listed in the exchange, contact the insurance companies that are participating to become credentialed on their panel. All new contracts should coincide with the Harkin Amendment, which states that health care providers (including optometrists) cannot be discriminated against participating, as long as they practice within their state regulations.

State-Run Health Insurance Exchanges

Most Americans can use HeathCare.gov to access the health insurance exchange in their state. States that administer their own exchange marketplaces include:

- California www.coveredca.com
- Colorado http://connectforhealthco.com
- Connecticut www.accesshealthct.com
- District of Columbia https://dchealthlink.com
- Hawaii www.hawaiihealthconnector.com
- Idaho www.yourhealthidaho.org
- Kentucky https://kyenroll.ky.gov
- Maryland www.marylandhealthconnection.gov
- Massachusetts www.mahealthconnector.org
- Minnesota www.mnsure.org
- Nevada www.nevadahealthlink.com
- New Mexico http://bewellnm.com
- New York https://nystateofhealth.ny.gov
- Oregon www.coveroregon.com
- Rhode Island www.healthsourceri.com
- Vermont www.vermonthealthconnect.gov
- Washington www.wahealthplanfinder.org

3. Medicaid Expansion

The ACA expands eligibility for the Medicaid program.

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limited resources and low incomes. Funding is shared and based on each state's per capita. States determine certain methodologies and rates for payment and reimbursement, and choose providers to participate in the program.

Because states have joint input regarding guidelines, eligibility varies. Most use similar eligibility guidelines for low-income adults under 65, unless they qualify in another eligibility group: children under 18, pregnant women, parents, the elderly or those with a disability.

The ACA has now redefined eligibility and expanded it to all states. The federal government is funding 100% of the expansion for the first three years, then 90% afterwards. (States still have the option to opt out of the expansion without losing their funding; indeed, 21 states—including Florida, Georgia, Texas, Virginia, Wisconsin and the Carolinas—are not expanding Medicaid coverage) With the new expansion, it's estimated that more than half of the uninsured will have access to Medicaid, including childless adults and everyone who is under 133% of the federal poverty line. (To view these groups, visit www.medicaid.gov/AffordableCareAct/Provisions/Eligibility.html or http://obamacarefacts.com/obamacares-medicaid-expansion.php.)

• ***How to plan:*** Optometrists are already highly involved in the Medicaid program, so with the Medicaid expansion we can expect an increase of patients. Check your state's Medicaid website to see if it participates with the ACA Medicaid expansion and the state reform provisions. (Go to Medicaid.gov to connect to your state's Medicaid site.) To accommodate the influx of new patients, and any additional mandates, make sure that your practice is running efficiently and productively.

If you don't participate in the Medicaid program, visit your state's Medicaid website to learn how to become a Medicaid provider.

While there is more federal funding to cover the additional uninsured, there is also an expansion of increased payments (similar to Medicare) for primary care physicians. However, optometry is not included in the specific definitions, so it's our responsibility to change that. Currently, the Optometric Equity in Medicaid Act (HR 855), supported by the American Optometric Association and state optometric associations, has been proposed and is gaining endorsement. If passed, it will require coverage under Medicaid to include optometrists as providers of medical and other eye health services, under the scope of each state.

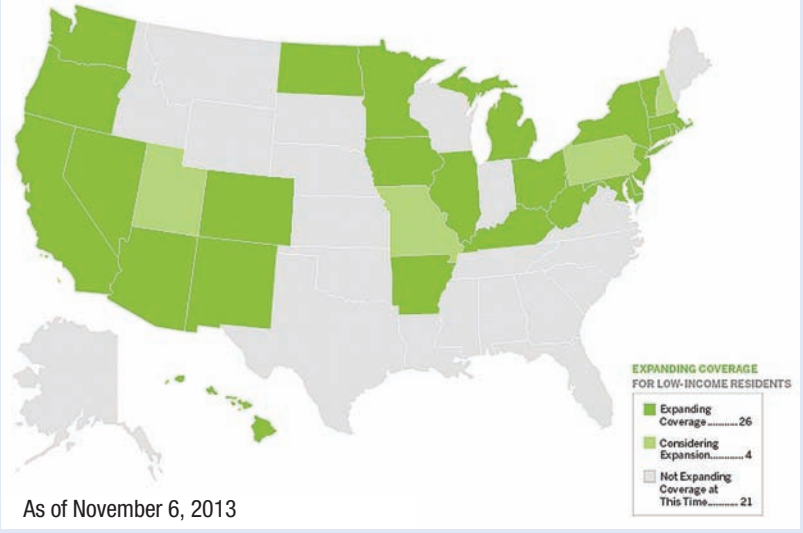
The ACA presents optometrists with new opportunities like never before. The business-as-usual idea is gone and ODs must now embrace this new world of health care. All modes of practice—private vs. corporate, independent contracts vs. employed, primary care vs. tertiary care—are faced with new challenges for 2014, but how you transform your practice can raise the bar not only for the profession, but also in patient care. How you modify your business model can point you toward success. ■

Dr. Rogoff is an independent practice consultant with expertise in areas of health care, business and operational management. He is also the partnerships and marketing liaison for the Maryland Optometric Association.

Thanks to Jon Hymes, interim executive director of the American Optometric Association, for providing information for this article.

Medicaid Coverage Expansion Map of the United States

Thanks to the Affordable Care Act, 25 states and the District of Columbia are expanding Medicaid eligibility.



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†The sixth measure was conjunctival staining.

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Reference: 1. Morgan P, Chamberlain P, Moody K, et al. Ocular physiology and comfort in neophyte subjects fitted with daily disposable silicone hydrogel contact lenses. *Cont Lens Anterior Eye.* 2013;36(3):118-125.

Getting Down to Business

When you enter your own private practice, eye care becomes the easy part.

By Susan Tran, OD

Sometimes the most unexpected things in life are the most pivotal. For me, it was a phone call from my optometrist.

At the time, I was working in an ophthalmology setting in San Francisco, where I'd stayed for more than two years after my residency at the San Francisco VA. Like an old friend, my OD asked what an L.A. girl was still doing in San Fran.

"I'm just waiting for the right opportunity to move back," I said.

I had no idea that the phone call was that opportunity.

My OD wanted to sell her practice—to me.

There was dead silence on the line. My thoughts jumbled. Did I really want to leave a stable job? Was I ready to be a business owner? I was happy where I was, after all. My schedule was filled with surgical consults, perioperative care and acute exams. My professional life was quite literally like clockwork: clock in, do a great job, clock out, go home, collect a paycheck. I'd wanted a job that drew upon my training, and I had it.

But even before that phone call, I knew something was missing. Something elemental.

I cranked through so many patients that I couldn't put a face to a name at the end of the day. When I joined the ophthalmology group, I'd underestimated how important personal relationships were to me, and I'd come to realize that having rapport with patients was as equally important to me as providing quality eye care.

Even before my OD rang, I'd thought about breaking free and starting a private practice. When I was a third-year in optometry school, it occurred to me during a practice management course that I might have my own practice someday. It was an exciting thought—but it terrified me. Starting my own private practice remained a "maybe, someday, one day" aspiration.

Call me practical.

But much like test-driving a car, I decided to take my OD's suggestion and try it out. She told me that I'd have to speak a lot of Vietnamese, because that comprised most of her client base. Vietnamese isn't my first language, but it proved to be negligible. I fell in love with the practice within a few days. I was worried I'd get bored easily if all I did were refractions and new glasses, but to my surprise, it was frequented by patients seeking primary eye care.

Quite frankly, though, I was still conflicted. The practical side of me wanted to just stay put, but the typically dormant risk-taker planted seeds that I couldn't shake off.

Sometimes opportunities don't knock twice.

So, I jumped. And I hit the ground running.

As it turns out, "someday" came and "one day" is today.

New Beginnings

As a new self-employed optometrist, I'm running into all sorts of new beginnings that I didn't even know existed. It's been three months, and every day is still a new day. Optometry has become the easiest part.

It's the nine-headed hydra of entrepreneurship that I'm learning on the fly.

While I attempt to brace myself

for the challenges ahead of me, I know that there will be plenty of things that I wish I had known. I'm sure I will run into things that no one told ever me before I became a business owner; things I never learned in school; things that I wish I at least had an inkling about.

Thus, I've decided to embark on yet another adventure by turning my experiences into an ongoing blog at www.revoptom.com.

I have no idea what's in store for the upcoming year, but I do know that I'm going to document it.

As I can only anticipate the challenges ahead, I will open with an introduction to the early months of life as a business owner.

Here are the instant lifestyle changes I've experienced.

Goodbye, Lunch

I used to look forward to my glorious 45-minute daily lunch. I could always count on this welcome break around noon, when I would have time to eat, lounge around, read magazines or do a little shopping.

Those days are gone.

Don't get me wrong; I have a designated lunch hour. But sometimes it's filled with all sorts of walk-in patients—some needing an urgent consult, others needing a frame adjustment, still others picking up their glasses. So now, I eat when I can. Some days I get a real 30-minute lunch, but more often than not, I wonder where that hour went.

Goodbye, Sleep

I used to sleep through the blare of the alarm clock. Now? Not so much. And no more nights of sleeping like a baby. My circadian rhythm is dictated by unfinished business from the day before. In these last few months, my eyes instinctively open at exactly 6 AM, and my mind automatically runs through the day's checklist.

My day used to end at 5:30 PM. Now it just … well, it doesn't end. At the end of my workday, I turn into a DIY lumberjack/painter/interior designer/accountant/whoever else I need to be that day.

Goodbye, Vacation

Snowboarding in Canada? Sure! Zipline in Mexico? Sure! Once upon a time, it was ridiculously easy to do anything and everything in my heart's desire. All I had to do was request vacation in advance.

I used to go on so much vacation, my friends wondered if I worked. But I've had to say goodbye to spontaneous vacation (for the foreseeable future, at least). Practices are fragile during doctor transitions; now is not the time for snowboarding or zip-lining.

So there you have it. When you get your own practice, the first few things you need to give up are lunch, sleep and vacation.

Lots of people have asked how I'm doing. My answer? There's so much information being thrown my way in every direction, perhaps too much. I'm taking in what I can, when I can. I'm living and learning, one day at time. These days, I'm a lot like a duck—calm on the surface, but paddling like the dickens underneath.

Stay tuned! ■

The Power of Getting Personal

By Erin Kelly, Senior Associate Editor

When Dr. Tran first embarked on her OD career after finishing her residency, her main priority was putting her training into practice. She didn't want to be limited to refractions and glasses prescriptions; she wanted to be challenged. After more than two years in an ophthalmology setting, she realized she wanted something else, too—a personal rapport with her patients. Although she was busy and driven, she felt like she didn't know her patients as well as she should.

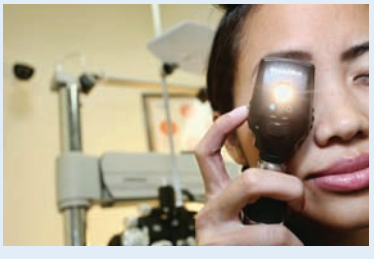
Today, she's getting what she asked for. As a business owner, she's challenged every day. As an OD, she sees patients for primary care, not just new glasses. And she's building relationships through it all.

"Dr. Susan Tran genuinely cares about her clients," says a Yelp reviewer from Temple City, Calif. "She also wants to get to know you, too."

Another reviewer writes: "My time with Dr. Tran was what a doctor/patient relationship should feel like. I have been a nurse for 10 years now (and) appreciate a good doctor when I see one. She sat there and made me feel like she was listening to my needs and didn't feel rushed."

Other comments:

- "[Dr. Tran] is upbeat and has such a friendly smile. She made small talk with me and got to know me personally before jumping into all the eye talk and procedures," says Denise D. "Within 10 minutes, I felt like she could be a friend. Most doctors are so mechanical and just want to see as many patients as possible in a day. Dr. Tran is different."
- "I've been to many optometry offices in the past and I've always felt like just another customer," says Johnny L., of Orange County, Calif. "Dr. Tran and her staff made me feel at ease and comfortable."
- Mike M. of Carlsbad writes: "Dr. Tran treated me like an old friend, not a new patient."



Keep up with Dr. Tran's progress by reading her new blog on www.revoptom.com.

Diagnostic Skills & Techniques

Pathology in Perspective:

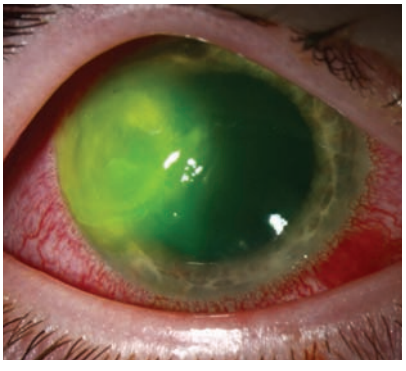
Know the Limits of Clinical Evaluation

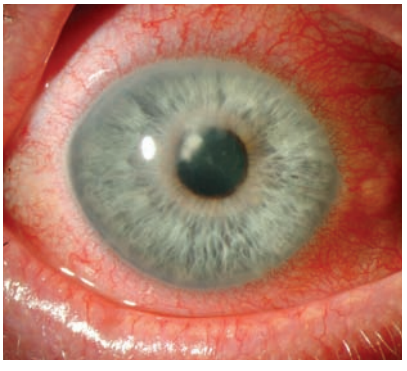
Looks can be deceiving. When exam findings are inconclusive, how can you make the diagnosis? Match wits with our experts in this unique self-test.

By Alan G. Kabat, OD, with photos and cases courtesy of Christine W. Sindt, OD

One of the great challenges in any health care profession is differentiating between similarly appearing conditions. Based solely on clinical appearance, it can often be quite difficult to definitively diagnose a particular lesion, finding or presentation. However, visual cues are typically our first indication that something is out of the ordinary; we formulate a differential and bolster our presumptive diagnosis by gathering further clues from the history, patient demographics and associated findings. Ultimately, we may need to rely on ancillary medical testing, such as pathology reports or radiographic imaging, to ascertain the true etiology of a condition.

In this article, we present a series of contrasting cases with similar findings and attributes. We ask you, the reader, to test your clinical acumen by answering a series of questions following each scenario. These questions are intended to represent key issues that a clinician may need to consider when encountering a similar situation. Following each case, a discussion highlights the most crucial concepts to know when facing these challenges.





CASE #1:

Above are two examples of patients who presented with acute red eyes. Both have a long history of contact lens use, and both were wearing their lenses when their eyes became red. Each patient reports pain, photophobia and excessive tearing in the involved eye.

Questions

1. Based upon the history and presentation, what can we assume about the etiology of these conditions?

a. The patient on the left has microbial keratitis; the one on the right does not.

b. The patient on the right has microbial keratitis; the one on the left does not.

c. Both have microbial keratitis, although the organism is likely different.

d. Neither has microbial keratitis.

2. The patient on the left is an otherwise healthy male in his mid-twenties. He was seen in your office Monday morning. He claims that he had no symptoms at all until late Friday evening, and that this all developed over the weekend. Given this information, what etiology must you suspect?

a. Bacterial infection, possibly *Pseudomonas*.

b. Corneal abrasion while sleeping.

c. *Acanthamoeba*.

d. Mooren's ulcer.

3. The patient on the right is a 48-year-old female with controlled hypertension. She is an avid gardener and claims that her symptoms began after getting some

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debris in her eye about a week ago. She experienced initial scratchiness and discomfort, which improved slightly but then got progressively worse. Given this information, what etiology must you suspect?

a. Corneal foreign body.
b. Fungal infection.
c. Herpes zoster infection.
d. Disciform keratitis.

4. Which of the following therapies would potentially be of benefit in BOTH of these cases?

a. Oral acyclovir.
b. Topical besifloxacin.
c. Subconjunctival triamcinolone.
d. Amniotic membrane (e.g., ProKera).

Answers

1) c; 2) b; 3) b; 4) d.

Discussion

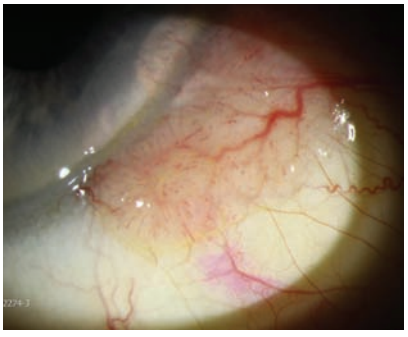
Both patients have microbial keratitis. The patient on the left has a bacterial keratitis of pseudomonal origin; the patient on the right has fungal keratitis secondary to *Fusarium*. Both were definitively identified after corneal scraping and culture in the appropriate medium. While bacteria can be detected within a day or two, fungal cultures may take up to a week to demonstrate growth. Contact lens wear enhances the risk of microbial keratitis, and should be considered any time a patient presents with an acute red eye. Anecdotally, young males with poor contact lens care habits demonstrate more frequent bacterial ulcers. While fungal keratitis is far less common, a history of prior vegetative injury is a common element.

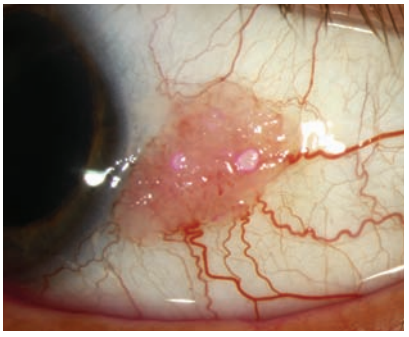
Acanthamoeba remains a potential etiology in virtually every early case of keratitis; however, it is still considered to be a rare occurrence by comparison. Also, the history of *Acanthamoeba* is typically slow and insidious, with persistence despite multiple therapies. Other cases of corneal trauma—such as foreign body, abrasion and recurrent erosion—usually are evident upon inspection; unlike microbial keratitis, these entities commonly present with a notable absence of corneal infiltrate.

Different microbial entities respond to different antimicrobial agents. Acyclovir is the prototypical antiviral agent used to manage herpetic infections, although there are several topical agents available (e.g., trifluridine, ganciclovir). A wide range of topical antibiotics are commercially available for the management of bacterial ocular infections, although only ciprofloxacin, levofloxacin and ofloxacin are FDA approved for keratitis.

Corticosteroids remain a controversial management option in cases of microbial keratitis and should be used judiciously, even by those with great experience.

A relatively new therapeutic modality—a sutureless amniotic membrane suspended in a plastic ring—has been shown to provide antimicrobial, anti-inflammatory and anti-angiogenic therapeutic elements that help to promote corneal healing. It is FDA approved for use in virtually all forms of microbial keratitis, and can be used in conjunction with topical and oral medications.





CASE #2:

Above are two patients who presented with chronic unilateral redness for several months. Both are white males in their early 70s; neither patient reports significant discomfort or visual impairment. During examination, the patient on the right displayed staining of the circumscribed lesion with rose bengal while the patient on the left did not.

Questions

1. Based upon the history and presentation, what can we assume about the etiology of these conditions?

a. The patient on the left has a malignant lesion; the one on the right does not.
b. The patient on the right has a malignant lesion; the one on the left does not.
c. Both patients have malignant lesions, although likely of different cell origins.
d. It is impossible to definitively determine a lesion's malignancy without biopsy.

2. Assuming the conjunctival lesions above are malignant, what diagnosis would be most plausible?

a. Squamous cell carcinoma.
b. Merkel cell carcinoma.
c. Amelanotic melanoma.
d. Kaposi's sarcoma.

3. What common, benign lesion of the conjunctiva is often initially suspected of

malignancy in elderly patients?

a. Squamous cell papilloma.
b. Phlyctenule.
c. Pinguecula.
d. Pterygium.

4. What course of action is most warranted for these individuals?

a. Photodocument and reassess in three months.
b. Initiate tobramycin/loteprednol QID and reassess in two weeks.
c. Refer promptly for excisional biopsy.
d. Refer for cryoablation at the patient's convenience.

Answers

1) d; 2) a; 3) a; 4) c.

Discussion

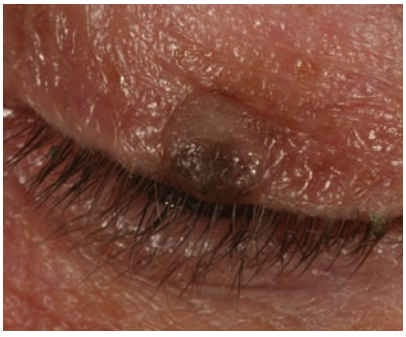
Newly identified lesions—or neoplasms—of the ocular surface must always warrant attention and consideration because of their potential for malignancy. This is especially true in patients over the age of 70. Unlike infectious or inflammatory disorders, ocular surface neoplasms often present insidiously and without significant discomfort or visual compromise. They may simply be noticed as an "unsightly bump" on the conjunctiva by the patient or another individual.

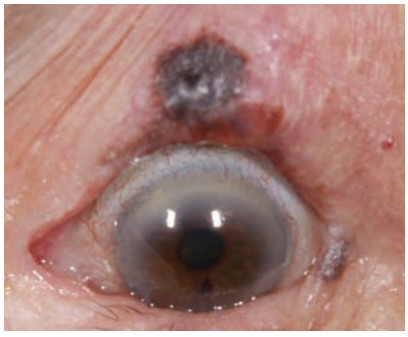
Many ocular surface neoplasms look similar, including those that are perfectly benign, such as papillomas, pingueculas and pterygia. Even malignancies exist as a continuum, from the borderline carcinoma-in-situ to the most severe invasive squamous cell carcinoma. Hence, there is no way to definitively identify a suspicious conjunctival lesion based on appearance or instinct. Definitive diagnosis requires a biopsy.

While both of the patients shown can be said to have neoplasms, only the patient on the right was diagnosed as having a malignant squamous cell carcinoma. The patient on the left was shown on biopsy to have a benign squamous cell papilloma—a common though often misdiagnosed conjunctival lesion of the elderly.

Key factors that should prompt greater urgency include two or more dilated conjunctival "feeder vessels," leukoplakia (i.e., a patchy, whitish keratosis overlying the lesion which stains with rose bengal) and a history of rapid growth. All of these are considered red flags for squamous cell carcinoma, and all were noted in the patient on the right. Interestingly, a history of other forms of skin cancer does not necessarily dictate the disposition of an ocular lesion.

As mentioned, any suspicious lesion of the conjunctiva—especially in an elderly patient—should be referred promptly for excisional biopsy to confirm its etiology. Photodocumentation may be used to help initially differentiate the lesion or to monitor for changes in lesions known to be benign, but should not be considered a definitive form of management. Likewise, topical therapy with antibiotics and/or corticosteroids is often used initially when clinicians are unsure of the diagnosis, but this should be avoided as it can lead to patient complacency and non-compliance when the condition fails to resolve.





CASE #3:

Both patients shown above presented with unilateral, pigmented lesions of the upper eyelid. The patient on the left noticed the lesion slowly progressing during the last four to five months; the patient on the right was referred by her primary care physician due to her "suspicious bruise."

Questions

1. What common historical element might be anticipated in both patients?

a. Injections of onabotulinumtoxinA for cosmetic enhancement.
b. Atopic dermatitis with eczema.
c. Chronic or excessive exposure to ultraviolet radiation.
d. Elevated serum cholesterol and lipids.

2. The patient on the left is a 68-year-old female who vacations frequently in South Florida, where she is an avid golfer and boater. She has noticed the lesion on her left upper lid developing during the last year. Upon inspection, you find similar, smaller lesions on her hands, scalp and ears. What is the *least* likely presumptive diagnosis?

a. Actinic keratosis.
b. Basal cell carcinoma.
c. Sebaceous cell carcinoma.
d. Seborrheic keratosis.

3. The patient on the right is an 88-year-old white female who lives in the mid-western United States. She has advanced Alzheimer's disease and cannot give an accurate history. A family member claims that the "bruise" on her upper lid was noticed about two weeks ago without any known trauma. Which of the following is *not* a red flag for potential malignancy?

a. Associated madarosis.
b. Non-uniform color and shape.
c. Location on the upper eyelid.
d. A satellite lesion at the outer canthus.

4. You determine that biopsy is necessary to definitively diagnose these lesions. To whom should this patient ideally be referred?

a. A board-certified oncologist.
b. A board-certified dermatologist.
c. A board-certified ophthalmologist.
d. A board-certified ocuoplastic surgeon.

Answers

1) c; 2) c; 3) c; 4) d.

Discussion

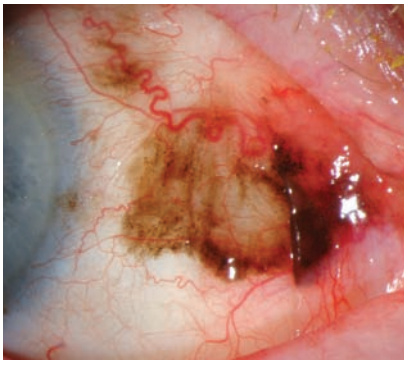
Patients of advancing age are more prone to the development of both benign and malignant skin lesions. Papillomas, keratoses, keratoacanthomas, xanthelasma, carcinomas and melanomas are all seen in the elderly population with much greater frequency. Many of these conditions can trace their development to increased ultraviolet exposure, especially in fair-skinned patients. This includes a variety of malignancies (e.g., basal cell carcinoma, squamous cell carcinoma and melanoma), precancerous lesions (e.g., keratoacanthoma and actinic keratosis) and benign lesions (e.g., seborrheic keratosis). Interestingly, sebaceous cell carcinoma does not appear to be influenced by UV radiation or race.

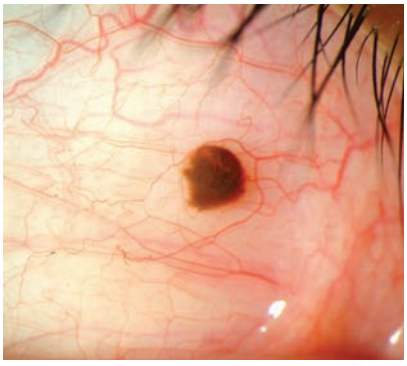
The patient on the left has actinic keratosis, a precancerous lesion with a predilection for sun-exposed area of the face, scalp and hands. Typically, these lesions do not exist in isolation; inspection of other affected regions can help to narrow the diagnostic spectrum. They are relatively slow to arise, often developing over six to 12 months before they are detected or prompt patients to seek treatment.

Established red flags for cutaneous malignancy include such elements as abrupt changes in size, shape or elevation; irregular borders; predilection toward bleeding or scab formation; variability in coloration; and presentation at multiple locations (e.g., "satellite lesions"). When dealing with eyelid lesions, the localized or generalized loss of lashes—termed madarosis—is another important consideration that may portend malignancy.

As with conjunctival lesions, any suspicious lid lesion warrants biopsy evaluation, particularly when it is encountered in an at-risk patient. While many physicians may be able to perform a biopsy, eyelid lesions ideally require the technique be performed by a qualified and experienced oculoplastic specialist. This is because of their knowledge of the intricate and delicate elements of the eyelid itself, and the extremely important role the lid plays in protecting the ocular surface.

Further, in cases of confirmed malignancy, it is valuable to identify a surgeon who is proficient in performing Mohs micrographic surgery, which is the preferred technique for removing cutaneous malignancies and carries the best prognosis for complete eradication without recurrence.





CASE #4:

Above are two examples of patients with pigmented conjunctival lesions. On the left is a 79-year-old white female with a history of cutaneous melanoma. On the right is a 34-year-old white male in otherwise good health. Both report mild foreign body sensation in the affected eye and stable vision.

Questions

1. Based upon the history and presentation, which patient warrants greater concern and urgency?

a. The 79-year-old woman on the left.
b. The 34-year-old man on the right.
c. Both require prompt intervention.
d. It is impossible to determine the level of urgency without biopsy.

2. What factors help to distinguish malignant conjunctival melanoma from

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benign conjunctival nevi?
a. Dilated conjunctival "feeder vessels."
b. Multiple areas of involvement.
c. Variability of color within the lesion.
d. All of the above.

3. What factors help distinguish racial melanosis from benign conjunctival nevi?
a. Predilection for fair-skinned patients.
b. Unilateral involvement.
c. Tendency toward a circumlimbal presentation.
d. Notable at birth or early childhood.

4. Ultimately, what treatment is typically reserved for patients with invasive conjunctival melanoma?
a. Localized conjunctivectomy.
b. Enucleation or exenteration.
c. Focal irradiation.
d. Systemic chemotherapy.

Answers

1) a; 2) d; 3) c; 4) b.

Discussion

Pigmented lesions, whether on the lid or conjunctiva, always warrant consideration and investigation. In general, round, regular, uniformly colored lesions of small size and long duration carry the lowest risk, while large, irregular, variably pigmented lesions of rapid progression are most suspicious.

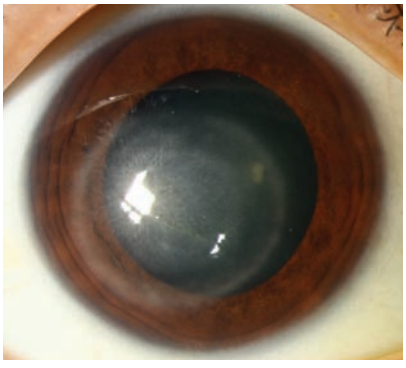
The patient on the left was found to have conjunctival melanoma; in all, eight locations were biopsy-positive, including samples from her superior fornix and right lower lid. This underscores the importance of searching for "satellite lesions" when suspecting melanoma. This is an extremely aggressive form of cancer when it involves the eyelids or conjunctiva. Though therapies including local excision, radiation or chemotherapy may be employed, most cases ultimately involve enucleation or exenteration.

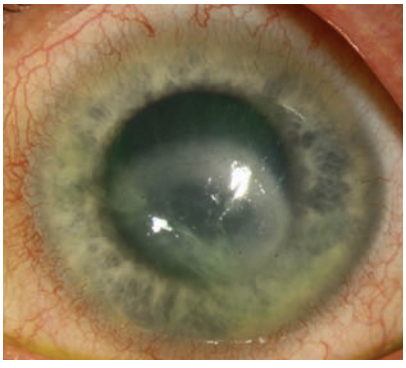
Conjunctival nevi are usually solitary lesions with distinct borders, as exhibited by our patient on the right. The color may range from pale yellow to dark brown, and can sometimes contain clear cystic lesions. Small, isolated nevi of long duration that have remained unchanged do not necessarily warrant aggressive action. Photodocumentation and periodic monitoring may be sufficient, but realize that in a very small percentage of patients, conjunctival nevi can undergo malignant transformation to become melanomas.

It is critical to differentiate nevi and melanomas from other forms of conjunctival pigmentation. Melanosis is a common finding in many practices. Dark-skinned individuals often display benign racial melanosis from an early age, which is generally bilateral and characterized by flat, even conjunctival pigmentation, often in a circumlimbal fashion. Acquired melanosis developing later in life can be more ominous.

Primary acquired melanosis (PAM) presents as flat, patchy conjunctival lesions, appearing unilaterally and typically occurring in fair-skinned patients. It has poorly defined margins and is usually located near the limbus. As many as 75% of conjunctival melanomas are believed to arise from PAM.

As with atypical lid lesions, should there be any doubt or concern regarding the disposition of a conjunctival lesion, excisional biopsy is warranted.





CASE #5

The two patients above presented for evaluation. The patient on the left has been having problems with her cornea "off and on" for several years, but recently relocated and cannot follow up with her regular doctor. She complains of reduced vision, mild discomfort and redness toward the end of the day. The patient on the right reports having steadily worsening redness, decreased vision and pain for the last month. She was treated with medication by another doctor but feels that there has not been any real progress. She wants a second opinion.

Questions

1. The patient on the left, a 35-year-old female, remembers that her previous doctor told her she had "herpes of the eye." Her first episode was in 2003, and her most recent event was six months ago. She cannot recall the name of her medication, but claims that it was very expensive and the solution had to be used many times each day.

Based upon this, what drug do you sup-

pose might have been prescribed?

a. Moxifloxacin.
b. Acyclovir.
c. Trifluridine.
d. Loteprednol.

2. Upon examination, the patient on the left displays central corneal haziness at the level of the stroma, with a few keratic precipitates. Instillation of vital dyes reveals sporadic punctate staining with sodium fluorescein and none with rose bengal. What do you suspect is her diagnosis?

a. Herpes simplex epithelial keratitis.
b. Herpes simplex disciform keratitis.
c. Herpes zoster keratitis.
d. *Acanthamoeba* keratitis.

3. The patient on the right is a 48-year-old contact lens wearer. Coincidentally, she also indicates that she had been treated for a herpes eye infection by her prior doctor. She was prescribed Zirgan (ganciclovir ophthamic gel, Bausch + Lomb), which she used five times per day. However, since her condition did not improve, you suspect that:

a. She likely has some other type of microbial keratitis.
b. The medication she received was probably expired.
c. She has developed a resistant strain of the herpes virus.
d. She requires a corticosteroid in addition to the antiviral therapy.

4. Based upon your suspicions, what action would you take next?

a. Obtain scrapings and cultures and confocal microscopy of the lesion.
b. Start the patient on a new sample of Zirgan after checking the expiration date.
c. Start the patient on trifluridine 1% nine times daily and add acyclovir 400mg five times daily.
d. Start the patient on trifluridine 1% four times daily and add prednisolone acetate 1% eight times daily.

Answers

1) c; 2) b; 3) a; 4) a.

Discussion

The herpes virus can cause a wide range of ophthalmic presentations, and both herpes simplex and herpes zoster can be implicated. Classically, herpes simplex keratitis (HSK) presents as a dendritic epitheliopathy that stains centrally with fluorescein and peripherally with rose bengal. HSK may be painful initially, but with recurrent attacks, patients develop corneal hypoesthesia; although the eye may become red and inflamed, there is little subjective discomfort.

HSK responds very favorably to both topical and oral antiviral medications. Viroptic (trifluridine 1%, GlaxoSmithKline), Zirgan and acyclovir are all extremely effective in arresting these infectious outbreaks. Topical acyclovir is only available outside the US, but the oral form can be used in lieu of topical medications at a dosage of 400mg five times daily for 10 to 14 days.

After the initial herpes simplex infection, it is possible to develop a noninfectious, cell-mediated immune reaction which stems from antibody/complement cascade against retained viral antigens in the cornea. Termed disciform keratitis, the condition presents with diffuse stromal edema that is almost invariably confined to the central cornea. Keratic precipitates and changes at the level of the endothelium may also be noted. This is the condition of our patient on the left. While disciform keratitis is often treated concurrently or prophylactically with antiviral medications (especially if the epithelium is compromised), the primary therapy required in this condition is a corticosteroid. Topical 1% prednisolone acetate (or equivalent) dosed every two to four hours is an excellent initial choice.

As mentioned, ocular herpes can take on a variety of presentations, many of which mimic other disorders. The patient on the right displays a large, ring-shaped infiltrate in her central cornea. But, according to her records, the earliest clinical findings included a dendritiform lesion—prompting her physician to initiate antiviral therapy. Further delving into the case revealed a history of contact lens wear and a corneal injury prompted by vegetative matter. These components, combined with the ring infiltrate and recalcitrant epithelial defect, must prompt the clinician to consider *Acanthamoeba*, a soil-borne pathogen that is often misdiagnosed upon initial presentation.

Acanthamoeba is a slowly progressing organism and will rarely present as a classic ring infiltrate. Therefore, it is not unusual for other, more common organisms to be suspected and treated first. A true classic finding of *Acanthamoeba* that may help in early suspicion of the organism is perineuritis, where the patient's pain is disproportionate to the findings. *Acanthamoeba* can only be definitively identified with scrapings and cultures; however, confocal microscopy may yield the pathognomonic cysts when scrapings are negative. Most doctors now start therapy based on confocal findings.

Acanthamoeba is a very challenging organism to eradicate, typically requiring multiple drug therapy. While some antibiotics (e.g., polymyxin, trimethoprim-sulfamethoxazole) and antifungals (ketoconazole, miconazole) may be helpful, antiviral agents appear to have little impact on the course of this corneal infection. Unfortunately, unless suspected and treated early, many cases of *Acanthamoeba* keratitis ultimately necessitate penetrating keratoplasty. ■

How to Provide Special Treatment for Exceptional Children

As the ACA provides eye care for previously uninsured families, you may see more young patients with special needs. Are you prepared to meet this demand?

By Thomas Wilson, OD, FCOVD

The Affordable Care Act (ACA) potentially will cover millions of previously uninsured families and guarantee eye care to their children. This means that many of these children—some of whom have special needs—could find their way into your practices for routine eye exams.

An inefficient visual system can significantly interfere with any child's ability to learn, but is especially debilitating for the approximately 10.2 million children in the US with language delays, autism and other developmental disorders. Visual disorders can be devastating to a child with special needs when the primary care optometrist entrusted with their care lacks the tools to diagnose and treat their visual problems.

With the help of some basic pearls, we can perform an exceptional, comprehensive examination on these young individuals. Also, this opportunity can provide a great deal of professional satisfaction in ensuring the visual capabilities necessary for the child to flourish.

This article reviews how you can more effectively provide vision care to children with special needs. Additionally, it offers several short case reports that underscore the fundamental importance of thorough pediatric examinations.

Setting the Stage

Preparing your office for a special needs child requires some due diligence from your staff. Many of these kids have complicated histories and are on multiple medications. In addition, they may have transportation issues that slow them down on the day of their appointment. Here are a few tips that can make the first visit easier:

- Have the "Welcome to the Office" form filled out before the visit.
- Encourage the child to bring a favorite toy or stuffed animal to have its eyes checked.
- Create a questionnaire to determine which problems may exist. Consider having the staff ask the child's parents these questions over the phone before the exam.
- Make sure the caregivers know how to get to your office.
- Invite the patient to meet the staff and get acquainted.
- When appropriate, have the staff confirm the appointment and talk with the patient to convey how much they are looking forward to having him or her come in to the office.
- Make sure to note special interests, and make light conversation about those topics to keep the child engaged and attentive.

The Examination

Kids can sense your anxiety level, so it is imperative that you enter the room relaxed and with a clear mind. Never enter the room feeling rushed or burdened.

There are many ways to examine a child with special needs. The first thing to consider is that time is of the essence. Too many preliminary tests can fatigue the patient. Your one-on-one time is invaluable; so, it

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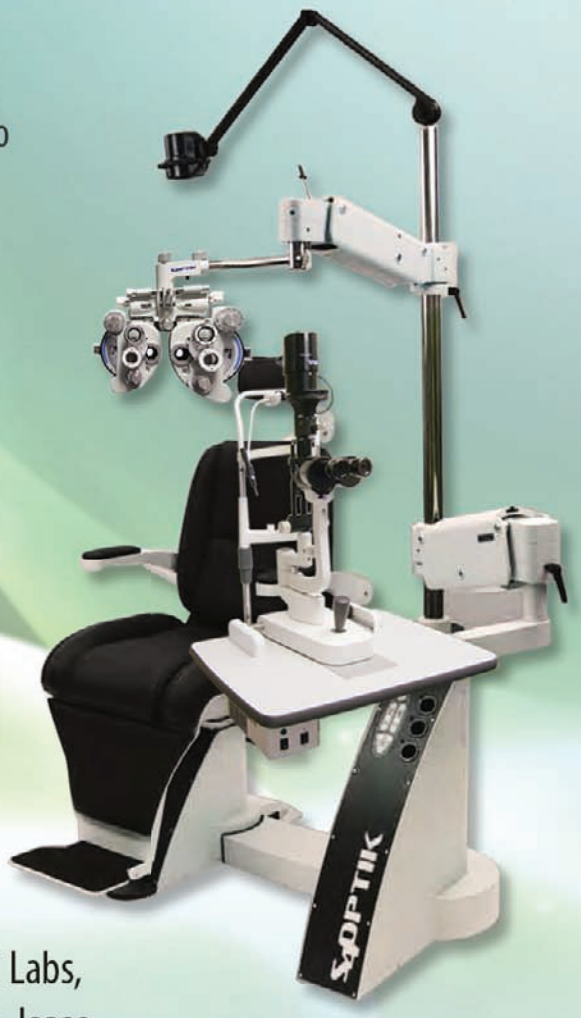

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may be wise to limit tech time and elicit the most pertinent information while the patient is receptive. Also, make an effort to document the most significant parts of developmental history, including ApGar scores, language skills and reading levels prior to the visit.

Keep in mind that if you walk into the room and try to be someone you're not, it could turn around to bite you. Think about going into a room with your brother or sister and trying to be Dr. Nice Guy if you aren't. It would seem out of character to them, and it won't go far with these kids either.

• ***Visual acuity*** testing, if attainable, is an invaluable part of the examination. It's not as important for you as it is for the parents. They often want to know what the child sees—even if it's an approximation.

LEA grating paddles can be used in a variety of ways to estimate visual acuities. It is possible to obtain an accurate acuity assessment on an uncommunicative individual by changing the distance and rotation of the gratings. An off-label variation of this product is similar to the preferential looking test. Using matching symbols tests can also be very effective. Most of the available computer-generated charts allow you to isolate symbols for accurate distance visual acuities.

• ***Binocular vision*** assessment can be performed easily, quickly and accurately with a Worth 4-dot test. One simple and effective modification is to have the child touch the dots. The examiner can tilt the red-green glasses and see which dots the patient touches. Using the reflection in the glasses can prove invaluable not only for evaluating suppression, but also when testing for concomitance in trophic cases.

• ***Motilities*** can be evaluated rapidly using the Heidi fixation targets. The smiling face on the paddle is a natural pull for any visual system. A Brock string with a fixation target, such as a sparkly bead found at most craft stores, also will attract the child's attention and can allow you to quickly assess convergence skills. The LEA Face Disposable Occluder (Good-Lite Co.) can be used to accomplish this, and also may be given to the child to keep, as a form of motivation and positive reinforcement.

Additionally, FixiStix produces Wiggle Pictures fixation targets that can be very mesmerizing for any child. Many times, the best targets are found in your giveaway toy box. Using targets with sound or lights often triggers fixation.

• ***Cover testing*** is usually best performed with a transilluminator. Fixation typically is reliable with a point source of light, especially if it hasn't been used until this point in the examination. The key is using monocular light fixation and the angle kappa as objective means of determining eye deviations, combined with prism neutralization.

Using a 6- to 8-power prism and watching the angle kappa can provide insight to binocularity and fixation. Instead of employing individual prisms, use a horizontal and a vertical prism bar to significantly reduce the time needed to determine the angle of deviation. Prism bars also can be extremely helpful when trying to neutralize diplopia.

• ***Refraction typically*** needs to be performed without the phoropter for children with special needs. Take note that standard of care dictates a cycloplegic refraction, as well.

The Smart System PC Plus (M&S Technologies) offers an excellent short video with red and green balloons. This provides a good target for distance fixation and also works as a quick screening for color when the child is able to respond.

Near retinoscopy is an invaluable tool in prescription determinations. A very effective near target is the press-on Flashing Fixation Stars (Good-Lite Co.). These provide a colorful stimulus with a center bright spot that allows for detailed near fixation.

Another technique for determining accommodative lag is to have the child perform natural Harmon distance tasks. In this instance, ask the child to play at their natural near working distance while performing near retinoscopy.

If you determine that a distance or near prescription is needed, trial frames and lens flippers can demonstrate the effectiveness of the prescription to both the patient and the parent. Loading one side with a "dummy" prescription and the other with the actual prescription allows the patient to react favorably to their new vision.

Facial expressions, recognition of details in objects of interest and vocalization are all cues to success.

The child's responses to "real" and "dummy" prescriptions also can alleviate anxiety for the parents regarding whether the glasses will make a difference. Remember—to the parents—it is a bit magical that you have come up with the correct prescription without actually asking, "Which is better, one or two?"

It's important to prescribe with respect to anisomoptropic demands to allow for equal accommodative demands between the right and left eyes. It's also helpful to prescribe full plus for esotropia and high esophoria, and minimum plus for exotropia and high exophoria.

High plus prescriptions do not tend to go towards plano, and most of those prescriptions level off when the patient reaches 18 months of age.[1]

• ***Visual fields***, while necessary, can be difficult or even impossible to perform. Remember that fields must be taken monocularly. Standard visual field testing for special needs patients requires more time and attention than routine screening fields. Frequency doubling technology (FDT) has become a standard in many practices for rapid, reliable fields measurements. However, FDT is not reliable on most kids under the age of eight—much less on those with special needs.

One effective approach is the monocular fixation in a binocular field (MFBF) test. Red and green glow stick flashlights can be purchased at many grocery and hardware stores.

For the MFBF technique, first place red-green glasses on the patient. Activate the green flashlight directly in front of the patient to use as a fixation target, while the red flashlight is deactivated in the patient's lower right visual quadrant. Then, once you activate the red light, the patient will respond. Set the red flashlight to flash, which will cause the patient to respond again.

The response can be a nod, noise or any other indication that there has been a change. Repeat this in the other three quadrants of the patient's visual field. Once the process is completed, repeat the test using the red flashlight as the fixation target and the green flashlight as the visual field stimulus.

Another way to assess visual field integrity and acuity is via optokinetic nystagmus testing.

It is important to note that the nystagmus reflex does not usually develop until the patient has reached six months of age in infants with normal development. Also, this technique should only be applied as an adjunctive screening tool in addition to acuity or visual field testing.

Correction Prescribing Tips

We know that children at the highest risk for amblyopia development are those with high aniosometropia and/or esotropia. It's also important not to interfere with the child's natural emmatropization process when prescribing corrective lenses.

For a variety of reasons, special needs children have an increased risk of amblyopia. They experience less visual stimulation from reading and computer work, and their underdeveloped neurological systems exhibit a predilection for esotropias and amblyopias. So, it's essential to avoid amblyogenic risks whenever possible.

Additionally, new research is investigating the relationship between pediatric near visual demands and how much time is spent outside.

Emmetropic children with two myopic parents who spent five hours or less per week outside had about a 60% chance of becoming myopic. However, the probability was reduced to 20% for emmetropic children with two myopic parents who spent 14 hours per week or more outside. Children with one or even no myopic parents also benefitted from more time outdoors.

This research suggests that outdoor activity might be protective against both amblyogenesis and myopia development.[2]

General prescribing guidelines include correcting hyperopia greater than 5.00D, myopia greater than 8.00D and astigmatism greater than 2.50D. It is also important to minimize amblyogenic anisometropia by prescribing for hyperopia greater than 1.00D, myopia greater than 3.00D or for astigmatism greater than 1.50D.

Also, be certain to remember that children with Down syndrome and cerebral palsy frequently do not accommodate well.

A general rule: When in doubt, prescribe if kids are behind or developmentally delayed. It's important to give them an advantage, especially when they have a hyperopic correction. A 2008 study published in *Archives of Ophthalmology* indicated that baseline visual motor integration scores are significantly more reduced in children with hyperopia than those with myopia.[3]

In simple terms—it's important to get what you can get when you can get it. You may need to have the patient return on another day to accurately assess the visual system, provide an accurate diagnosis and create a realistic treatment plan. Sometimes, it's helpful to intentionally avoid referring to refractive data from a previous encounter as a way to confirm your suspicions.

Most of these kids are familiar with going to the doctor, which can work to your advantage; however, they may be unfamiliar with the fancy equipment in your exam room. It may be helpful to meet them first or just say "hello" when they are playing with toys in the waiting room.

One final suggestion: Have fun with these truly special kids. You will learn as much from them as they will from you. ■

Dr. Wilson is in private practice in Colorado Springs, Colo. He has worked with special needs children for the last 27 years. He also is the coauthor of "SportsVision: Training for Better Performance."

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A Tale of Two Children

A specialist in neurodevelopmental vision describes the challenges, and rewards, of managing autism at work and home.

By Cathy Wittman, OD

Jeremy practicing visual rehabilitation techniques with his mother.

Children with developmental delays often have vision problems that affect their academic performance. Working with these children has helped me come to terms with my own son's autism spectrum disorder and find ways for him to reach and expand his potential.

My Patient, Aidan

When Aidan first came into my office last year, he seemed disconnected from this world. His mother explained that she and her husband had long suspected that he was on the autism spectrum. He had verbal delay and preferred to play alone. His parents had difficulty getting his attention, and he was afraid of loud noises. He did not enjoy physical affection, make eye contact or adjust well to changes in his daily schedule.

At age four, Aidan's teacher told his mom that, in order for him to color a page, she would have to pick out a crayon for him. The teacher reported that it seemed like all of the colors in the Crayola box were too overwhelming for Aidan to consider, and he did not know which one to choose.

Aidan's parents expressed concern to his regular pediatrician, who agreed that he likely had autism. His pediatrician then referred him to a developmental pediatrician, who also suspected an underlying learning disability.

In kindergarten, Aidan's problems continued. Thankfully, the school nurse recommended that he get an eye exam. So, his mom brought him to my office.

I diagnosed Aidan with refractive amblyopia of his left eye and prescribed spectacles. Within eight weeks, his parents noticed that his listening skills improved and that he was making more eye contact. We began vision therapy to improve the acuity in his amblyopic eye and eliminate suppression, as well as to work on the visual skills he needed to succeed in school and life.

My Son, Jeremy

When I think of Aidan, I'm reminded of my own son, Jeremy. When he was Aidan's age, Jeremy displayed many of the same characteristics. Jeremy had verbal delay, difficulty making eye contact and a sensory integration disorder. Perhaps we were in denial, but we thought he would outgrow it.

By the time Jeremy was seven or eight, it became obvious that he was on the spectrum. Over time, his sensory integration disorder became more of a problem. One time, we had to leave Rain Forest Café in San Antonio because the flashing lights and noise overwhelmed him.

Jeremy also fixated on subjects that interested him. For example, he knew every Pokémon character, their behaviors, what they evolved into and what region they were from. (There are hundreds of them, and he knew them all.) His speech patterns also clued us in. It became apparent that he was not going to outgrow over-pronouncing his words or exhibiting the flat inflection that is characteristic of Asperger syndrome.

In fifth grade, Jeremy was officially diagnosed so that he could receive an individual education plan (IEP) at school. Jeremy is now in eighth grade, and as his peers have behaviorally matured into teenagers—he has not. He does well academically, but from a social perspective, he is well below grade level.

To help people empathize with him, I tell them to imagine putting the brain of an academically advanced eight-year-old into the body of a thirteen year old and expect that child to navigate the world of junior high. Middle school has been a challenge, and my husband and I keep homeschooling as an option, should things become too difficult for Jeremy.

A Growing Interest in Developmental Optometry

During the last few years, I've become increasingly fascinated by the study of neuro-developmental optometry. This interest began after I became good friends with a PhD candidate at Texas

Tech in the College of Education, whose special interest was traumatic brain injury. Together, we are intrigued by the principle of neuroplasticity and its possible application in visual training and rehabilitation.

Additionally, I've recently met several highly influential physicians and eye care professionals who have dramatically enhanced my understanding of autism's impact on the visual system.

For my blog (wittmanvision.com), I was able to interview pediatrician Janna Jennings, MD, of Benton, Ark., whose autistic son experienced dramatic improvement following vision therapy. Additionally, I met Nancy Torgeson, OD, FCOVD, who has a large vision therapy practice near Seattle that specializes in treating kids on the spectrum.

I've also had the opportunity to learn from Robert Sanet, OD, FCOVD and his wife Linda Sanet, COVT, of Lemon Grove, Calif. Both individuals are leading experts in behavioral optometry.

Then, at the 2013 Annual Neuro-Optometric Rehabilitation Association meeting in San Diego, I met Susan Daniel, OD. I ran into her on the deck of the conference center. We talked for an hour about her son, who has autism and was non-verbal for much of his life. Dr. Daniel informed me that, via specialized vision training, she was able to help improve her son's symptoms tremendously. So, this summer I took Jeremy to her office in Carlsbad, Calif. for an evaluation and to experience her program. She has the latest technology and techniques to help her patients with autism, as well as those with traumatic brain injury.

The last two years have been filled with many of these seemingly coincidental encounters. I can't help but take this as confirmation that this is where I should be focusing my career. Currently, I am interested in finding novel methods to help people on the autism spectrum, patients with traumatic brain injury and those with vision-related learning problems.

At times, I feel both guilty and saddened that we did not start working with Jeremy when he was Aidan's age. It would have been better for Jeremy and our entire family if we had addressed his issues earlier; however, I remain hopeful, because I've learned that neuroplasticity continues after childhood. In conjunction with my husband (who now works with children with autism for our local school district), I am trying many approaches to improve Jeremy's symptoms.

For Aidan, my goal is not just for him to be better connected to the world around him, but also to help him succeed in school and meet other goals he decides to set for himself. Helping young patients achieve better vision for their entire lifetime is one of the most rewarding accomplishments in my career, and I look forward to serving my community in this capacity for many years to come.

Dr. Wittman is in private practice in Lubbock, Texas.

Diagnostic Skills & Techniques

Tricky Retinal Masqueraders: Unmasked!

Chorioretinal conditions can mimic each other, resulting in diagnostic dilemmas.

By Mohammad R. Rafieetary, OD, and Eric J. Sigler, MD

Pathological alteration and destruction of biological tissue is due to a variety of etiologies. Whether infectious, inflammatory, autoimmune-related or traumatic, the presentations can have similar clinical findings. This often results in a diagnostic challenge, or even misdiagnosis. Duration of disease and chronicity may further contribute to the diagnostic dilemma, as long-standing disease or pre-clinical appearance may differ from our classically held assumptions regarding morphology.

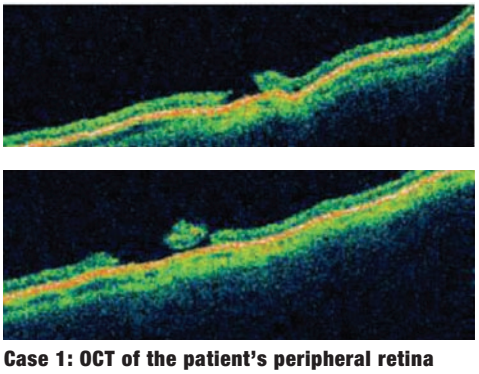
Here, we present some examples of retinal or chorioretinal disorders that may mimic each other. The approach to the patient should always begin with a basic, patterned routine for developing a differential diagnosis. In general, chorioretinal processes are considered primarily infectious, inflammatory, infiltrative, degenerative, dystrophic or idiopathic. Although the ophthalmoscopic appearance of many posterior segment diseases may be similar among these processes, careful examination of structural features—along with the use of current imaging—will help you differentiate most lesions.

Chorioretinal Atrophy: Inflammation vs. Degeneration

A number of degenerative, infectious, inflammatory, autoimmune conditions (e.g., the spectrum of "white dot syndromes") can result in chorioretinal atrophic changes and fibrosis (scarring) that may have a similar appearance, complicating accurate diagnosis.

• *Case 1* shows the progression of a patient with high myopia and pathologic chorioretinal atrophy. The ophthalmoscopic findings include peripapillary atrophy and linear chorioretinal atrophy corresponding to a lacquer crack. Over several years of follow-up, the patient developed choroidal neovascularization, which was successfully treated with anti-VEGF therapy.

This may be a challenging differential diagnosis, as patients with high myopia, particularly females, may develop white dot syndromes, such as multifocal choroiditis with panuveitis (MCP) and punctate inner choroidopathy. Differentiating between these lesions is best performed with SD-OCT. MCP lesions appear as eruptive pigment epithelial detachments and are *elevated* RPE lesions. Lacquer cracks and primary atrophic changes appear *excavated* on SD-OCT.



Case 1: OCT of the patient's peripheral retina shows operculated retinal break.

The presence of a cellular reaction in any ocular segment may be helpful but does not rule out chorioretinal inflammation, so we advocate a structural evaluation using SD-OCT.

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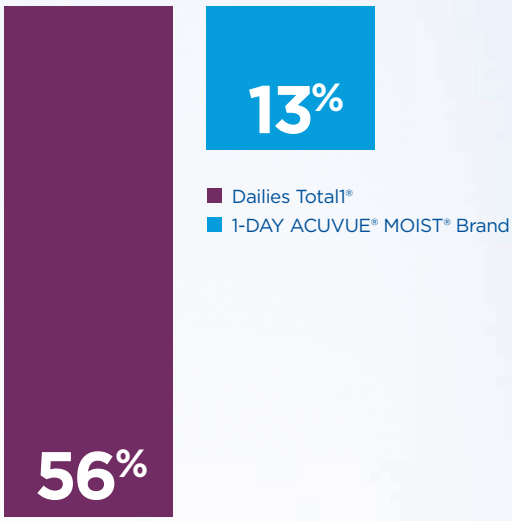

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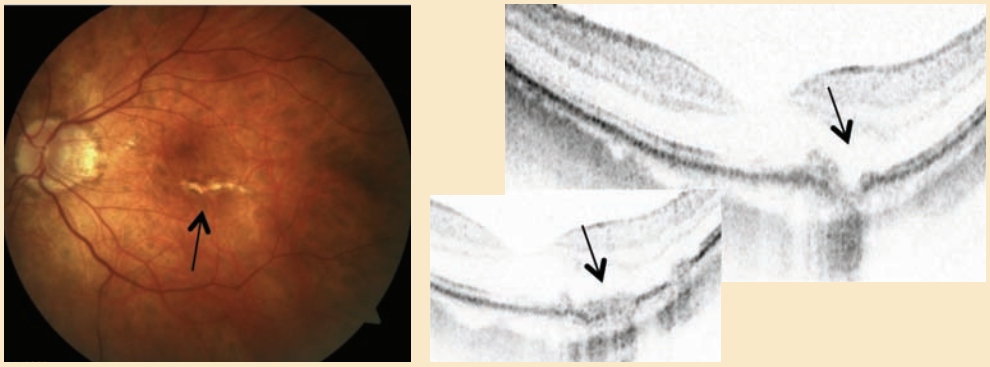
Case 1

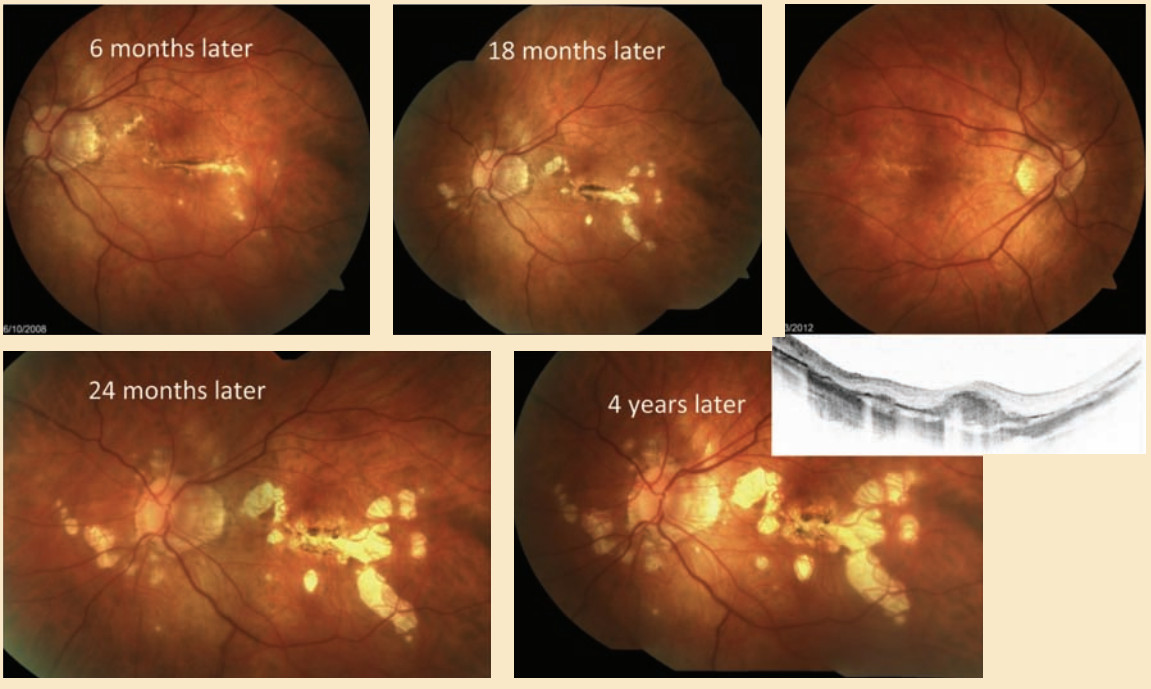
A 49-year-old white female initially presented in 2007 with an acute posterior vitreous detachment and a retinal tear in the left eye, for which she had focal photocoagulation. The patient's entering visual acuity was -7.00 20/20 OD and -10+0.75X150 20/40 OS.



Photograph shows the lacquer crack inferior to fovea. OCT shows views of the crack and CNV.

One year later, she returned with complaints of seeing a spot in the central vision of her left eye. At this point, her visual acuity with contact lenses was 20/30 OS. Examination revealed a juxtafoveal lacquer crack with small, associated choroidal neovascular membrane. She was treated with intravitreous bevacizumab.

Over the course of four years, the patient's left eye has exhibited progression of chorioretinal atrophy and recurrences of choroidal neovascularization (with as-needed treatment) and visual acuity of 20/200, while the right eye has had a stable lacquer crack.



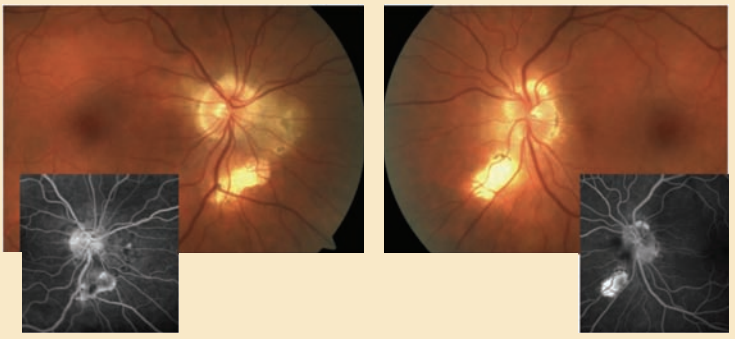
Serial photos show progression of degenerative myopia with extension of chorioretinal atrophy and Fuchs' spots. OCT corresponding to the last photo (*inset*) shows choroidal thinning, RPE and outer-retinal atrophy and juxtafoveal membrane.

• *Case 2* illustrates a presentation of serpiginous choroidopathy that may be confused with degenerative myopia or peripapillary choroidal neovascularization. In contrast to the lacquer crack and degenerative myopia, this lesion typically begins in the peripapillary region, with symptoms such as photopsia or enlarged blind spot. This patient is not highly myopic, which further narrows the differential diagnosis.

In contrast to atrophic or degenerative lesions, the lesions of serpiginous choroidopathy begin in the choroid, and appear as outer retinal and RPE whitening. SD-OCT may also be used for a structural differentiation, as the acute lesions of serpiginous choroiditis demonstrate

Case 2

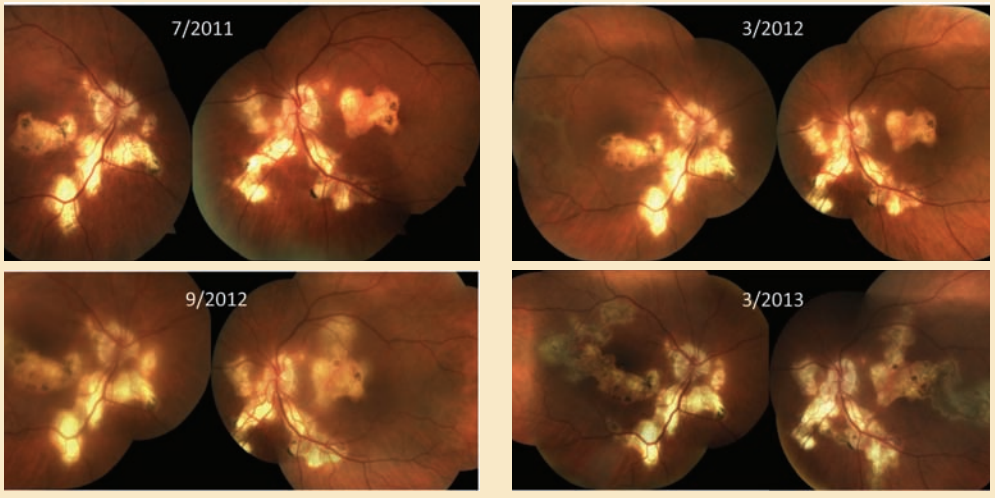
A 42-year-old white female presented in August 2007 for evaluation of peripapillary chorioretinal scars. She had no significant visual complaints and her uncorrected visual acuity was 20/20 OU. Examination revealed that the scars were inactive at that time. We asked the patient to return in two months for close observation; however, she did not come back to the office until a year later, with complaints of floaters.



After a year lost to follow up, this patient presented with peripapillary chorioretinal lesions OU. Fluorescein angiography OD showed mild leakage and staining of the lesion inferior to the ONH as well as minimal ONH leakage, while FA OS revealed only transmission defects.

Two years later, photos and fluorescein angiography show progression of the patient's chorioretinitis.

Visual acuity remained 20/20 OU. There was mild angiographic activity OD, and OCT of the area revealed RPE and outer-retinal atrophy. We diagnosed her with chorioretinitis; the differential included serpiginous choroidopathy. We referred the patient for a medical workup and asked her to return in six to eight weeks for close observation.



Serial photographs over three years show progression of the chorioretinal degenerative disease.

The patient returned two years later with complaints of vision loss OS (20/100; OD remained 20/20). There was significant bilateral progression of chorioretinal lesions, which confirmed serpiginous choroidopathy.

Over the next three years, the chorioretinal degenerative process has continued in spite of immunosuppressive therapy. At her latest exam, visual acuity was 20/30 OD and 2/200 OS.

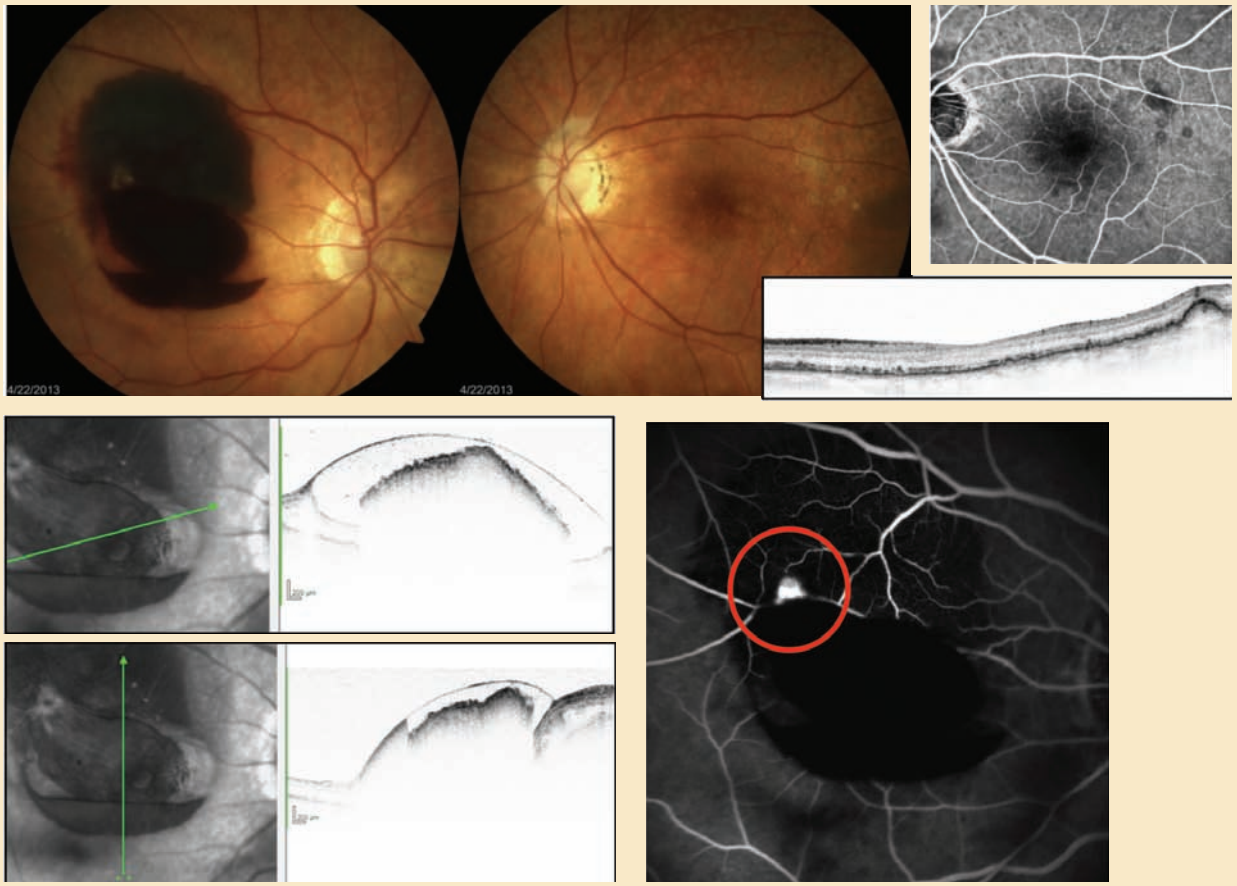
a diffusely thickened choroid, outer retinal disorganization and subretinal fluid. Nevertheless, the end result of both processes often presents with a similar clinical appearance, with both peripapillary and macular atrophy, often along with choroidal neovascularization.

Mixed Macular Hemorrhage: Choroidal vs. Retinal Vascular

Posterior segment hemorrhage may be due to retinal or choroidal blood vessels. Choroidal neovascularization is the most common cause of subretinal and sub-RPE bleeding. Retinal vascular disease, such as diabetic retinopathy and retinal vein occlusion, typically produce isolated intraretinal and vitreous hemorrhages in the absence of subretinal or sub-RPE blood. Few disorders include hemorrhage in all three layers—concomitant subretinal, intraretinal

Case 3

An 80-year-old white female was referred for evaluation of wet age-related macular degeneration OD. Her visual acuity measured 20/200 OD and 20/30 OS. Examination revealed a large combined macular hemorrhage OD, and multiple large drusen and pigment epithelial detachment OS. Angiographic study showed the cause of the hemorrhage as a retinal macroaneurysm.



Photograph of right eye shows mixed retinal hemorrhage. OCT illustrates that most of it is in a subhyaloid location. Drusen is visible in the photo of the left eye, and the OCT scan also shows pigment epithelial detachment. The reflective spot (in the red ring) shown on fluorescein angiography of the right eye is pooling of the dye by the retinal arterial aneurysm.

and vitreous (or preretinal) hemorrhage. These traditionally include retinal macroaneurysm, the polypoidal choroidal vasculopathy variant of macular degeneration, globe trauma and non-accidental infant trauma ("shaken baby" syndrome). Terson syndrome, or vitreous hemorrhage secondary to a subdural or subarachnoid hemorrhage, is an additional cause of hemorrhage in all three layers and is typically peripapillary. Therefore, the differential diagnosis of multilayer hemorrhage, particularly in a peripapillary location, must include intracranial processes.

• *Case 3* illustrates the appearance of a multilayer macular hemorrhage secondary to a ruptured retinal macroaneurysm. This 80-year-old patient, like many over age 65, has a clinical picture that at first glance is consistent with age-related macular degeneration. The presence of a subretinal hemorrhage in the context of adjacent drusen and drusenoid pigment epithelial detachment in the fellow eye is typically present in AMD.

However, the presence of a large area of preretinal ("sub-hyaloid") hemorrhage is atypical for AMD. The fluorescein angiogram reveals the true pathology to be an underlying retinal macroaneurysm, which is also a common finding in elderly patients with a history of hypertension, particularly those with previous retinal vein occlusion.

SD-OCT is again helpful in dif-

ferentiating these lesions and identifying the location of hemorrhage. The presence of sub-internal limiting membrane hemorrhage is typical of retinal macroaneurysm.

• ***Case 4*** provides an additional example of macular hemorrhage, but with isolated subretinal and sub-RPE hemorrhage. This patient exhibits many similar features as those discussed in case 3; however, examination and SD-OCT reveal the absence of preretinal or intraretinal hemorrhage, and the presence of a large pigment epithelial detachment likely indicates a choroidal neovascular process.

The differentiating features play a key role because these similar-appearing lesions have distinct clinical management strategies. Although retinal macroaneurysm may be treated initially with anti-VEGF therapy, observation until hemorrhage resolution is typically undertaken until focal laser photocoagulation can be performed, which leads to occlusion of the macroaneurysm and resolution of hemorrhagic and exudative potential. On the other hand, AMD is treated with ongoing anti-VEGF therapy.

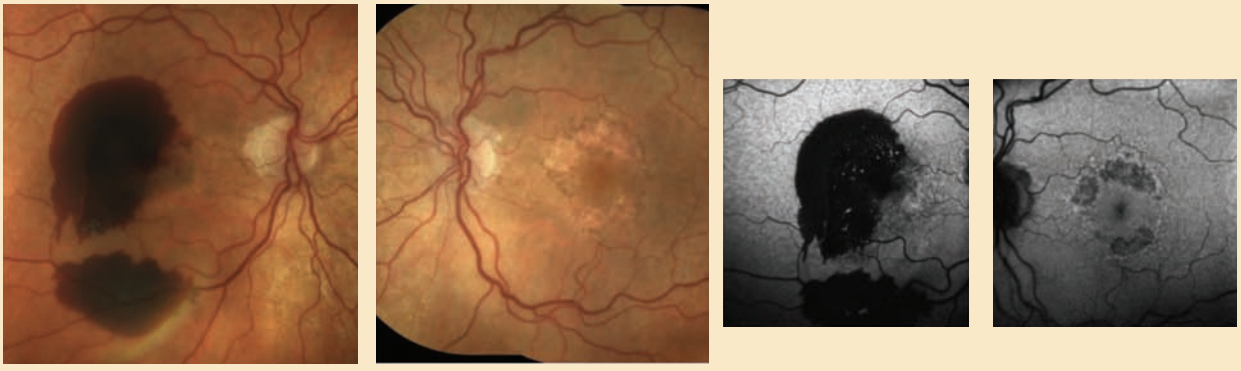
As eye care providers, we encounter a number of challenging cases with very similar presentations yet with different underlying etiologies. Cases presented in this paper highlight the variety of these challenging conditions. These examples also demonstrate how clinical findings combined with imaging, such as OCT, can aid in the proper diagnosis of these conditions. ■

Dr. Rafieetary is a consultative optometric physician at the Charles Retina Institute in Memphis. Dr. Sigler is a retina specialist at the Charles Retina Institute and an upcoming associate of Ophthalmic Consultants of Long Island, Division of Retina and Vitreous.

This article has been abridged. Read the full text of this article—with three more cases—on www.revoptom.com.

Case 4

An 86-year-old black female was referred with branch retinal vein occlusion and sudden vision loss OD. The patient (a non-smoker) has been treated for systemic hypertension for 20 years. Her visual acuity was 20/400 OD and 20/40 OS. Examination revealed a mixed macular hemorrhage that was mainly subretinal and sub-RPE in location. The fellow eye had evidence of atrophic age-related macular degeneration.



A large macular hemorrhage was noted OD. Geographic atrophy and drusen were visible OS.

OCT of the right eye shows subretinal and sub-RPE hemorrhage, with a large pigment epithelial detachment and subretinal membrane.

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Essential Uses of Radiologic Testing in Eye Care

Know the primary diagnostic applications of CT and MRI testing for a variety of sight-threatening conditions. **By Albert David Woods, MS, OD, and Michelle K. Caputo, OD**

With dramatic improvements in radiologic imaging technology during the last several years, we are now able to evaluate internal anatomical structures to a degree that was only dreamed about just a decade ago. Enhanced precision of the two primary imaging modalities used in clinical practice—computed tomography (CT) and magnetic resonance imaging (MRI)—has fundamentally changed how eye care providers both diagnose and manage many diseases that affect the visual system. As primary care providers, optometrists often are the first individuals to see patients who exhibit visual signs and symptoms of an underlying pathology that could potentially cause blindness or death.

Even with the sophisticated, high-resolution diagnostic techniques available today, imaging studies should be ordered judiciously after a thorough clinical examination. But first, it is essential to know the type of imaging study needed, the anatomic location where the study should be performed (i.e., brain, orbit or other body region), if contrast is indicated, whether angiography should be considered, and how urgently the results must be obtained and evaluated. Further, it is crucial to communicate any relevant clinical findings to the radiologist so that he or she can effectively localize the lesion and determine if any special imaging techniques are indicated.

Here, we provide guidelines to determine the precise imaging studies required, as well as the specific parameters within each of these studies, depending upon the patient's clinical presentation.

CT Scans

Computed tomography scanners use an X-ray source and a corresponding detector to measure the intensity of signal attenuation through various tissues of different densities. Following the scan, a computer readout presents the device's findings as pixelated images for evaluation. Denser structures such as bone are displayed as white; less dense structures such as brain and muscle tissue are displayed as gray; and empty air/liquid space is displayed as dark gray or black.

In the 1980s, CT scanners were

Release Date: January 2014
Expiration Date: January 1, 2017
Goal Statement: Computed tomography and magnetic resonance imaging have fundamentally changed how eye care providers both diagnose and manage many diseases that affect the visual system. Here, we provide guidelines to determine the precise imaging studies required, as well as the specific parameters within each of these studies, depending upon the patient's clinical presentation.
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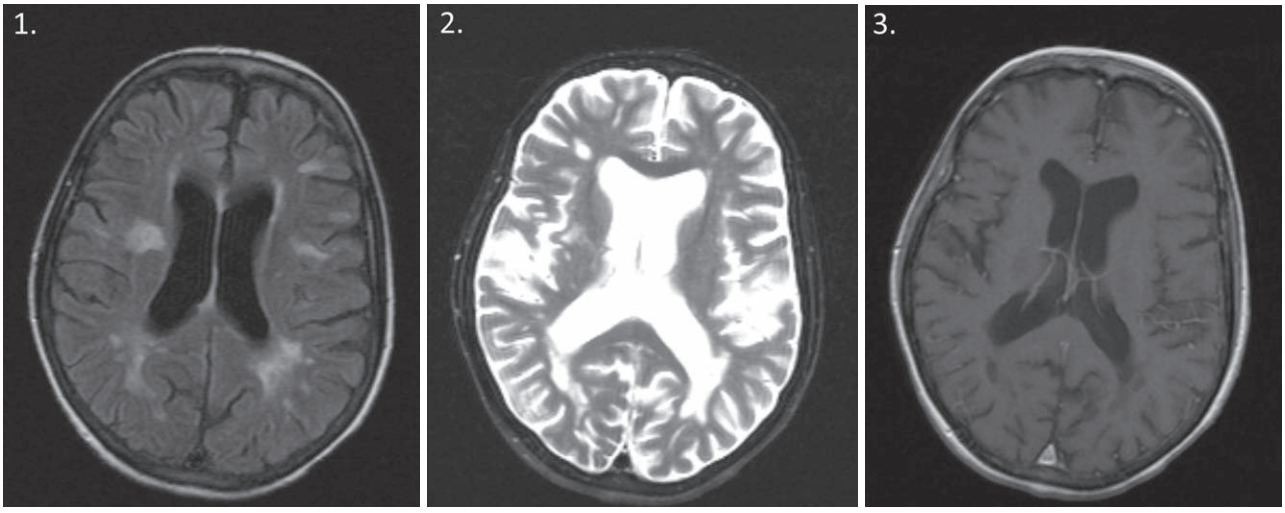


On MRI, this multiple sclerosis patient exhibited white matter lesions. Image #1 illustrates use of the fluid-attenuation inversion recovery (FLAIR) specialized sequence on a T2-weighted (T2W) scan; image #2 shows a standard T2W scan; and image #3 shows a T1-weighted (T1W) scan with the use of gadolinium contrast.

developed to continuously rotate the X-ray source/detector apparatus around the patient as he or she moved through the gantry.[1] Because the X-rays would spiral around the patient, this technique was referred to as a "spiral CT." The advantage of spiral CT is that the patient moves at a constant speed through the gantry, facilitating faster scanning, less radiation exposure, fewer body movement artifacts and the ability to create three-dimensional reconstructions of tissue structures.

It is important to know that the CT scanner captures different angles and slice thicknesses, depending on whether it is imaging the orbit or brain. So, when ordering a CT, be sure to indicate which area should be scanned.[2]

Because CT devices employ X-rays, you must exercise extreme caution when referring pregnant patients or children for imaging studies. In such instances, an MRI may be warranted to avoid exposing such individuals to any ionizing radiation.

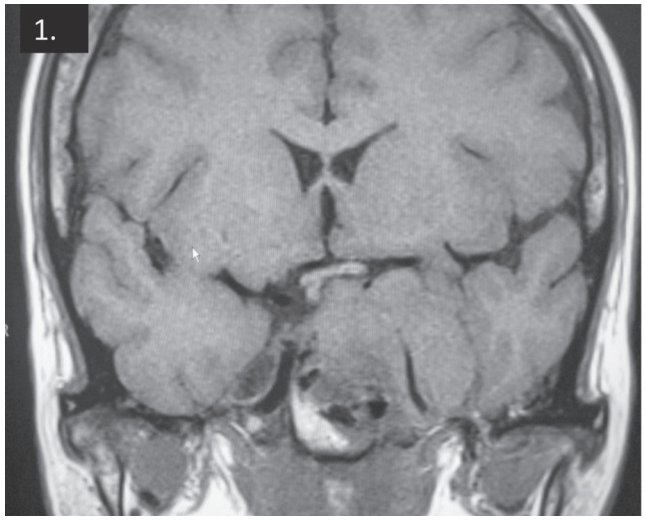
If you or the radiologist is considering the use of contrast, you must ascertain whether the patient has a documented history of shellfish allergy or has exhibited a prior reaction to any iodinated contrast. Contrast should not be ordered in patients with renal failure, or in those considered to have an acute intraorbital or intracranial hemorrhage (which can obscure the view of an underlying lesion).[3,4] Finally, avoid the use of contrast in patients with suspected thyroid eye disease (TED), because the iodinated material may worsen the systemic symptoms.[5]

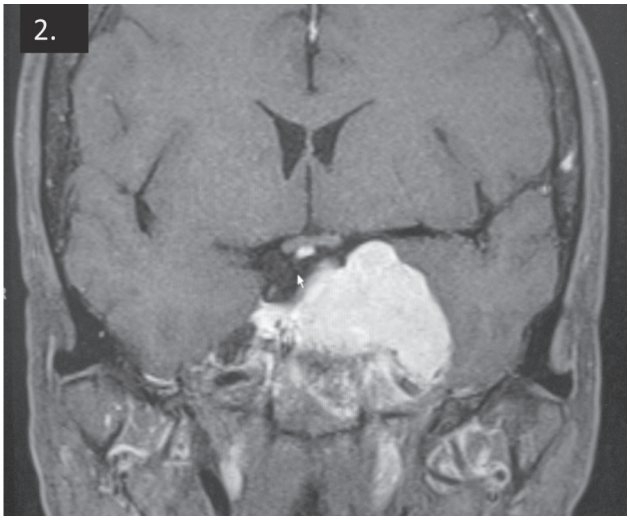
Magnetic Resonance Imaging

While CT imaging uses conventional X-ray technology, magnetic resonance imaging combines the use of the hydrogen atoms inside the body with an intense magnetic field to create images without the consequence of radiation exposure.

An MRI requires a series of large magnets to generate a magnetic field around the patient while the protons of the patient's hydrogen atoms align along the magnetic field. Radiofrequency (RF) coils deliver a pulse that causes the hydrogen protons to "resonate." Following the RF pulse, the hydrogen atoms cease resonation and realign along the magnetic field. In doing so, each hydrogen atom emits a radio wave of its own during this realignment, or "relaxation time." The scanner uses this feedback to pinpoint where each nucleus is. It also allows the scanner to identify the tissue composition of the various structures of the body to create a mapped image.

- *T1-weighted (T1W)* images have a shorter time sequence for the RF pulse and relaxation time, which yields sharper images and are best suited to evaluate pertinent anatomy. T1W images cause the fat signal to be bright and the fluid signal to be dark. Because fatty tissue appears bright on T1W imaging, orbital T1W scans should be taken with fat suppression (short TI inversion recovery, or STIR), which enhances visualization of orbital structures, gadolinium-enhanced lesions or associated inflammation.
- *T2-weighted (T2W)* images use a longer time sequence that causes fluid to be bright, so it's useful for detecting edema. Although certain pathologies may be easier to see on T2W images, the longer acquisition time may yield slight blur secondary to random body movement.





These scans clearly illustrate the importance and clinical utility of gadolinium contrast. Image #1 shows a coronal T1W MRI of a meningioma without contrast, and image #2 shows a coronal T1W MRI of a meningioma with contrast. Note the dramatic enhancement.

• *Fluid-attenuation inversion recovery (FLAIR)*, one of the more common specialized sequences ordered in eye care, is applied in conjunction with T2W imaging. With FLAIR, any free water signal such as cerebrospinal fluid (CSF) appears dark, while water associated with pathologic edema remains bright. FLAIR sequences are particularly useful in diseases like multiple sclerosis (MS), where demyelinating lesions around the periventricular area can be hidden by the hyperintense signal of CSF in the ventricles on standard T2W imaging.

• *Diffusion weighted imaging (DWI)*, another specialized sequence used in eye care, is sensitive to the random motion of water molecule protons, known as the apparent diffusion coefficient. Any particles in free water exhibit random movement (Brownian motion). When cytotoxic edema occurs (e.g., in patients with an acute ischemic infarct), there is restriction of water diffusion in the extracellular space, which appears as a bright signal. DWI can reveal such abnormalities within minutes of the ischemic event, which is significantly earlier than on CT or conventional MRI.[6]

• *Gadolinium contrast* can be used with MRI T1W scans to highlight areas of tissue where the blood-brain barrier has been compromised. It's particularly useful to enhance the visibility of blood vessels and brain tumors.

Take note that gadolinium contrast is contraindicated in patients with renal and/or liver disease, or in those who've recently received a liver transplantation, because it can lead to an incurable, potentially fatal disease—nephrogenic systemic fibrosis.[7]

Additional contraindications for MRI scans include the presence of metallic intraocular foreign bodies, cardiac pacemakers, aneurysm clips (unless they are made of titanium) and cochlear implants. However, intraocular lens implants are not contraindicated for MRI.

Clinical Indications and Recommended Scan Protocols

Acute Vision Loss

In a patient with acute vision loss, you must topographically localize the offending lesion. To a significant degree, this will depend on whether the loss is unilateral or bilateral. If it is unilateral vision loss, the focus of the imaging studies should be from the optic nerve back to the optic chiasm. If the vision loss is bilateral, then the focus of the imaging studies should be on the chiasm and post-chiasmal visual pathway. In bilateral vision loss, ischemia is a common underlying cause (unless bilateral optic neuritis is suspected).

For acute vision loss, MRI imaging is the study of choice, and should include T1W with both pre- and post-contrast scanning combined with fat suppression/STIR, along with T2W scanning. Also, the MRI scans should include both orbital and brain images. If you suspect a demyelinating disease, FLAIR should be included. Additionally, DWI should be added if an ischemic event is suspected.

Optic neuritis is one of the more common causes of unilateral acute vision loss. It typically is seen in young adults, and often manifests with pain upon eye movement if the retrobulbar portion of the optic nerve is involved. During the acute stage of optic neuritis, orbital contrast-enhanced T1W imaging with fat suppression will show nerve enhancement. Additionally, T2W and FLAIR brain imaging should be

included when looking for periventricular white matter in demyelinating lesions.

A finding that is strongly associated with MS on MRI imaging is Dawson fingers, which are demyelinating plaques that are arranged at right angles to the lateral ventricles.[8] The detection of white matter lesions on MRI imaging has been considered a prognostic risk for developing MS in a patient with an optic neuritis. However, the Optic Neuritis Treatment Trial showed that even in the absence of white matter lesions, the risk of MS is still present, and that 25% of patients without initial white matter lesions still developed MS at 15-year follow-up.[9]

In patients with bilateral acute vision loss who present with homonymous hemianopia, the most common etiology usually is ischemic in nature. Therefore, MRI with DWI should be ordered. While most chiasmal conditions presenting with bitemporal hemianopsia are slowly progressive due to mass lesions, a sudden vision loss or visual field progression may be a sign of hemorrhage associated with a pituitary apoplexy, and should be referred for urgent CT or MRI imaging without contrast.[10]

Other exceptions to ordering an MRI for acute vision loss include TED patients, those who exhibit optic nerve compression from enlarged extraocular muscles or in cases of traumatic optic neuropathy (ON). In these instances, initial imaging should include both orbital and brain CT scanning.

Progressive Vision Loss

Several pathologies can cause unilateral vision loss at the level of the orbit, including TED, optic nerve and orbital tumors (e.g., optic nerve glioma and optic nerve sheath meningioma), and low-grade inflammatory or infiltrative disorders.[11]

While the primary imaging modality for suspected tumors is an MRI, a CT can provide better delineation of any bony involvement, or more accurately indicate if any calcification is present within a mass. And while CT is commonly used to rule out optic nerve compression in TED patients, an MRI can be used to detect mild interstitial edema within the extraocular muscles during the early disease stage if there are questionable findings about the diagnosis.[12]

When patients present with bilateral progressive vision loss, the etiology often is a slowly expanding mass. The more common tumor types that impact the chiasm include pituitary adenomas, meningiomas and craniopharyngiomas. Non-tumor lesions can include internal carotid artery aneurysms and dilatation of the third ventricle. MRI scanning, both with and without contrast, often will reveal the underlying pathology.

Elevated Optic Nerve Head

A common unilateral cause of an elevated optic nerve head (ONH) is non-arteritic ischemic optic neuropathy (NAION). While neuroimaging usually isn't performed on NAION patients, an orbital MRI with contrast can help in specific instances when the diagnosis is uncertain because of multiple overlapping clinical findings. In patients with ON, optic nerve enhancement typically is present and tends to impact the entire intraorbital optic nerve to some degree. In NAION patients, however, enhancement usually is either partial or completely absent.[13] (Keep in mind that there are many instances where only partial optic nerve enhancement is observed in ON patients.)

Bilateral (and sometimes unilateral) elevated ONHs can be seen in patients with optic nerve head drusen, infiltrative ON and papilledema. In most instances, B-scan ultrasonography can accurately document ONH drusen; however, questionable cases can be confirmed with tight 1.0mm cuts on CT scanning, which often reveals areas of calcification.[14]

While MRI can help diagnose infiltrative ON, additional systemic and cerebrospinal fluid analysis with opening pressure is needed to determine the underlying etiology. In patients with papilledema, the MRI will determine if there is an underlying pathology, such as intracranial tumors, hemorrhages, meningeal disease, hydrocephalus, ventricular stenosis or the formation of a fistula. A magnetic resonance venogram (MRV) also should be ordered to rule out dural venous sinus thrombosis as a cause of elevated intracranial pressure.

The absence of the aforementioned pathological abnormalities in a patient with increased intracranial pressure points to a diagnosis of idiopathic intracranial hypertension (IIH). Several findings observed on MRI are indicative of IIH, including an empty sella, globe flattening, venous sinus compression and dilation of perineuronal subarachnoid space surrounding the optic nerves.[15]

CN Palsies

Cranial neuropathies involving CN III, IV or VI are imaged using MRI with pre- and post-contrast. A recent development in MRI sequence is constructive interference in steady state, which permits high-resolution imaging of smaller lesions within the cranial nerve.[16]

Because of the underlying risk of an aneurysm in patients with a third nerve palsy, particularly those who exhibit pupillary involvement, you should follow an urgent protocol that includes both structural imaging and angiography. The recom-

mended combination is either CT/computed tomography angiography (CTA) or MRI/magnetic resonance angiography (MRA).[2] Although CTA involves injection of a venous iodinated contrast agent, it can uncover aneurysms less than 3mm in diameter with greater sensitivity and rapidity than MRA.[17]

While CT/CTA or MRI/MRA are commonly the initial studies performed with pupil-involved third nerve palsies, catheter angiography is the gold standard for detecting cerebral aneurysms. A catheter angiography is particularly useful when suspicion remains high for a possible aneurysm after negative CTA or MRA findings.

Sixth nerve palsies also carry a risk of significant morbidity, primarily due to their association with underlying mass lesions. While the majority of acute sixth nerve palsies are ischemic in nature, an MRI with and without contrast should be ordered in patients with no documented history of diabetes or hypertension, and/or if the palsy does not improve, shows signs of progression or features additional neurological involvement.[18]

Horner's Syndrome

Clinically, this condition is characterized by ptosis, miosis, a dilation lag and greater anisocoria in the dark. In recently acquired Horner's syndrome (HS), imaging studies often are required to uncover an underlying pathology, such as ischemia, tumor, demyelination and arteriovenous malformations.

In pharmacologically confirmed cases of HS, you should order an MRI with and without contrast of the head and neck down to the level of the second thoracic vertebra (T2).[6] In HS patients with shoulder or arm pain, Pancoast syndrome with a tumor at the lung apex should be ruled out via use of contrast-enhanced axial CT of the upper chest.[19] In patients who present with acute HS accompanied by head or neck pain and possible altered taste sensation (dysgeusia), an internal carotid artery dissection should be ruled out by T1W MRI with contrast and fat suppression, as well as an MRA of the neck.[20]

Proptosis

When a patient presents with the globe(s) protruding anteriorly, the most common etiology is TED. However, a multitude of other conditions can cause proptosis, including masses (both primary and metastatic), inflammatory states (e.g., idiopathic orbital inflammatory syndrome, Wegener's granulomatosis, systemic lupus erythematosus), vascular complications (hemorrhage, arteriovenous abnormalities) or infections (e.g., periorbital cellulitis).

The recommended initial imaging for the evaluation of a new onset proptosis is CT scanning without contrast of the orbit and sinuses.[2] Often, CT imaging should be followed by an orbital MRI with contrast and fat suppression to obtain more detailed images. If the lesion extends intracranially, a head MRI is also indicated. For vascular lesions of the orbit, or a cavernous sinus thrombosis where secondary clinical findings may include ptosis and CN III, IV, V and VI palsies, additional imaging may include T1W and T2W MRI with FLAIR, MRA or CTA.[21,22]

Hemifacial Spasm

This condition results from involuntary contractions of the muscles innervated by the ipsilateral facial nerve. While the most common etiology of hemifacial spasm (HFS) is dilichoectatic vessel compression where the facial nerve exits the pons, space-occupying lesions such as pontine glioma, demyelination or an ischemic occlusive event must also be excluded. To rule out compressive or ischemic lesions, an MRI should be focused on the course of CN VII as it exits the brainstem and travels in close proximity to the auditory canal.[23] Further, an MRA can confirm the presence of vascular compression at the nerve root as it exits the pons, or whether a surgical decompression procedure may be required.[24,25]

Orbital Trauma

Significant orbital trauma is most commonly seen in association with motor vehicle accidents or physical assaults, and usually presents clinically with periorbital ecchymoses and subconjunctival hemorrhages. In orbital fractures, the inferior wall is most commonly involved, followed by the medial wall. While orbital fractures can be followed for several weeks before surgery, a rare condition after trauma called orbital compartment syndrome (OCS) requires urgent imaging and management.

Post-traumatic retrobulbar hemorrhage is the most common cause of OCS, which leads to a marked increase in orbital pressure, vascular compromise of the globe and optic nerve, and possible irreversible blindness. Findings consistent with OCS include ecchymosis defined by the orbital rim with tense eyelids, very limited globe motility, decreased vision, afferent pupillary defect and markedly elevated IOP.[26]

Emergent orbital decompression (e.g., lateral canthotomy) is usually performed first, followed by CT of the orbit with direct coronal views. With most current CT imaging systems, the resolution is sufficiently detailed so that coronal views are reconstructed from the axial cuts; however, with direct coronal views, the patient's head is tilted back to precisely image the coronal plane.

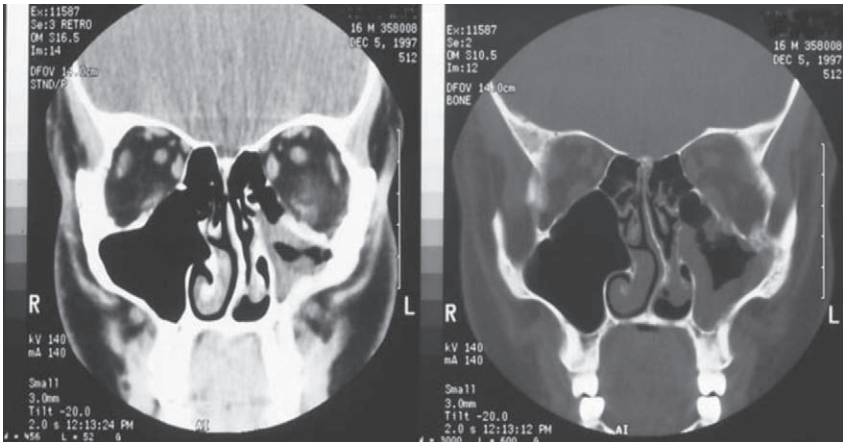
Be aware that there may not be significant visible signs of trauma in

children with orbital floor fractures (i.e., a "white-eyed" blowout fracture).[27] Also, in pediatric patients, the orbital floor has a tendency to "greenstick," where the fractured bone bounces back to entrap the inferior rectus muscle (whereas the fracture is more likely to remain down in adults).

Additional findings with orbital fractures are limitation of motion with associated diplopia, crepitus, ptosis and enophthalmos. Axial and coronal CT imaging with contiguous cuts of 1.5mm to 3.0mm is usually recommended. Using a CT protocol called bone window—where the imaging of soft tissue is reduced within the CT to focus primarily on the bone—can help uncover small fractures and breaks with muscle entrapment.

Up to 5% of orbital trauma patients may also have a traumatic optic neuropathy.[28] Associated clinical findings typically include reduced visual acuity, visual field loss, afferent pupillary defect, and reduced bright light and red color perception. Optic nerve damage can result from direct injury secondary to a penetrating object, bone fragments, vibrational transmission from trauma, shearing forces at the optic canal, or compression from a hematoma or edema within the nerve.[29] CT scanning should include axial and coronal cuts of the orbit and head, with 1.0mm cuts through the optic canal to the skull base.

As optometry has become an integral part of the health care system, it is important that we recognize the indications for radiological studies and take the necessary steps to refer these cases to experts who can accurately interpret the tests and begin the process of repair. Keep in mind that an imaging study simply is another tool to help confirm a suspected diagnosis based on the patient's clinical presentation. After the results of the imaging study are reviewed, you should be able to correlate the findings to the patient's clinical signs and symptoms and confidently make a diagnosis. ■



This patient presented with an orbital floor fracture. We referred the individual for both standard coronal CT (left) and CT with the specialized bone window protocol (right). Notice that the bone window scan shows enhanced evidence of fracture and muscle entrapment.

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Dr. Caputo is the staff optometrist of the neuro-ophthalmology service at the Bascom Palmer Eye Institute in Miami.

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OSC QUIZ

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1. All are advantages of spiral CT, *except*:
a. Rapid scanning.
b. Fewer body movement artifacts.
c. No radiation exposure.
d. Ability to create three-dimensional reconstruction.

2. Patients with which condition should NOT undergo CT scanning with contrast?
a. Shellfish allergy.
b. Acute intraorbital hemorrhage.
c. Acute intracranial hemorrhage.
d. All of the above.

3. In regard to MRI studies, which specialized imaging sequence is best for viewing anatomy?
a. T1W.
b. T2W.
c. DWI.
d. FLAIR.

4. Which protocol associated with MRI scanning reduces the fat signal intensity to better facilitate viewing of orbital contents?
a. FLAIR.
b. STIR.
c. DWI.
d. Gadolinium contrast.

5. The best specialized imaging sequence for detecting white matter lesions in a multiple sclerosis patient is:
a. FLAIR.
b. DWI.
c. T1W.
d. ADC.

6. A patient presents with a history of kidney failure and a pacemaker. Following clinical examination, you suspect that he may have a brain tumor. Which scan should you order?
a. MRI of the brain and orbits with gadolinium.
b. MRI of the brain and orbits without gadolinium.
c. CT of the brain and orbits with contrast.
d. CT of the brain and orbits without contrast.

7. A 40-year-old patient presents with a sudden onset of unilateral vision loss in her right eye and associated pain upon eye movement. You suspect a diagnosis of optic neuritis. What is the best confirmatory imaging study?
a. CT scan of the orbits with contrast.
b. MRI scan of the orbits with fat suppression and gadolinium.
c. MRI of the brain with gadolinium.
d. CT of the brain.

8. The diagnosis of optic neuritis was confirmed following the scan that you selected for the 40-year-old patient outlined in question #7. Now, what scan should you order to evaluate for multiple sclerosis in this individual?
a. T2W MRI of the brain.
b. MRI of the orbits with gadolinium.
c. CT of the brain with contrast.
d. CT of the orbits with contrast.

9. A new onset right homonymous hemianopsia and right-sided weakness was found in a 73-year-old patient. Which specialized imaging sequence would be best suited to reveal an acute onset stroke?
a. T1W.
b. T2W.
c. FLAIR.
d. DWI.

10. Which imaging study could potentially confirm the diagnosis in NAION suspects?
a. MRI of the brain.
b. MRI of the brain with contrast.
c. MRI of the orbit.
d. MRI of the orbit with contrast.

11. Which imaging study will most effectively help you visualize a venous sinus thrombosis?
a. MRI with FLAIR.
b. MRV.
c. MRA.
d. MRI with DWI.

12. Which imaging technique is most sensitive for the detection of a small, 3mm aneurysm in a pupil-involved third nerve palsy?
a. CTA.
b. MRA.
c. MRV.
d. MRI of the orbits.

13. A patient presents with an acute, painful, pupil-involved third nerve palsy and no history of trauma, diabetes or hypertension. An aneurysm was not detected via CT or CTA. What is the best next course of action?
a. Nothing needs to be done, as no aneurysm was detected.
b. Catheter angiography.
c. Repeat the CT in three weeks.
d. Refer to a primary care physician for a medical evaluation.

14. Palsies of which cranial nerve carry a significant risk of morbidity due to their frequent association with underlying mass lesions?
a. CN III.
b. CN IV.
c. CN V.
d. CN VI.

15. If dysgusia is present in a patient who

OSC QUIZ

presents with recently acquired Horner's syndrome, the scan of choice is:
a. CT of the brain.
b. MRI of the brain.
c. MRI and MRA of the neck.
d. MRA of the brain.

16. What is the most common cause of proptosis?
a. Tumor.
b. Arteriovenous fistula.
c. Thyroid eye disease.
d. None of the above.

17. What imaging study is best suited to uncover the underlying etiology in patients who present with hemifacial spasm?
a. CT.
b. CT and CTA.
c. MRI and MRA.
d. MRV.

18. Which finding is NOT typically seen in a patient with an orbital floor fracture?
a. Diplopia.
b. Exophthalmos.
c. Periorbital ecchymoses.
d. Ptosis.

19. What imaging scan should you order order if you suspect an orbital floor fracture?
a. CTA.
b. Bone window CT.
c. B-mode ultrasound.
d. T1W MRI.

20. Which finding is NOT consistent with a traumatic optic neuropathy?
a. Decreased visual field.
b. Decreased visual acuity.
c. Decreased blue color perception.
d. Afferent pupillary defect.

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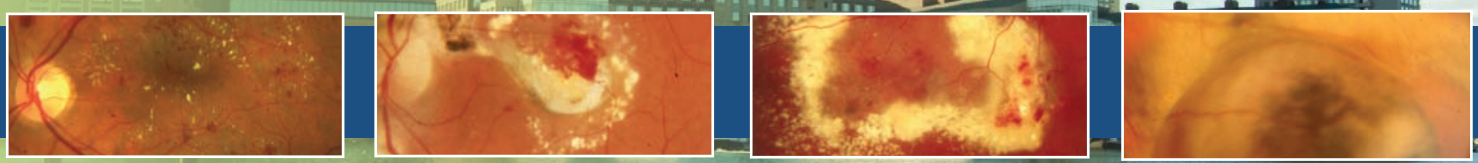

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Glaucoma: When to 'Say Uncle'

Seek a second opinion when the patient shows progression or isn't compliant—or simply if you feel in over your head. **Edited by Paul C. Ajamian, OD**

Q I feel comfortable treating most glaucoma, but when a patient's compliance is questionable or the field defects seem to fluctuate over time, I get a bit nervous. When should I "say uncle" and send these patients for a second opinion?

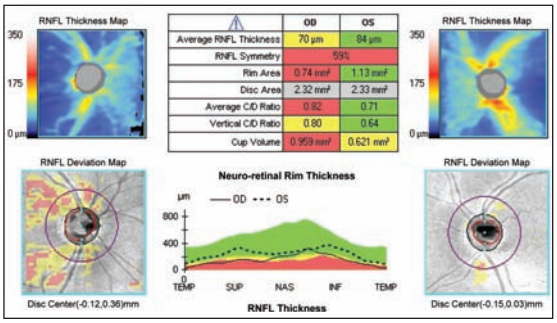
A First, relax and have confidence in yourself and your care. "Remember that glaucoma is a slowly progressing disease, so there's rarely such a thing as a glaucoma emergency," says Robert Vandervort, OD, director of ocular disease services for Heartland Eye Consultants, in Omaha. Be sure to confirm any progression with a series of visual fields or OCT scans.

That said, it's smart to "say uncle" when:

• **You're outside of your comfort zone.** "Every optometrist and ophthalmologist has their own comfort level for treating and managing glaucoma," Dr. Vandervort says. "If you're at all uneasy about how your patient is doing, then absolutely get input."

That doesn't necessarily mean: *when in doubt, refer it out*. Because it usually progresses slowly, "glaucoma is a disease that really works well for collaboration," he says. A phone call for a second opinion or advice on target IOP might be all you need.

• **The patient shows progression while on therapy.** "When you have progression occurring while the patient is on therapy and you can document the progression over a period of six months to a year, then you need to get additional input or refer that patient out," Dr. Vandervort says. "And, if the patient has a lot of advanced nerve damage to begin with, that'd be a much shorter window."



OCT may show glaucomatous damage earlier in the disease process than visual fields.

Signs of progression include the development of optic nerve disc hemorrhages or any noticeable changes in visual field, OCT, stereo fundus photos or optic nerve head appearance.

"Recent studies have shown that OCT is more likely than visual fields to pick up glaucomatous damage earlier in the disease process," Dr. Vandervort says.[1] "So, by the time you start picking up visual field loss, you probably have already seen significant damage of the optic nerve." If OCT shows definite progression regardless of visual field loss, get a second opinion, he says.

Another concerning sign: high variability of IOP with or without optic nerve progression damage.[2] "If a patient's IOP is fluctuating from visit to visit by 10mm Hg—or even 6mm Hg to 8mm Hg—on medication, even without progression, I would get additional input," he says.

• **The patient is not compliant.** "We know that when you add a second or a third IOP-lowering medicine, the patient's compliance goes down," Dr. Vandervort says. "And those second-line meds are at BID and TID dosing. So, now you've got not only another medicine but more drops per day. The chance that the patient is going to miss one of those drops goes up significantly."

Instead of adding more drops, send the patient directly for selective laser trabeculoplasty. "I go to SLT very early on if there's any question whatsoever about the control of the patient," he says.

Last but not least, don't let glaucoma patients get lost to follow up. "If patients fail to show up, you need to make an effort in writing to get them back in—and good documentation if they refuse to come back in," Dr. Vandervort says.

In short, "if you're going to manage glaucoma, you need a good recall system. If you're not managing a tight protocol for recall, then you need to be thinking about sending these people out for opinions a lot faster," he says. ■

1. Lisboa R, Leite MT, Zangwill LM, et al. Diagnosing preperimetric glaucoma with spectral domain optical coherence tomography. Ophthalmology. 2012 Nov;119(11):2261-9.
2. Rao HL, Addepalli UK, Jonnadula GB, et al. Relationship between intraocular pressure and rate of visual field progression in treated glaucoma. J Glaucoma. 2013; 22(9):719-24.

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This Mite Be Trouble

Demodex infestation requires timely, appropriate treatment, as it can cause a number of ocular complications. **Edited by Joseph P. Shovlin, OD**

Q I've noticed in my pre-fit assessment of potential contact lens patients that some occasionally show general signs of *Demodex* infestation, especially patients over 50. Which of these patients pose a problem, if not treated? And, if you choose to treat, what do you recommend using prior to wearing lenses?

A *Demodex* is a species of intradermal parasitic mites that reside in the ducts of the sebaceous glands that are near or connected to hair follicles in humans and animals.[1] Incidents of *Demodex* infestations often are linked to blepharitis and rosacea, and increase with age, making patients older than 50 a high-risk group.

"The *Demodex* species, namely *Demodex brevis* and *Demodex folliculorum*, are found in large numbers in the eyelash follicles and/or meibomian glands of patients presenting with *Demodex* blepharitis," explains Amir Azari, MD, who practices at Wills Eye Hospital in Philadelphia. "The classic sign of this condition is the development of 'waxy sleeves' around the base of the eyelashes."

These mites have the capability of inciting severe inflammation of the meibomian glands and the eyelid margins, according to Dr. Azari. This can lead to complications such as meibomian gland dysfunction and tear film disruption.

"These disorders can cause dry eye, conjunctival inflammation and even corneal scarring," adds Dr. Azari.

In addition to these complications, these mites exhibit symptoms similar to those of ocular allergies, according to Milton Hom, OD, who practices in California.

"As a result, we now always look for *Demodex* in contact lens patients who report allergy symptoms," says Dr. Hom.

Swiftly treating any contact lens patients presenting with a *Demodex* infestation will greatly improve lens comfort, according to Scott Schachter, OD, of Advanced Eyecare in Pismo Beach, Calif.

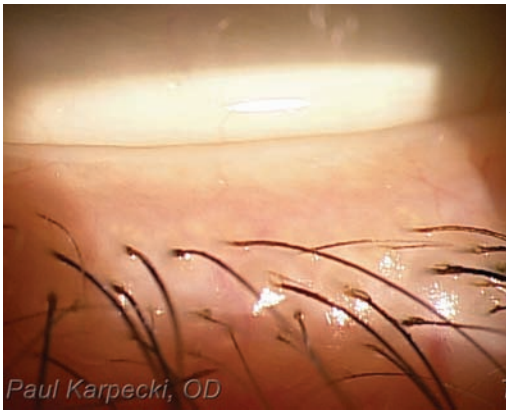
"Because contact lens use can also exacerbate dry eye, patients suspected of having *Demodex* blepharitis may benefit from treatment prior to contact lens wear," says Dr. Azari.

There are a number of available treatment options for this condition, and your choice should be dependent upon the severity of the presentation.

"I manage mild cases of the condition with tea tree oil wipes (Cliradex) twice per day for a month, then re-evaluate the patient," says Dr. Schachter.

According to Dr. Azari, Cliradex eyelid scrubs contain 4-terpineol, which is the effective ingredient in tea tree oil, for reducing the population of the *Demodex* species.

"For moderate-to-severe cases, a combination of a once-a-week in-office application of 50% tea tree oil and daily lid scrubs with 5% tea tree shampoo at home has also been advocated," says Dr. Azari. "All of these treatments should continue for several weeks until all of the signs and symptoms have resolved."



Credit: Paul M. Karpecki, OD

Common *Demodex* treatment options include eyelid scrubs and tea tree oil wipes.

According to Dr. Schachter, it's also important to always evaluate the family members of a patient presenting with *Demodex* infestation, as the mites can be spread by direct contact between individuals. All make-up should be discarded, and any linens should be immediately washed and then dried on a high-heat setting.

"*Demodex* is a ubiquitous mite that is also found on many normal patients," says Dr. Schachter. "I tell patients that if we see any signs of blepharitis, they likely have too many, and we need to reduce the population." ■

1. Czepita D, et al. Demodex folliculorum and Demodex brevis as a cause of chronic marginal blepharitis. Ann Acad Med Stetin. 2007;53(1):63-7.

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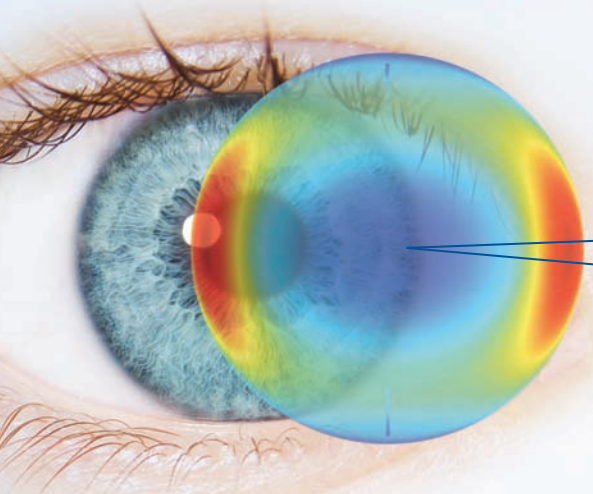

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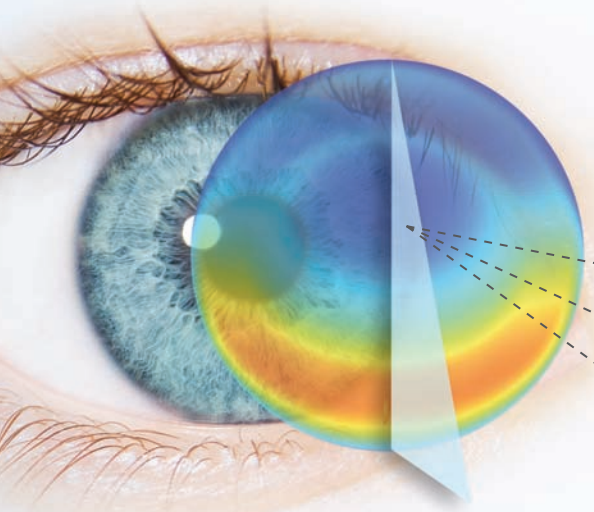

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Axis of Activity

Thyroid eye disease is the most common extra-thyroidal manifestation of Graves' disease, an autoimmune disorder. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

The endocrine system operates like a physiological factory: hormones are produced and stored until the body needs them, then delivered through the bloodstream to specific targets, like organs, tissues or cells. The thyroid gland, which affects metabolism, growth and maturation, is one of the most valuable players in this process.

The state of normal thyroid function is called euthyroidism, but abnormalities of the thyroid gland are fairly common, affecting 1% to 5% of the population, mostly women.[1] Disorders of the thyroid gland cause hormone imbalance, which can complicate personal health in many ways.

The Axis

The thyroid gland is attached to the lower part of the larynx and upper part of the trachea. It has two sides (or "lobes"), each about 4cm long and 1cm to 2cm wide, which are connected by a narrow isthmus.[2] The thyroid gland is influenced by the pituitary gland, which produces thyroid-stimulating hormone (TSH), and the hypothalamus, a small part of the brain above the pituitary, which produces thyrotropin-releasing hormone (TRH).[2]

The hypothalamus and pituitary detect low levels of thyroid hormones in the blood. When TRH is released, it stimulates the pituitary to release TSH. In turn, increased levels of TSH stimulate the thyroid gland to produce more thyroid hormone, thereby returning the level of thyroid hormone in the blood back to normal.[2,3] The three structures and the hormones they produce make up the hypothalamic-pituitary-thyroid axis.

Graves' and TED

Thyrotropin is a glycoprotein that stimulates the thyroid gland to produce and secrete the hormone thyroxine. Graves' disease (GD) is an autoimmune disorder characterized by hyperthyroidism—the overproduction of thyroid hormones—due to circulating autoantibodies. Thyroid-stimulating immunoglobulins (TSIs) bind to and activate thyrotropin receptors, causing the gland to grow and its follicles to increase synthesis of thyroid hormone.[4] While several conditions may result in hyperthyroidism, GD is the most common. Because thyroid hormones affect multiple body systems, signs and symptoms associated with GD are wide ranging.

Thyroid eye disease (TED), which has also been referred to as Graves' orbitopathy or ophthalmopathy, affects up to 60% of patients with GD.

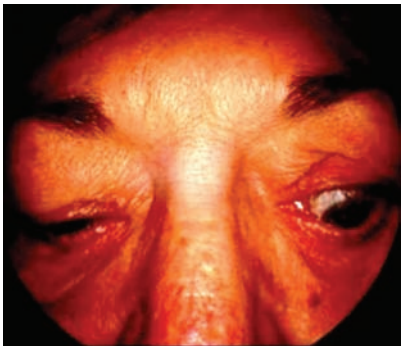
Thyroid eye disease (TED), which has also been referred to as Graves' orbitopathy or ophthalmopathy, affects up to 60% of patients with GD. Despite our detailed understanding of the etiology of hyperthyroidism in GD, the pathogenesis of TED remains uncertain.[4] This has limited the development of targeted therapies, particularly those that alter the course of TED.

Table 1. Review of Systems for Graves' Disease

- Weight loss, despite normal eating habits
- Enlargement of thyroid gland (goiter)
- Change in menstrual cycles
- Erectile dysfunction or reduced libido
- Frequent bowel movements or diarrhea
- Bulging eyes
- Double vision
- Thick, red skin, usually on the shins or tops of the feet
- Anxiety and irritability
- Difficulty sleeping
- Fatigue
- Rapid or irregular heartbeat
- Fine tremor of hands or fingers
- Increase in perspiration
-

NO SPECS: Werner Classification of Ocular Findings in Graves' Disease[9]

- N No signs or symptoms
- O Only signs
- S Soft tissue involvement (signs and symptoms)
- P Proptosis
- E Extraocular muscle involvement
- C Corneal involvement
- S Sight loss (due to optic nerve compression)



Evaluate the patient for upper eyelid abnormalities, such as retraction (lid rests above superior limbus) and von Graefe's sign (lid does not follow along smoothly on downgaze).

Table 2. Associated Factors That Modify GD/TED[5,6]

Other Autoimmune Disorders

- People with other disorders of the immune system, such as type 1 diabetes or rheumatoid arthritis, have an increased risk.
- Optimal control of these other conditions is essential.

Emotional or Physical Stress

- Stressful life events or illness may act as a trigger for the onset of GD/TED among people who are genetically susceptible.

Thyroid Function Status

- Control of thyroid function could mitigate severity.
- Monitor thyroid function every 4-6 weeks during initial phase of TED.

Tobacco Use

- Smoking has been identified as an important contributing environmental factor in the development of GD.
- Retrospective studies suggest that stopping tobacco use is associated with less severe TED, especially with regard to diplopia and proptosis.

Signs of TED

TED usually begins with orbital/periorbital inflammation, which may be progressive and can last for six months to two years.[5] Expansion of the extraocular muscles and orbital fat occurs during this period, and can result in proptosis, eyelid abnormalities, extraocular muscle motility deficits and compressive optic neuropathy.

In GD, the onset of hyperthyroidism and TED usually occur within 18 months of one another. A small minority of patients never develop thyroid dysfunction and are referred to as having "euthyroid GD."[4,5] The majority of patients with TED have mild, self-limiting disease. Nonetheless, even patients with mild disease experience a reduced quality of life.

Diagnostic Work-up

Diagnostic testing of free T4 (thyroxine) and TSH or serum TSH (thyrotropin) are highly sensitive and specific. Serum TSH is useful to establish a diagnosis of hyperthyroidism or hypothyroidism. Usually, the TSH is low in hyperthyroidism and high in hypothyroidism. Radiologic testing using a small amount of radioactive iodine may be implemented. A high uptake of radioactive iodine indicates that the thyroid gland is overproducing hormones.

Neuroimaging may be necessary if a diagnosis of TED cannot be established clinically. MRI is more sensitive than CT in showing compressive optic neuropathy. CT scanning usually reveals thick extraocular muscles with tendon sparing. The inferior rectus and medial rectus muscles are usually involved.

Treatment and Management

• ***Graves' Disease.*** Treatment of GD centers on correction of the thyrotoxic state. Normalization of thyroid hormone levels can be achieved with agents that block the synthesis of thyroid hormones or by treatment with radioactive iodine. Systemic beta-adrenergic blockers are prescribed to reduce the effect of hormones on the body.[4,5]

• ***Mild/Moderate TED.*** Most patients with TED can be observed, with the follow-up interval depending on disease status. Evaluation for corneal exposure, optic neuropathy and diplopia should be performed at these visits. Visual field and color vision testing may help in early detection of visual loss.[4,5]

The use of prism may be beneficial to patients with diplopia, if the deviation is small-angle and relatively comitant. If large-angle and/or incomitant, tape occlusion of one lens or segment of the glasses may be helpful. If this doesn't work, an occluder or vaulted eye patch (with care not to touch the cornea or compress the orbit) may be indicated. If a patient has dry eye symptoms, prescribe preservative-free artificial tears during the day and lubricating ointment at night, and consider punctal plugs.

• ***Severe TED.*** Systemic steroids represent the primary treatment for patients with moderate to severe, active TED. An IV steroid of 250mg of methylprednisolone given weekly over six weeks can be effective and have fewer side effects compared to oral steroids.[8] Surgical decompression of the orbit may be considered when vision is threatened by compressive optic neuropathy. Orbital radiotherapy remains an adjunctive strategy.

Targeted immunotherapies have the potential to alter disease progression, but further evidence is needed to establish safety and efficacy.[5,7]

Some of the immunosuppressive agents are azathioprine, cyclosporine and methotrexate. In severe cases of TED, treatment should be customized for each patient in close collaboration with the endocrinologist.[8]

Patient education

Patients living with TED should be advised that the condition usually runs a self-limited, but prolonged, course over one or more years, and no immediate cure is available. In addition, we must encourage patients to stop smoking, to decrease the risk of congestive orbitopathy. Sleeping with the head of the bed elevated may decrease morning lid edema.

TED likely involves both genetic and environmental factors. Until ongoing research establishes a target antigen, clinicians must focus on early signs and symptoms, along with timely treatment and management. ■

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A Two-Year Hiatus

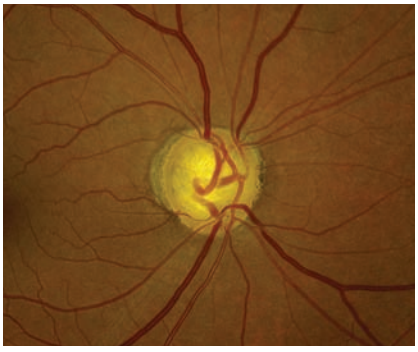
This glaucoma patient with a history poor compliance hasn't been seen in two years. Now, he has vision loss in his left eye. What's wrong? **By Mark T. Dunbar, OD**

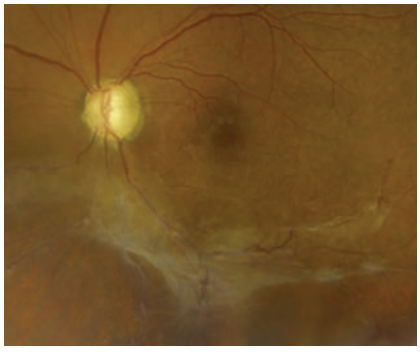
A 64-year-old black male with a history of primary open-angle glaucoma presented for a follow-up examination. We diagnosed him with glaucoma four years ago. Since then, the patient has exhibited poor compliance with his follow-up schedule and topical dosing regimen. We instructed him to use 0.5% timolol BID and latanoprost QHS OU.

His last eye exam was two years ago. At that time, his visual acuity was 20/20 OD and 20/30 OS. His IOP measured 9mm Hg OD and 12mm Hg OS. Additionally, his medical history was significant for medically controlled hypertension and high cholesterol.

At this visit, he reported that the vision in his left eye had decreased during the past six months. His best-corrected visual acuity was 20/20 OD and 20/70 OS. Confrontation fields were full to careful finger counting OD; however, he had generalized 360° constriction in the left eye. Pupils were equally round and reactive to light, with evidence of afferent defect OS. The anterior segment examination was unremarkable in both eyes. His intraocular pressure measured 19mm Hg OD and 15mm Hg OS.

Dilated fundus exam showed large cups OU, measuring approximately 0.90 OD and 0.95 OS. The vessels, macula and periphery in the right eye were normal; however, the fundus evaluation of the left eye revealed significant changes (*figures 1 and 2*). We also obtained an optical coherence tomography (OCT) scan and a fluorescein angiography (FA) of the left eye (*figure 3*).





1, 2. The fundus exam showed retinal hemorrhages around the left macula (OD left, OS right).

Take the Retina Quiz

1. What do the changes seen along the inferior arcade represent?
   a. Congenital retinal telangiectasis.
   b. Neovascularization with fibrous proliferation.
   c. Dense epiretinal membrane.
   d. Tractional retinal detachment (TRD).

2. Based on the clinical appearance, what is the correct diagnosis?
   a. TRD.
   b. Proliferative sickle cell retinopathy.
   c. Old branch retinal vein occlusion (BRVO) with neovascularization.
   d. Old branch retinal artery occlusion.

3. What is the likely cause of the reduced acuity in his left eye?
   a. Macular edema.
   b. Advanced glaucoma.
   c. Ischemia.
   d. Potentially all of the above.

4. How should this patient be managed?
   a. Laser photocoagulation.
   b. Intravitreal anti-VEGF injection.
   c. Observation.
   d. Pars plana vitrectomy and a scleral buckling procedure.

For answers, please turn to page 90.

Discussion

After looking at both optic nerves, it was evident that our patient had advanced glaucoma. Previous visual fields revealed inferior arcuate defects (OS > OD). At his last exam two years ago, the visual acuity in his left eye measured 20/30. Since then, it decreased to 20/70. Still, it was difficult to know if this was principally caused by glaucoma progression or another confounding factor.

With his history of noncompliance and advanced disease, it would be easy to assume that the visual acuity loss was a result of advanced glaucoma. But, not so fast!

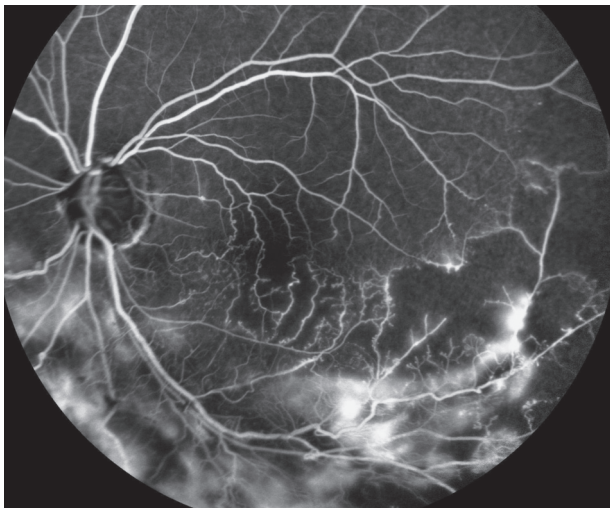
A large area of fibrous proliferation that followed the inferior-temporal and inferior-nasal arcade was located inferior to the optic nerve. Within the area of fibrous tissue, we noted extensive neovascularization. In fact, the neovascularization was quite pronounced on FA testing, with a massive amount of leakage. The FA also revealed large areas of non-perfusion, which was more evident peripherally to the areas of neovascularization (especially temporally).

So, what's the underlying cause? Based on the clinical presentation and FA, our patient had a resolved ischemic BRVO. (We noted "resolved" in the patient's record because we can no longer see the wedge-shaped area of intraretinal hemorrhages that's classically associated with a BRVO presentation.) While the hemorrhages weren't evident, the retina was highly ischemic. As a result, the patient developed neovascularization.

Ischemic complications secondary to BRVO are fairly common. In 1986, the National Eye Institute's Branch Vein Occlusion Study indicated that approximately 50% of patients with large BRVOs had significant areas of capillary nonperfusion (greater than five disc areas).[1] Forty percent of those patients exhibited neovascularization, and 60% developed vitreous hemorrhages.[1] The study concluded that, in patients who developed neovascularization, panretinal photocoagulation (PRP) was shown to be beneficial in preventing the development of vitreous hemorrhage.

In the era of anti-VEGF therapy, however, is PRP still the treatment of choice for neovascularization associated with ischemic BRVO? It is not clear from the literature.

The primary advantage of anti-VEGF treatment is that it swiftly resolves neovascularization. The downside is that the therapeutic effect usually is not sustained, so patients typically require multiple injections.



3. The late-phase fluorescein angiography showed extensive leakage in the left eye.

Further, there is a risk of tractional retinal detachments in patients with neovascularization and fibrous proliferation due to rapid involution combined with accelerated fibrosis and posterior hyaloidal contraction as a response to decreased VEGF levels.[2] As a result, many retinal specialists prefer PRP or a combination of PRP and anti-VEGF therapy.

Given our patient's history of poor compliance, we referred him for two sessions of PRP—which helped resolve the neovascularization. Other questions still remain, however, including the cause of his vision loss and why the BRVO developed in the first place.

Increased intraocular pressure is a known risk factor for the development of BRVO (and central retinal vein occlusion); however, it's usually not considered a significant contributor until IOP reaches 30mm Hg or higher. Based on a review of his records, his IOP never increased beyond 23mm Hg.

Hypertension is the other significant risk factor in his age group. And indeed, our patient has a long history of high blood pressure—although he reported good control.

It is difficult to pinpoint the fundamental cause of our patient's vision loss. The OCT scan revealed no macular edema, in addition to a well-preserved inner segment/outer segment junction. The FA did, however, show some ischemia —a potential contributing cause.

We have not performed a visual field test since the development of the BRVO. Presently, our chief goal is to reduce the patient's IOP as much as possible. We repeatedly stressed the importance of medication compliance, and scheduled him for a one-month follow-up appointment. Fortunately, the patient heeded our advice—his IOP measured 10mm Hg OU at the follow-up. Now, we simply have to wait until we can perform another visual field test after the BRVO stabilizes completely. ■

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Kill 'Em With Chemicals

In most states, ODs can safely and effectively cauterize benign skin lesions with dichloroacetic acid. **By Alan G. Kabat, OD, and Joseph W. Sowka, OD**

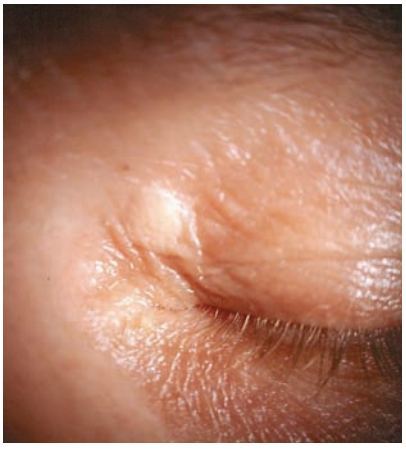
As the man in the AT&T commercial might ask, "Which is better: More or less?" Of course, we all like more when it comes to our phone and Internet service. Likewise, we might prefer a bigger office, car or dinner portion at our favorite restaurant. But in some cases, more is definitely *not* better—especially when it involves the growth or proliferation of our skin into unsightly lesions.

When initially encountering a skin lesion of the eyelid or adnexa, it is crucial to differentiate benign from malignant. Keep in mind that skin cancer remains the most common form of cancer in the United States.[1] So, if there is any question or suspicion of malignancy, you must promptly refer the patient for a biopsy evaluation. If the lesion is known or shown to be benign, however, you must then determine its etiology.

Non-cancerous Presentations

Papillomas, xanthelasmas and seborrheic keratoses are benign lesions that often are encountered in optometric practice, and can cause both functional and cosmetic concerns for our patients.

• *Papillomas* are one of the more common benign epithelial skin lesions, exhibiting a characteristic configuration of lobular projections that can resemble mulberries or cauliflower.[2] They may be flat or pedunculated (i.e., on a stalk). Further, the lesions may be solitary or multiple in presentation, and can vary in coloration—although the lesion typically approximates the patient's skin tone.



A 45-year-old woman exhibited xanthelasma of the left upper eyelid.

Papillomas may be of viral origin. In this instance, they are referred to as verrucae, viral papillomas or warts.[3] Those of a non-viral origin are termed acrochordons, squamous papillomas or skin tags.[4] Papillomas may occur at any age, although there is a greater predilection toward the viral variety before age 30 and the squamous variety beyond age 30.[5]

• *Xanthelasmas* are seen clinically as oval or elongated yellowish plaques located just beneath the skin of the periorbital region. Patients typically are more than 40 years of age, and women are affected nearly twice as often as men.[6] The lesions are neither inflammatory nor painful, and there is no tendency toward malignancy; however, the lesions may enlarge and/or coalesce over time. In rare instances, abnormally large xanthelasmas can interfere with lid function.

• *Seborrheic keratoses* present as sharply demarcated, round- or oval-shaped lesions on the eyelids or adnexa. They usually are elevated, ranging from a few millimeters to several centimeters in diameter. Seborrheic keratoses are classically described as having a "stuck on" quality to them. For this reason, and because they are seen more frequently in elderly patients, they have earned the moniker "barnacles of aging." The color may vary to extremes, much in the same fashion as papillomas. Nonetheless, most seborrheic keratoses appear a dull brownish-grey. These lesions generally are asymptomatic, but occasionally irritation or trauma may occur with subsequent itching, pain, redness, bleeding or crusting.[7]

Surgical Treatment Options

The aforementioned benign lesions may be treated in-office in a variety of ways.

• *Electrocautery and surgical curettage* is an effective treatment option that dermatologists and oculoplastic surgeons often employ for both ocular and non-ocular lesions.[8] However, these methods can be painful unless local anesthesia is used, and might yield cosmetically displeasing scarring.

• *Cryotherapy with liquid nitrogen* is another option—although treating periocular lesions in this fashion presents a significant risk

for hypo- or hyperpigmentation (especially in dark-skinned individuals), as well as nerve damage when therapy is too aggressive.[8] Also, the inadvertent application of cryogens to the ocular surface can cause severe inflammation and scarring.

• *Laser ablation* is also an option for many of these lesions, but optometrists are currently restricted from using this class of surgical instrumentation in all but two states (Oklahoma and Kentucky).

Chemical Cautery

Historically, optometrists did not have the ability to manage such lesions via surgical intervention. But today, there is a simple and effective method for removing benign lid and adnexal skin growths: chemical cautery with dichloroacetic acid. This approach is fully within the scope of optometric practice in many states, and is the safest and least invasive way to remove such lesions.

Dichloroacetic acid is a colorless, mildly pungent agent that has both keratolytic and cauterant properties. It can be obtained from a compounding pharmacist at a relatively low price (e.g., $45 from Gale Compounding Pharmacy in Pearson, Texas), or purchased as part of a complete treatment kit, such as the Derma-Cauter-All (approximately $260 from North Pine Enterprises).

The treatment process is extremely straightforward and easy to perform. It may be completed in or out of the slit lamp, but use of magnification generally is advisable. Be sure to wash your hands thoroughly and wear gloves during the procedure.

Prior to treatment, anesthetize the eye(s) with topical 0.5% proparacaine or tetracaine to prevent reflex lacrimation or other irritation from the acid's fumes. Then, apply a liberal amount of petrolatum around the base of the lesion, extending out at least 2mm to 3mm from the border of the intended treatment area. This helps protect the surrounding skin from incurring collateral damage. Take care not to coat the lesion itself, as this will render the tissue impervious to the acid's effects.

Next, use the dropper to transfer a small amount of acid from the bottle into a glass vial. Use wooden applicators (which resemble fancy toothpicks) to absorb a small amount of the acid from the glass receptacle, and apply this directly to the lesion. Repeat the process as many times as necessary in order to cover the entire area. Discard used applicators directly into a biohazard

bag to avoid unintentional burns or contamination. Advise the patient that the acid usually produces a slight burning or stinging sensation upon application and for a short while thereafter (in practice, this feeling frequently has been compared to an "ant bite").

When executed correctly, the treated area will turn a milky greyish-white color, indicating that the lesion has been cauterized and the small blood vessels were destroyed. This process causes the tissue to become necrotic and die, leaving behind only normal, viable cells.

In the ensuing days, the patient will notice the lesion becoming darker and scalier, forming an "eschar." After a week or two, this area will slough off completely to reveal healthy pink tissue that ultimately changes to match the patient's normal skin tone.

Although not critical, we recommend treating the eschar with a topical antibiotic/steroid ointment (e.g., TobraDex [tobramycin/dexamethasone, Alcon] ung TID to QID) to protect against infection and minimize inflammation and discomfort. Should any portion of the original lesion remain after two weeks, the area may be retreated; however, multiple studies have shown that chemical cautery is effective in eliminating a majority of lesions with just a single application.[9,10]

While lid "lumps and bumps" may occasionally represent an ominous situation, most of these lesions are benign and merely a cosmetic nuisance for our patients. With proper training in those areas that permit it, optometrists can provide a valuable service for their patients by helping to eliminate these unsightly growths quickly, easily and affordably. ■

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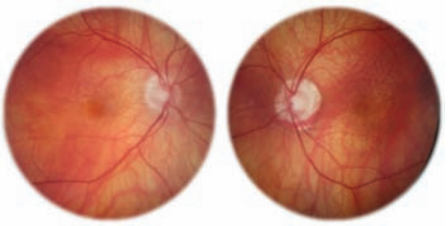
The Acid Test

This AMD patient presented with low PSA levels. Should he stop taking omega-3 fatty acid supplements? **By Diana L. Shechtman, OD, and Paul M. Karpecki, OD**

A 78-year-old white male visited the clinic for a re-evaluation of age-related macular degeneration (AMD) after an initial diagnosis of dry AMD five years ago. His medical history was remarkable for benign prostate enlargement and low prostate specific-antigen (PSA) levels. The patient is scheduled for surgery to remove part of the prostate. Current medications included an AREDS product plus 1,000mg of omega-3 fatty acids (FAs). His best-corrected visual acuity measured 20/30 OU. Slit-lamp evaluation was only remarkable for mild nuclear sclerosis OU. Dilated fundus examination revealed bilateral macular drusen, with no evidence of coexisting choroidal neovascular membrane.

Would you modify his current supplement regimen?



Dilated fundus examination of our 78-year-old patient. Medical history includes benign prostate enlargement with a low PSA. Should supplements be modified?

The Benefits of Fish Oil

Inflammation is a known contributing factor in various distinct systemic and ocular conditions. Thus, the anti-inflammatory activity of omega-3 supplementation is of primary interest to both patients and health care professionals. A number of debatable health benefits are associated with increased intake of the long-chain omega-3s, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).[1-11]

• *AMD.* In the past, omega-3 supplementation has been shown to decrease the prevalence and progression of AMD. However, the first published results from AREDS2 indicated that fish oil supplementation had no impact on progression toward more advanced disease stages.[12,13]

• *Heart disease.* A recent study determined that there was insufficient evidence to support previous findings that omega-3 FAs reduced the risk of cardiovascular-related mortality or morbidity.[14]

• *Prostate cancer.* Several studies have shown an inverse correlation between omega-3 FAs and prostate cancer.[5-12] One such study indicated an associated risk reduction for prostate cancer among men who ate fish at least three times a week.[8] A separate study followed 45,000 men for 14 years and found an association between high-level EPA and DHA intake and reduced incidence of prostate cancer.[11]

The SELECT Study

The original Selenium and Vitamin E Cancer Prevention Trial (SELECT) was intended to determine the effects of vitamin E, alone or with selenium, on prostate cancer.[15] More than 35,500 subjects enrolled in the initial study, which showed a 17% increased risk of prostate cancer among those taking 400IU of vitamin E supplementation.[16] Further analysis from SELECT determined that the high plasma phospholipid concentration of long-chain omega-3 polyunsaturated fatty acids also was associated with an increased incidence of prostate cancer.[15] The specific omega-3 FA measurements analyzed in the blood were comprised of DHA, EPA and docosapentaenoic acid (DPA).

The retrospective case-controlled cohort design compared blood samples of 834 men diagnosed with prostate cancer (of which 156 had a "high-grade" diagnosis) to an age-matched subcohort of 1,393 men. Overall, there was a 43% increased risk of prostate cancer among those who had the highest level of omega-3 FA measurements in their blood. Men with the highest blood plasma level of omega-3 FAs had a 44% increased risk of developing "low-grade" prostate cancer, and a 71% increased risk of developing "high-grade." Increased levels of blood serum linoleic acid (primarily comprised of omega-6 fatty acids) were associated with a decreased risk of "low-grade" prostate cancer.[15]

However, a closer look at the study raised several questions.

• Its original goal was to evaluate the relationship between prostate cancer and vitamin E, not omega-3 FAs.

• No other study has shown an increased risk of prostate cancer related to omega-3 FA supplementation.

• Specific variables with regard to the omega-3 FAs were not addressed. This included, but was not limited

to, the type of omega-3 FA supplements taken, quantity of omega-3 FA intake, distinction between dietary intake and supplementation, and/or quality of omega-3 FAs intake.

- Red blood cell analysis of DHA and EPA is the conventional and acceptable marker for measuring omega-3 FA intake. Blood plasma evaluation may not yield an adequate assessment.
- Singular measurements were taken at commencement of the study.
- The data does not explain the relationship as cause and effect.
- Prostate cancer is associated with several contributing factors. The study accounted for some, not all.
- An intervention design, rather than a retrospective study, would have been more adequate to determine a definitive relationship between omega-3 FAs and prostate cancer.
- In some cases a digital rectal exam was conducted, but no biopsies.

Given our patient's overall clinical picture, we suggested that he cease the use of omega-3 supplementation, but continue with an AREDS product that substituted lutein for beta-carotene. ■

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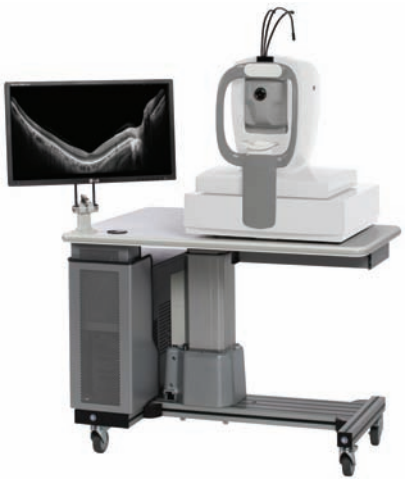
Product **Review**

Diagnostic Technology

Anterior and Posterior OCT

Optovue introduced the cross-functional spectral-domain Avanti Widefield Enface OCT.



Avanti provides an all-in-one solution for posterior and anterior high-resolution OCT imaging at 70,000 A-scans per second, with widefield 3D motion correction and 3 micron digital resolution. In addition to fast and accurate imaging, Avanti offers simultaneous multi-layered assessment of peripheral retina pathology; deep choroidal imaging; fovea location recognition; pachymetry mapping; and optic disc, RNFL and ganglion cell complex assessment.

Optovue says the device was designed as a scalable platform with next-generation OCT in mind.

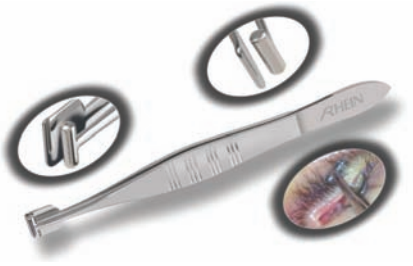
Visit www.otopvue.com.

Patient Care

Meibomian Compressors

Compress your patients' eyelids with greater comfort and efficiency with Rhein Medical's Batlle Eyelid Compression Forceps.

These forceps, developed in coordination with Juan Batlle, MD, are designed with a mirror-polished paddle on one side and a mirror-polished round appendage on the other. Insert the paddle into the inside of the eyelid, and the round appendage on the outside of the eyelid. When compressed, the instrument forces meibium to be expressed out of the glands.



Visit www.rheinmedical.com.

Lacrimal Dilation

Achieve true dilation of the lacrimal duct with FCI Ophthalmics' now FDA-approved OphtaCath catheter. This tool is designed to rapidly and effectively treat the symptoms of epiphora, the company says, and is considered a less-invasive alternative to incisional procedures like dacryocystorhinostomy.

OphtaCath comes packaged in sterile 2mm or 3mm kits with one or two balloon catheters and an easy-to-use disposable inflation device. The catheter's tapered tip and low profile are designed for easy insertion and removal.

Visit www.fci-ophthalmics.com.

LOW VISION

Task Specific Spectacles

Not all spectacles are created equal, according to Tech-Optics International. The company, which touts its history of innovation in low-vision spectacles, offers Task-Vision high plus aspheric lenticular and microscopic spectacles with optical quality CR-36 lenses.

The lenticulars and microscopics are scratch-coated, accurate and bench-aligned with a line of alloy frames in contemporary colors like ebony, coffee, burgundy, gunmetal blue, platinum and gold, and Full-vue frames in demi-amber and crystal. The company also offers blackout and frosting at no additional charge.

Visit www.techopticsinternational.com.

Eyewear

Custom Sunglasses

The first of Costa Sunglasses' 2014 line kicks off with Hamlin, durable-style lenses featuring co-injected nylon molded frames and no-slip nose pads and temple tips. The extra-large fit features a wrap shape to protect incoming glare.

Hamlin is available in Costa's patented 580 lens technology, which blocks yellow light from entering the eye, creating razor-sharp color enhancement and high polarization levels, the company says. The style is named for legendary angler Capt. Ron Hamlin, and all Costa sunglasses are custom-built and hand assembled with a lifetime warranty.

Visit www.costadelmar.com.

High-Performance

Those who lead a high-performance, active lifestyle could benefit from Maui Jim's Switchbacks.

This eyewear offers the unique ability to switch lenses at the touch of a button through the company's proprietary interchangeable lens system. When light conditions change, the lenses change with them. Switchbacks are available in a modified rectangle shield frame with a form-fitting design and non-slip temples and nose pads.

Visit www.mauijim.com.

Advanced Gaming

Time to level up, gamers. An all-new line of glasses has been unveiled by Gunnar Optiks, maker of computer and gaming eyewear.

The Intercept Color Collection is a new assortment of colored frames that deliver a personalized, multi-chromatic touch to the retro-infused, high-tech optics of Gunnar's existing collection, according to the company. The collection was designed with hardcore gamers, creative designers and technophiles in mind. The wide lens design gives gamers a visually enhanced, unobstructed view of game worlds, and the proprietary lens design allows for longer play, the company says.

Visit www.gunnars.com.

Fashion

A new online retailer has launched in the market with several signature frames, including the Brittany, Rio and Village.

Spiffy Society says it will offer unique, bold, handmade, vintage-retro eyewear that serves as a "counterpoint to staid classics." Their spiffy styles feature rounded curves and textures, and favor jewel tones and bold colors over neutrals. The retailer, which offers augmented Virtual Mirror Try-On, also has sunglasses available, including their signature Marigot, a bold acetate frame for the quintessential trendsetter.

Visit www.SpiffySociety.com.

Cosmetics

Eyelash Thickener

For patients interested in the appearance of fuller, more aesthetically pleasing eyelashes, brows or eyelids, non-prescription Lash Advance offers a unique blend of natural, cosmeceutical ingredients in a clear gel that will not irritate eyes, according to MediNiche, Inc.

Lash Advance works as a lash plumper under mascara and tightens wrinkled skin. According to the company, Lash Advance can be used safely with contact lenses, and by those who have undergone refractive surgery.

Visit www.lashadvance.com.

Meetings + Conferences

January 2014

■ **16-19.** *New Technology & Treatments in Vision Care.* The Westin Resort & Casino, Aruba. Program Chair: Paul Karpecki, OD. Faculty: Jimmy Bartlett, OD, Ben Gaddie, OD, and Kimberly Reed, OD. Hosted by: *Review of Optometry*. CE hours: 14. Contact Lois DiDomenico at ReviewMeetings@Jobson.com. Visit www.revoptom.com/conferences.

■ **18-20.** *Berkeley Practicum - 25th Annual.* DoubleTree Hotel, Berkeley Marina, Berkeley, Calif. Hosted by: University of California, Berkeley, School of Optometry. CE hours: 20. Email optoCE@berkeley.edu. Visit http://optometry.berkeley.edu/ce/berkeley-practicum.

■ **19-25.** *2014 Island Eyes Conference.* Grand Wailea, Maui, Hawaii. Hosted by: Pacific University College of Optometry. Contact Jeanne Oliver at jeanne@pacificu.edu or (503) 352-2740. Visit www.pacificu.edu/optometry/ce.

■ **24.** *2014 Winter CE.* PCLI, Pearl District, Portland, Ore. Hosted by: Oregon Optometric Physicians Association. CE hours: 8. Email lynne@oregonoptometry.org. Visit www.oregonoptometry.org.

■ **30-February 3.** *Women of Optometry Spa Cruise.* Celebrity Constellation, Bahamas. Hosted by: AEA Cruises Optometric Cruise Seminars. Email aeacruises@aol.com. Visit www.OptometricCruiseSeminars.com.

February 2014

■ **2.** *Winter CE Meeting 2014.* Tinley Park Convention Center, Tinley Park, Ill. Hosted by: Illinois Optometric Association. Key faculty: Robert Dunphy, OD. CE hours: 6. Email ioabb@ioawewb.org. Visit www.ioaweb.org.

■ **5-6.** *Michigan Optometric Association Winter Seminar.* Kellog Hotel and Conference Center, East Lansing, Mich. Hosted by: Michigan Optometric Association. CE hours: 12. Email optla@bellsouth.net. Visit www.optla.org.

■ **8-9.** *Mid Winter CE Meeting 2014.* New Orleans Marriott, New Orleans. Hosted by: Optometry Association of Louisiana. CE hours: 12. Email optla@bellsouth.net. Visit www.optla.org.

■ **9-10.** *2014 Advocacy Boot Camp & Free CE.* Salem Conference Center/Grand Hotel, Salem, Ore. Hosted by: Oregon Optometric Physicians Association. Email lynne@oregonoptometry.org. Visit www.oregonoptometry.org.

■ **14-16.** *53rd Annual Heart of America Contact Lens and Primary Care Congress.* Sheraton Kansas City Hotel at Crown Center, Kansas City, Mo. Hosted by: Heart of America Contact Lens Society. Email registration@thehoacls.org. Visit www.hoacls.org.

■ **15-19.** *SkiVision 2014 Winter Opthalmic Congress.* Viceroy Luxury Resort Hotel, Snowmass Village, Colo. Hosted by: SkiVision LLC. Key faculty: Murray Fingeret, OD, Leo Semes, OD, Ben Gaddie, OD, Robert Fechtner, MD, Jack Schaeffer, OD, Kathy Dumbleton, OD, John Flanagan, MD, William Katowitz, MD. CE hours: 20. Email questions@skivision.com or call 1-888-754-2530. Visit www.skivision.com.

■ **15-24.** *AEA Cruises Southern Caribbean Cruise Seminar.* Celebrity Summit, San Juan. Hosted by: AEA Cruises. Key faculty: Charles Fico, OD. CE hours: 10. Contact Marge McGrath at aeacruises@aol.com or 1-888-638-6009. Visit www.optometric-cruiseseminars.org.

■ **16-23.** *AEA Cruises Southern Caribbean Cruise Seminar.* Royal Princess, Caribbean. Hosted by: AEA Cruises. Key faculty: S. Barry Eiden, OD. CE hours: 10. Contact Marge McGrath at aeacruises@aol.com or 1-888-638-6009. Visit www.optometriccruiseseminars.org.

■ **23.** *Winter CE Meeting 2014.* Marriot Bloomington-Normal Hotel, Bloomington, Ill. Hosted by: Illinois Optometric Association. Key faculty: Joe Pizzimenti, OD. CE hours: 6. Email ioabb@ioawewb.org. Visit www.ioaweb.org.

■ **27-March 1.** *2014 Winter Educational Symposium.* Huntley Lodge, Big Sky, Mont. Hosted by: Montana Optometric Association. Faculty: Blair Lonsberry, OD, Christopher Wolfe, OD. CE hours: 13. Email sweingartner@rmsmanagement.com. Visit www.mteyes.com.

■ **28-March 1.** *2014 Third Party/Practice Management Seminar.* Eugene Hilton, Eugene, Ore. Hosted by: Oregon Optometric Physicians Association. Email lynne@oregonoptometry.org. Visit www.oregonoptometry.org.

March 2014

■ **9-14.** *31st Annual Johns Hopkins Current Concepts in Ophthalmology.* Vail Marriot, Vail, Colo. Hosted by: Johns Hopkins University School of Medicine. CE hours: 40. Contact Kathy Case at kcase5@jhmi.edu. Visit www.hopkins.edu/CourseDetail.aspx/80031806.

■ **12-16.** *SECO International 2014.* Building A, Georgia World Congress Center, Atlanta. Hosted by: SECO. CE hours: 400+. Contact cweems@secostaff.com. Visit www.seco2014.com.

■ **14.** *Binocular Vision Forum.* The Ohio State University School of Optometry, Columbus, Ohio. Hosted by: Ohio State University School of Optometry. CE hours: 49. Contact Marjean Kulp, OD, at kulp.6@osu.edu. Visit www.optometry.osu.edu.

■ **21-23.** *POA Spring Conference.* Nittany Lion Inn, State College, Penn. Hosted by: PA Optometric Association. Key faculty: Joe Sowka, OD, Andy Gurwood, OD, Al Kabat, OD. CE hours: 10. Contact Ilene Sauertieg at ilene@poaeyes.org. Visit www.poaeyes.org.

■ **22-23.** *Spring Conference.* Nova Southeastern University Ft. Myers Campus, Ft. Myers, Fla. Hosted by: Nova Southeastern University. Contact oceaa@nova.edu. Visit http://optometry.nova.edu/ce/index.html.

■ **26-30.** *Vision Expo East.* Javits Center, New York. Hosted by: International Vision Expo. Visit www.visionexpoeast.com.

■ **27-29.** *OAOP Annual Spring Congress.* Embassy Suites

Hotel and Conference Center, Norman, Okla. Hosted by: Oklahoma Association of Optometric Physicians. Key faculty: Walt West, OD, Blair Lonsberry, OD, David Talley, OD, Larry Henry, OD, Jason Ellen, OD, Chad Chamberlain, DO, Joyce Ardrey, CPC. CE hours: 18. Contact Heatherlyn Burton at heatherlyn@oaop.org. Visit www.oaop.org.

April 2014

■ **3-6.** *OptoWest 2014.* Esmeralda Hotel, Indian Wells, Calif. Hosted by: California Optometric Association. Key faculty: Marc Bloomenstein, OD, Sharon Carter, ECOC, Melissa Chun, OD, Laurie Guest, CSP, Lynn Hellerstein, OD, FCOVD, Richard Hom, OD, MPA. CE hours: 40+. Contact Rachael Van Cleave at contact@coavision.org. Visit www.coavision.org.

■ **19-20.** *2014 MOS Primary Care Spring Symposium.* Cincinnati Marriott Northeast, Mason, Ohio. Hosted by: The MidWest Optometric Society and The Ohio State University College of Optometry. Contact Marci at (513) 321-2020. Visit www.midwestoptometricsociety.com.

■ **24-26.** *Mountain West Council of Optometrists Annual Congress.* Caesars Palace, Las Vegas. Hosted by: MWCO. CE hours: 24. Call 1-888-376-6926. Visit www.mwco.org.

■ **24-27.** *Arkansas Optometric Association Spring Convention.* The Peabody, Little Rock, Ark. Hosted by: Arkansas Optometric Association. Email aroa@arkansasoptometric.org. Visit www.arkansasoptometric.org.

May 2014

■ **2.** *Berkeley Glaucoma Day - 2nd Annual.* DoubleTree Hotel, Berkeley Marina, Berkeley, Calif. Hosted by: University of California, Berkeley, School of Optometry. Email optoCE@berkeley.edu. Visit awww.aoa.org/events/ucb-glaucoma-day.

■ **2-3.** *Educational Meeting 2014.* Mission Inn, Howey-in-the-Hills, Fla. Hosted by: Florida Chapter of the American Academy of Optometry. Featured speakers: Leo Semes, OD, Albert Woods, OD, and Tim Underhill, OD. CE hours: 10. Contact Arthur T. Young, OD, at eyeguy4123@msn.com.

■ **3-4.** *Morgan Symposium - 29th Annual.* DoubleTree Hotel, Berkeley Marina, Berkeley, Calif. Hosted by: University of California, Berkeley, School of Optometry. Email optoCE@berkeley.edu. Visit http://optometry.berkeley.edu/ce/morgan-symposium.

To list your meeting, please send the details to:

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Edited by Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

DALK Pays Dividends

Reduced risk of rejection, longer graft lifespan and a simpler postoperative drug regimen all characterize this sometimes overlooked procedure. **By Aaron Bronner, OD**

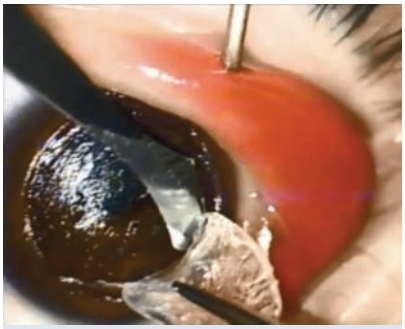
Deep anterior lamellar keratoplasty (DALK) is a form of lamellar corneal transplant that has probably not been given its due within eye care. Whereas most of the lamellar graft procedures (e.g., DSAEK, DMEK) are posterior lamellar transplants used for patients with endothelial decompensation, DALK stands alone as an anterior lamellar transplant. Indicated for anterior pathology, such as keratoectasias, scars and stromal dystrophies, DALK is a surgical alternative to penetrating keratoplasty—not to the DSAEK or DMEK group of procedures.

Endothelial-sparing Surgery

The chief advantage of DALK lies in its preservation of the host endothelium; it remains intact in the patient. Donor endothelium and Decemet's membrane (DM) are removed from the graft prior to transplant. This influences two very important postoperative features: the expected lifespan of the graft and the immunologic risk to it.

Concerning the first point, consider that PK grafts are bound by a finite lifespan of roughly 20 years. Thus, given a normal postoperative course without any complications, a PK patient can only expect to keep that graft for two decades at best. Because a DALK procedure does not transplant endothelium, decompensation occurs at physiologic levels and therefore the graft may persist indefinitely.

The second point, regarding the immunology of corneal transplantation, is difficult to briefly outline. But, in appreciating the virtues of DALK, it is necessary to understand two concepts: (1) the only permanent target of immunologic rejection by the host directed against a PK graft is the donor endothelium, and (2) the type of rejection that commonly causes failure of a PK is endothelial targeted rejection.



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

DALK allows the host to keep his or her own endothelium, and removes this potential long-term stimulus for rejection-derived graft failure from the mix of potential postoperative complications. This greatly reduced risk of rejection enables important trickle-down benefits as well.

While DALK patients are placed on corticosteroids in first year postoperatively, they are less dependent upon them over the long term, and can taper off the regimen more rapidly than is advisable with PK patients. This limits the risk of the steroid-induced side effects, such as glaucoma, cataract and infection.

The Optometrist's Role

As an OD, what do you need to know for your patients considering this surgery? To be a good candidate, the patient needs to have a relatively uninvolved and well-functioning endothelium. Therefore, patients with penetrating scars or a history of hydrops are not ideal candidates for the procedure, although it still may be attempted.

Even in good candidates, 30% of DALK procedures will be converted intraoperatively to PK as a result of perforations to DM during the surgery. Although DALK has certain advantages over PK, it should be noted that both procedures require sutural fixation of the graft (unlike posterior lamellar grafts). Therefore, both DALK and PK share the same slow visual recovery, relatively high risk of irregular astigmatism and dependence on gas permeable contact lenses. In fact, in many cases, it can be clinically impossible to differentiate a DALK from a PK in the microscope.

Despite a few challenges, DALK has enormous postoperative advantages over PK—athough the complexity of the surgery and slow visual recovery, which are identical to that of PK, has dampened its dissemination in the field. However, when available, it is my opinion that procedure generally is the transplant treatment of choice for any of these pathologies. ■

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

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Diagnostic **Quiz**

Will This Floater Sink?

By Andrew S. Gurwood, OD

History

A 47-year-old white female presented with a chief complaint of a floater in her left eye that had persisted for four weeks.

The patient explained that she first noticed the moving spot after being accidently hit near the eye by another person while out dancing. Her systemic and ocular histories were unremarkable. She reported no known allergies of any kind.

Diagnostic Data

Her best-corrected entering visual acuity measured 20/20 OU at distance and near. Her external examination was normal, with no sign of afferent pupillary defect.

The biomicroscopic examination of the anterior segment was normal. The patient exhibited no evidence of iris neovascularization.

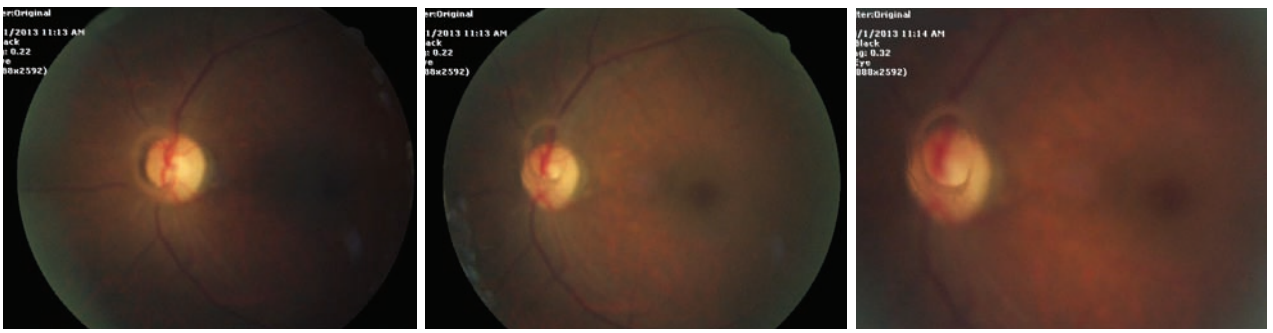
Her intraocular pressure measured 15mm Hg OU. We documented peripheral pathologies in both eyes. The pertinent clinical finding is illustrated in the photographs.

Your Diagnosis

How would you approach this case? Does the patient require any additional tests? What is your diagnosis? What is the likely prognosis?

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

Thanks to Carolyn Majcher, OD, of San Antonio, and Julie Hutchinson, OD, of St. Louis, for their contributions to this case.



Multiple fundus images of our 47-year-old patient's left eye. What do you notice, and how should she be managed?

Retina Quiz Answers *(from page 70):* 1) b; 2) c; 3) d; 4) a.

Next Month in the Mag

February features our annual Pharmaceuticals Issue.
Topics include:

- *The Top 10 Oral Meds in Eye Care*
- *Doxycine Do's and Don'ts*
- *Learn to Identify Drug-Seeking Behavior*

Feedback

Review of Optometry welcomes questions and comments. E-mail Jack Persico, editor-in-chief, jpersico@jobson.com, with "Letter to the Editor" as the subject line.
Or, write to *Review of Optometry*, 11 Campus Blvd., Suite 100, Newtown Square, PA 19073.

REVIEW OF OPTOMETRY (ISSN 0147-7633) IS PUBLISHED MONTHLY, 12 TIMES A YEAR BY JOBSON MEDICAL INFORMATION LLC, 100 AVENUE OF THE AMERICAS, NEW YORK, NY 10013-1678. PERIODICALS POSTAGE PAID AT NEW YORK, NY AND ADDITIONAL MAILING OFFICES. POSTMASTER: SEND ADDRESS CHANGES TO REVIEW OF OPTOMETRY, PO BOX 2025, SKOKIE, IL 60076. SUBSCRIPTION PRICES: US: ONE YEAR $56; TWO YEARS $97, CANADA: ONE YEAR $88, TWO YEARS $160, INT'L: ONE YEAR $209, TWO YEARS $299. FOR SUBSCRIPTION INFORMATION CALL TOLL-FREE (877) 529-1746 (USA ONLY); OUTSIDE USA, CALL (847) 763-9630 OR FAX (847) 763-9631. PUBLICATIONS MAIL AGREEMENT NO: 40612608. CANADA RETURNS TO BE SENT TO BLEUCHIP INTERNATIONAL, P.O. BOX 25542, LONDON, ON N6C 6B2.

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References: 1. Based on third-party industry report, 12 months ending July 2013; based on unit sales, Alcon data on file. **2.** Wolffsohn J, Hunt O, Chowdhury A. Objective clinical performances of 'comfort-enhanced' daily disposable soft contact lenses. *Cont Lens Ant Eye*. 2010;33(2):88-92. **3.** Alcon data on file, 2010, 2013.

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