A Lesson Plan for Pediatric Practice Building, *Page 24*

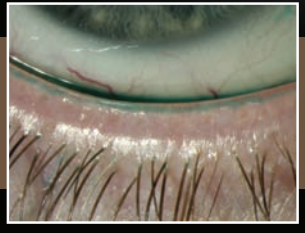
REVIEW® OF OPTOMETRY

September 15, 2014

www.revoptom.com

Can You Identify These Optic Nerve Anomalies?

An expert overview of presentations and pathology for the practicing clinician. Page 34



15th Annual Dry Eye Report

DESIGN *comes* NATURALLY *to* US

DESIGNS THAT STAND OUT FROM THE PACK.

Since 1983, highly skilled individuals, innovative design, outstanding customer service and cutting edge technology have defined Marchon. It is this commitment and passion which has helped to solidify our superior reputation in the market.

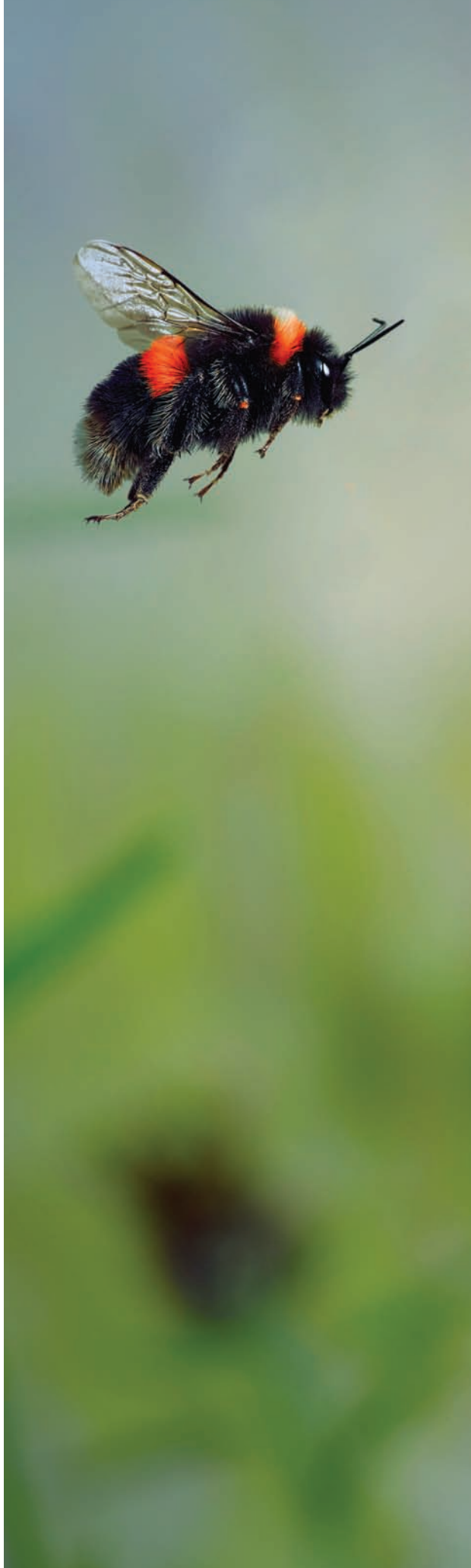
MARCHON®
FOCUSED ON YOU

marchon.com / 1-800-645-1300

CUSTOMER SERVICE *comes* NATURALLY *to* US

WE'RE BUSY 24/7 WORKING TO PROVIDE YOU WITH EVERYTHING YOU NEED TO KEEP YOUR BUSINESS BUZZING.

Since 1983, highly skilled individuals, innovative design, outstanding customer service and cutting edge technology have defined Marchon. It is this commitment and passion which has helped to solidify our superior reputation in the market.



MARCHON®
FOCUSED ON YOU

marchon.com / 1-800-645-1300

VOL. 151 NO. 9 ■ SEPTEMBER 15, 2014

Study Offers 'Foolproof' Eye Test for Diagnosing ADHD

Researchers combine attention assessment with eye tracking to create an objective test for ADHD. But is it truly 'foolproof'? **By John Murphy, Executive Editor**

A test of involuntary eye movement "accurately indicates attention deficit hyperactivity disorder (ADHD)," according to a new study by researchers at Tel Aviv University in Israel.

However, more research is needed to confirm whether this test is truly accurate.

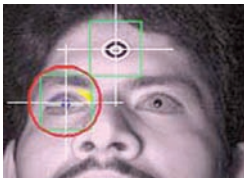


Photo: SR Research

Researchers assessed visual attention using eye tracking to reach a diagnosis of ADHD.

ADHD is one of the most common neurobehavioral disorders in children in America, according to the Centers for Disease Control and Prevention. But it can be tricky to diagnose. Currently, there is no single objective test to diagnose ADHD, and many other disorders—anxiety, depression and certain types of learning disabilities—can have similar symptoms.

"We had two objectives going into this research," says lead author Moshe Fried, PhD, who was diagnosed with ADHD as an adult. "The first was to provide a new diagnostic tool for ADHD. The second was to test whether ADHD medication really works—and we found that it does. There was a significant difference between the two groups, and between the two sets of tests taken by ADHD participants unmedicated and later medicated."

In the study, the researchers administered a computer test, the Test of Variables of Attention (TOVA), to two groups of 22 adults. The first group of participants, already diagnosed with ADHD, initially performed the 22-minute test unmedicated and then took it again after a prescribed dose of methylphenidate, known as the brand name Ritalin (Novartis). The second group, not diagnosed with ADHD, constituted the control group. The researchers also used an eye-tracking system to monitor the participants' involuntary eye movements as they took the tests.

"We found that the average microsaccade and blink rates were higher in the ADHD group, especially in the time interval around stimulus onset," the researchers concluded. In other words, the study showed a direct correlation between ADHD and the inability to suppress eye movement in the anticipation of visual stimuli. The study also showed that methylphenidate does work. "It is certainly not a placebo, as some have suggested," Dr. Fried says.

He adds, "This test is affordable and accessible, rendering it a practical and foolproof tool for medical professionals. With other tests, you can slip up, make 'mistakes'—intentionally or not. But our test cannot be fooled. Eye movements tracked in this test are involuntary, so they constitute a sound physiological marker of ADHD."

Sound, maybe, but the term "foolproof" is inappropriate for scientific publications, says Darrell G. Schlange, OD, DOS, an associate professor at Illinois College of Optometry, whose research in pediatric vision includes reading eye movements and ADHD.

Also, Dr. Schlange says, the size of the test group in this study was insufficient to state that involuntary eye movements "accurately reflect" the presence of ADHD. "This is impressive research, but more is needed to more fully understand the complexities of oculomotor testing for attention disorders, including ADHD," he says.

To that end, the researchers are currently conducting more extensive trials on larger control groups to further explore applications of the test.

For information about the diagnosis of ADHD, visit: www.cdc.gov/ncbddd/adhd/diagnosis.html.

See also "How to Handle a Hyper Kid," page 84.

Fried M, Tsitsiashvili E, Bonneh YS, et al. ADHD subjects fail to suppress eye blinks and microsaccades while anticipating visual stimuli but recover with medication. Vision Res. 2014 Aug;101:62-72.

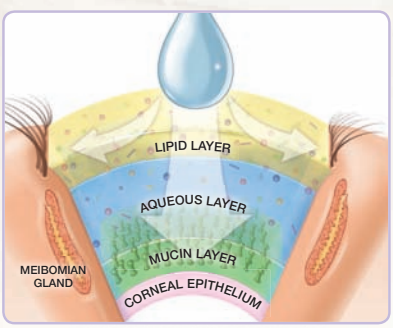
For the 75% of dry eye patients worldwide with evaporative dry eye (MGD) symptoms[1]...

DRY EYE CAN BE RELENTLESS

CALM THE STORM WITH LASTING RELIEF

SYSTANE® BALANCE Lubricant Eye Drops:

Protecting the Ocular Surface by Increasing Lipid Layer Thickness (LLT)



SYSTANE® BALANCE Lubricant Eye Drops forms a protective matrix that is designed to replenish the lipid layer for long-lasting relief from the symptoms associated with evaporative dry eye (MGD). This unique formulation is designed to work on all 3 layers of the tear film, specifically increasing LLT. This helps create a protective environment for the ocular surface.[2]

SYSTANE® Brand products are formulated for the temporary relief of burning and irritation due to dryness of the eye.

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

Your recommendation counts.

Make sure your patients get the lasting symptom relief they need by offering them SYSTANE® BALANCE Lubricant Eye Drops.[2]

Systane® Family of Products

Relief that lasts

Alcon®
a Novartis company

 01/14 SYS14005JAD

Retinal Changes Predict Dementia

Researchers have found that retinal thinning is one of the earliest signs of frontotemporal dementia (FTD) in people with a genetic risk for the disorder—even before any cognitive or behavioral changes appear.

A team at the Gladstone Institute of Neurological Diseases at the University of California San Francisco studied individuals who had a certain genetic mutation known to result in FTD. Using conventional spectral-domain optical coherence tomography, the researchers discovered that these individuals showed a significant thinning of the retina before any observable cognitive signs of dementia, when compared with people who did not have the gene mutation.

"It is important to note that our findings were based on the average retinal thickness of at-risk or healthy people as a group, and that a single retinal scan couldn't be used to predict dementia risk in an individual person. This is because there is significant variation in retinal thickness among even healthy individuals," says lead author Michael Ward, MD, PhD. "However, longitudinal imaging of the same person over multiple years could be useful to look for changes in the rate of retinal thinning in people at risk for dementia, especially as a potential biomarker in the setting of a clinical study. We are currently evaluating this possibility."

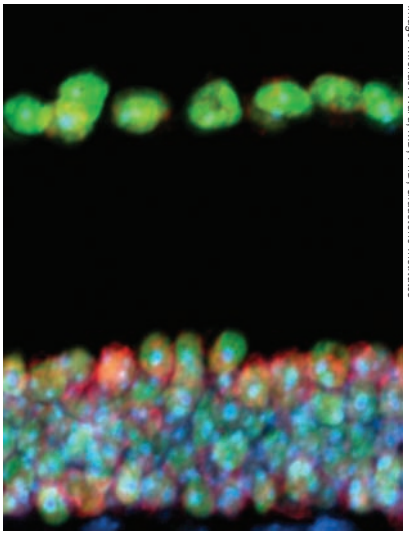


Image: Michael Ward, MD, PhD, Gladstone Institutes

Cross-section of the retina in a healthy mouse, showing TDP-43 staining (green), Ran staining (red) and nuclei (blue).

The researchers also discovered new mechanisms by which cell death occurs in frontotemporal dementia. They identified the depletion of a crucial protein, TDP-43, from the cell nuclei before any signs of neurodegeneration occurred, signifying that this loss may be a direct cause of the cell death associated with FTD. TDP-43 levels, they determined, are regulated by another cellular protein called Ran. By increasing expression of Ran, the researchers were able to elevate TDP-43 levels in mice with the FTD gene mutation, and prevent neuronal cell death.

Ward ME, Taubes A, Chen R, et al. Early retinal neurodegeneration and impaired Ran-mediated nuclear import of TDP-43 in progranulin-deficient FTLD. J Exp Med. 2014 Aug 25. [Epub ahead of print]

California OD Bill is Shelved Again

A bill to expand optometrists' scope of practice in the state of California has been pulled. The bill, SB 492, was drastically amended and subsequently shelved by its sponsor, Sen. Ed Hernandez (D-West Covina), following disagreements with state health care groups.

After discussion with Sen. Hernandez, the California Optometric Association (COA) has decided not to move the bill forward, which was reduced to only authorizing ODs to give limited immunizations.

"SB 492 was diminished to a state that would not have been beneficial for Californians seeking proper eye care," says COA President John Rosten, OD. "We are grateful for the effort put forth by Sen. Hernandez and look forward to working with the legislature to pass meaningful scope of practice legislation next year."

The goal is to allow optometrists to practice in a way that is more fully aligned with their education, training and experience, the COA says, and it believes legislators will be true to their word and open to genuine discourse next year.

The original bill, which had also been put on the back burner last year, would have expanded the role of optometrists to diagnose and treat certain common systemic diseases, including diabetes mellitus, hypertension and hypercholesterolemia. It would have also authorized ODs to use specified therapeutic pharmaceutical agents approved by the Food and Drug Administration.

Additionally, the bill would remove limitations on the types of diagnostic tests an optometrist could order.

D-KAT Digital

The first FDA 510(k) digital applanation tonometer

40H Slit Lamp

The next generation slit lamp featuring advanced Keeler optics

Vantage Plus Wireless

The world's best selling binocular indirect ophthalmoscope

MULTIPLY YOUR SAVINGS

BUY MORE SAVE MORE

Keeler's 40H Slit Lamp is quickly gaining the reputation as a world class device.

Featuring Keeler's famous optics, 6 to 40x magnification, LED Illumination in a beautifully crafted, thoughtful design, giving you the functionality and performance you want, need and expect from a Keeler Slit Lamp.

BUY 1 / GET 1
Purchase a Keeler 40 H Slit Lamp and you'll receive a FREE Keeler D-KAT Digital

BUY 2 / GET 2
Purchase 2 Keeler 40H Lamps and you'll receive 2 FREE Keeler D-KAT Digitals

BUY 3 / GET 3 + 1
Purchase 3 Keeler 40H Slit Lamps and you'll receive 3 FREE Keeler D-KAT Digitals + a FREE Keeler Vantage Plus BIO

Outfit your offices with Keeler 40H Slit Lamps before October 31, 2014 and take advantage of these great offers!

Offer valid: May 1st to October 31, 2014. No other Keeler offers can be combined.

Keeler OPTICS

Keeler Instruments, Inc. • 456 Parkway • Broomall, PA 19008
Tel: (800) 523-5620 • Fax: (610) 353-7814 • email: keeler@keelerusa.com

Tracking IOP With an IOL

Two new prototype intraocular lens devices aim to continuously measure intraocular pressure from the inside of the eye for glaucoma patients.

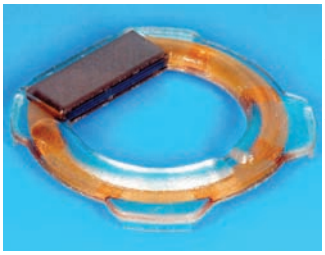


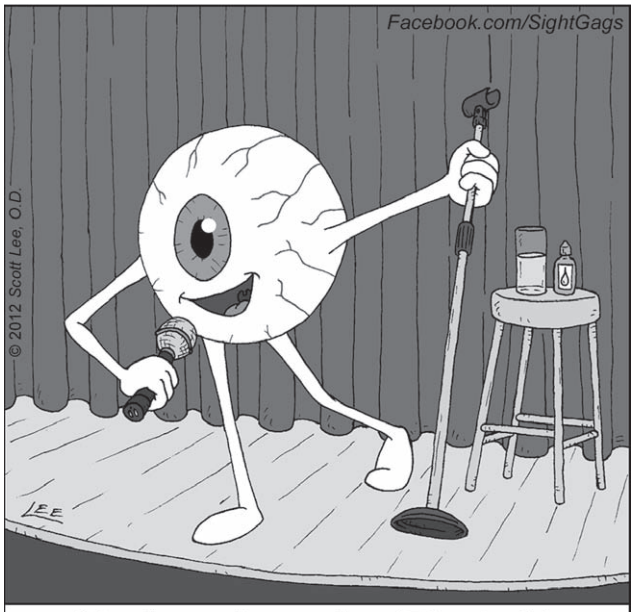
Photo: Implandata Ophthalmic Products GmbH

Pro-IOP Sensor.

Researchers at Stanford University and Bar-Ilan University in Israel are jointly developing an IOL implant that would allow patients to use a smartphone or Google Glass to check their IOP level.

Meanwhile, German-based Implandata Ophthalmic Products is working on the Pro-IOP, an implant that is powered telemetrically by a hand-held device. The patient or doctor would obtain IOP data from either the device or a smartphone app. ■

Sight Gags by Scott Lee, OD



"So the patient asks, 'What's this floater doing in my eye?' and the doctor says, 'The backstroke!' Thank you! Now that's what I call vitreous humor!"

REVIEW OF OPTOMETRY

Jobson Professional Publications Group

BUSINESS OFFICES
11 Campus Blvd., Suite 100
Newtown Square, PA 19073

CEO, INFORMATION SERVICES GROUP
Marc Ferrara
(212) 274-7062 • mferrara@jobson.com

PUBLISHER
James Henne
(610) 492-1017 • jhenne@jobson.com

SALES MANAGER, SOUTHEAST, WEST
Michele Barrett
(610) 492-1014 • mbarrett@jobson.com

VICE PRESIDENT, OPERATIONS
Casey Foster
(610) 492-1007 • cfoster@jobson.com

VICE PRESIDENT, CLINICAL CONTENT
Paul M. Karpecki, OD, FAAO
pkarpecki@jobson.com

PRODUCTION MANAGER
Scott Tobin
(610) 492-1011 • stobin@jobson.com

SENIOR CIRCULATION MANAGER
Hamilton Maher
(212) 219-7870 • hmaher@jhihealth.com

CLASSIFIED ADVERTISING
(888) 498-1460

SUBSCRIPTIONS
$56 a year, $88 (US) in Canada,
$209 (US) in all other countries.

SUBSCRIPTION INQUIRIES
(877) 529-1746 (US only);
outside US, call (847) 763-9630

CIRCULATION
PO Box 2025
Skokie, IL 60076
Tel: (toll free) 1-877-529-1746
Outside US: (847)763-9630
Fax: (847)763-9631

jmi

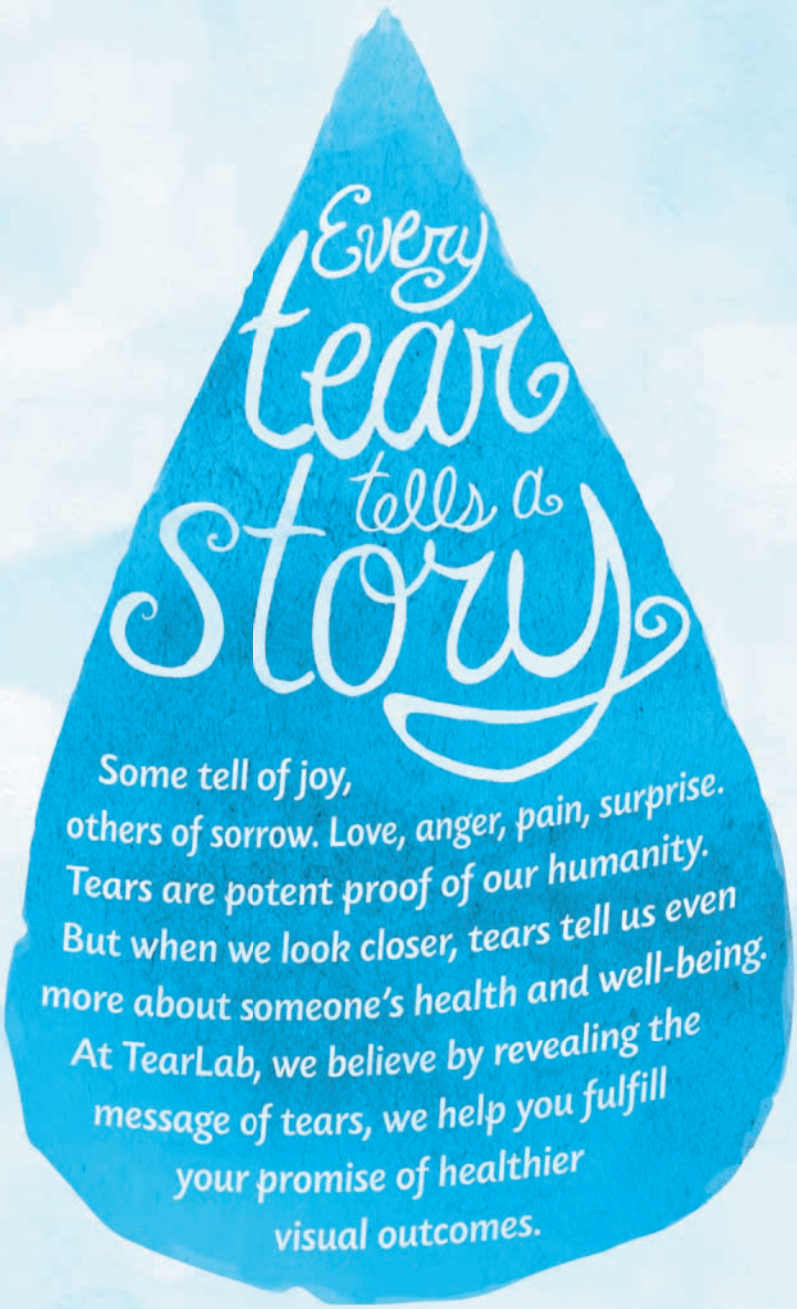
CEO, INFORMATION SERVICES GROUP
Marc Ferrara

SENIOR VICE PRESIDENT, OPERATIONS
Jeff Levitz

SENIOR VICE PRESIDENT, HUMAN RESOURCES
Lorraine Orlando

VICE PRESIDENT, CREATIVE SERVICES & PRODUCTION
Monica Tettamanzi

VICE PRESIDENT, CIRCULATION
Emelda Barea



Join us at Academy 2014 and see how together we can harness the power of tears.

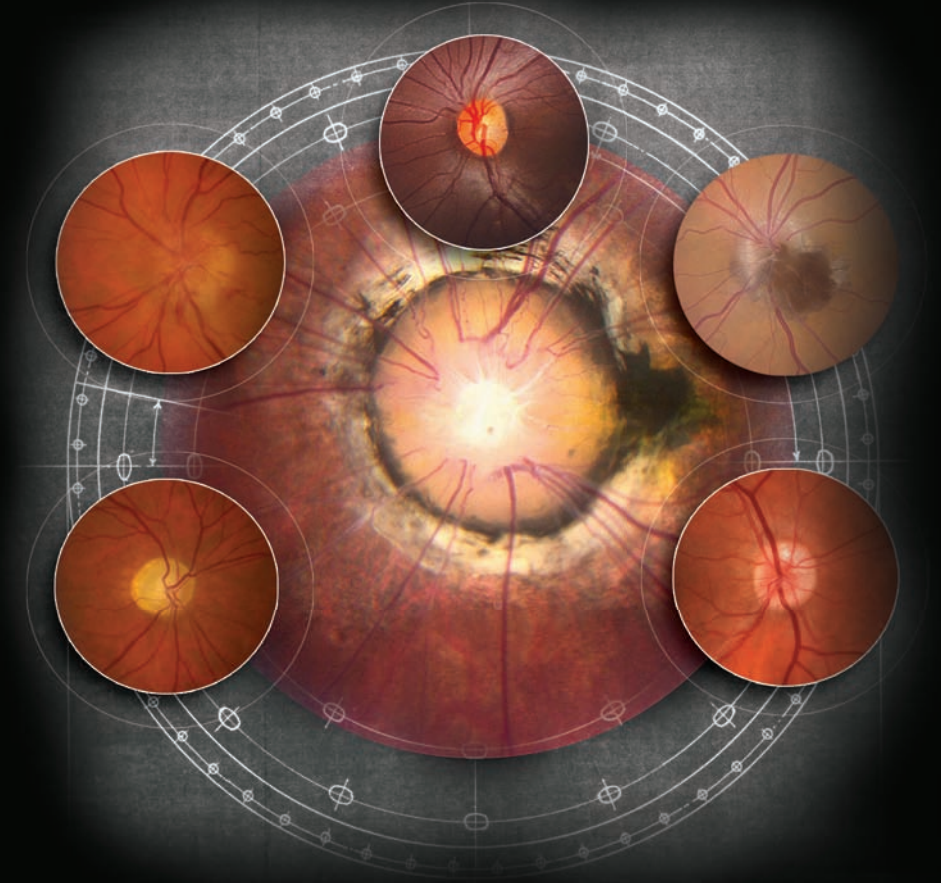
TearLab®

SOMETIMES DREAMS NEED A PARTNER

- Up to 100% Financing
- Dedicated Construction Team
- Construction Lending Experts
- Other Financing Options include:
  Acquisitions, Expansions & Refinancing

LIVE OAK BANK™

liveoakbank.com/healthcare • 877.890.5867

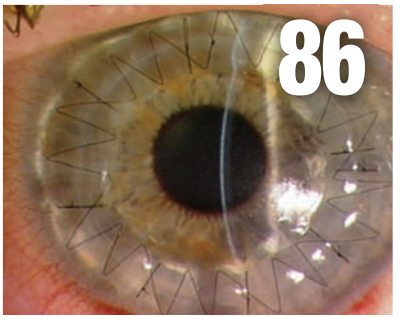


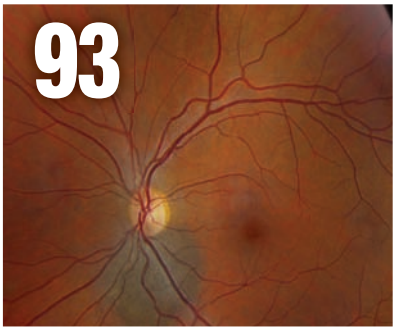
Contents

Review of Optometry September 2014

Departments

Review of Optometry September 2014





On The Web »

Check out our multimedia and continuing education @ *www.revoptom.com*

Digital Edition

Left your *Review of Optometry* at the office? No problem! Access *Review* on your computer or mobile device!

Go to www.revoptom.com and click on the digimag link to for the current issue.

Facebook and Twitter

For daily updates, "Like" our page on Facebook or "Follow" us on Twitter!

- www.facebook.com/revoptom
- http://twitter.com/#!/revoptom

REVIEW OF OPTOMETRY

PRINTED IN U.S.A.

FOUNDING EDITOR
FREDERICK BOGER
1891-1913

EDITORIAL OFFICES
11 CAMPUS BLVD., SUITE 100
NEWTOWN SQUARE, PA 19073
EMAIL • REVIEWOFOPTOMETRY@JOBSON.COM
WEBSITE • WWW.REVOPTOM.COM

SUBSCRIPTION INQUIRIES
1-877-529-1746
CONTINUING EDUCATION INQUIRIES
1-800-825-4696

EDITOR-IN-CHIEF • JACK PERSICO
(610) 492-1006 • JPERSICO@JOBSON.COM

EXECUTIVE EDITOR • JOHN MURPHY
(610) 492-1021 • JMURPHY@JOBSON.COM

MANAGING EDITOR • MICHAEL HOSTER
(610) 492-1028 • MHOSTER@JOBSON.COM

SENIOR ASSOCIATE EDITOR/WEB EDITOR • ERIN KELLY
(610) 492-1005 • EKELLY@JOBSON.COM

ASSOCIATE EDITOR • FRANK AULETTO
(610) 492-1043 • FAULETTO@JOBSON.COM

DIRECTOR ART/PRODUCTION • JOE MORRIS
(610) 492-1027 • JMORRIS@JOBSON.COM

ART DIRECTOR • JARED ARAUJO
(610) 492-1032 • JARAUJO@JOBSON.COM

DIRECTOR OF CE ADMINISTRATION • REGINA COMBS
(212) 274-7160 • RCOMBS@JOBSON.COM

SPECIAL PROJECTS • JANE COLE
(610) 492-1043 • JCOLE@JOBSON.COM

Jobson Professional Publications Group

JOBSON MEDICAL INFORMATION LLC

jmi

Restasis®
(Cyclosporine Ophthalmic Emulsion) 0.05%

For patients with decreased tear production presumed to be due to ocular inflammation associated with Chronic Dry Eye

THE DRY EYE TREATMENT SHE NEEDS TODAY. BECAUSE TOMORROW MATTERS.

RESTASIS® twice a day, every day, helps patients experience increased tear production

Increased tear production was seen at 6 months.[1]

Indication and Usage

RESTASIS® (cyclosporine ophthalmic emulsion) 0.05% is indicated to increase tear production in patients whose tear production is presumed to be suppressed due to ocular inflammation associated with keratoconjunctivitis sicca. Increased tear production was not seen in patients currently taking topical anti-inflammatory drugs or using punctal plugs.

Important Safety Information

Contraindications

RESTASIS® is contraindicated in patients with known or suspected hypersensitivity to any of the ingredients in the formulation.

Warnings and Precautions

Potential for Eye Injury and Contamination: To avoid the potential for eye injury and contamination, individuals prescribed RESTASIS® should not touch the vial tip to their eye or other surfaces.

Use With Contact Lenses: RESTASIS® should not be administered while wearing contact lenses. If contact lenses are worn, they should be removed prior to the administration of the emulsion.

Adverse Reactions

In clinical trials, the most common adverse reaction following the use of RESTASIS® was ocular burning (upon instillation)—17%. Other reactions reported in 1% to 5% of patients included conjunctival hyperemia, discharge, epiphora, eye pain, foreign body sensation, pruritus, stinging, and visual disturbance (most often blurring).

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

Reference: 1. RESTASIS® Prescribing Information.

ALLERGAN

APC20AF14 141005

RESTASIS® (Cyclosporine Ophthalmic Emulsion) 0.05%

BRIEF SUMMARY—PLEASE SEE THE RESTASIS® PACKAGE INSERT FOR FULL PRESCRIBING INFORMATION.

INDICATION AND USAGE

RESTASIS® ophthalmic emulsion is indicated to increase tear production in patients whose tear production is presumed to be suppressed due to ocular inflammation associated with keratoconjunctivitis sicca. Increased tear production was not seen in patients currently taking topical anti-inflammatory drugs or using punctal plugs.

CONTRAINDICATIONS

RESTASIS® is contraindicated in patients with known or suspected hypersensitivity to any of the ingredients in the formulation.

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination

To avoid the potential for eye injury and contamination, be careful not to touch the vial tip to your eye or other surfaces.

Use with Contact Lenses

RESTASIS® should not be administered while wearing contact lenses. Patients with decreased tear production typically should not wear contact lenses. If contact lenses are worn, they should be removed prior to the administration of the emulsion. Lenses may be reinserted 15 minutes following administration of **RESTASIS®** ophthalmic emulsion.

ADVERSE REACTIONS

Clinical Trials Experience

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

In clinical trials, the most common adverse reaction following the use of **RESTASIS®** was ocular burning (17%).

Other reactions reported in 1% to 5% of patients included conjunctival hyperemia, discharge, epiphora, eye pain, foreign body sensation, pruritus, stinging, and visual disturbance (most often blurring).

Post-marketing Experience

The following adverse reactions have been identified during post approval use of **RESTASIS®**. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.

Reported reactions have included: hypersensitivity (including eye swelling, urticaria, rare cases of severe angioedema, face swelling, tongue swelling, pharyngeal edema, and dyspnea); and superficial injury of the eye (from the vial tip touching the eye during administration).

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects: Pregnancy Category C

Adverse effects were seen in reproduction studies in rats and rabbits only at dose levels toxic to dams. At toxic doses (rats at 30 mg/kg/day and rabbits at 100 mg/kg/day), cyclosporine oral solution, USP, was embryo- and fetotoxic as indicated by increased pre- and postnatal mortality and reduced fetal weight together with related skeletal retardations. These doses are 5,000 and 32,000 times greater (normalized to body surface area), respectively, than the daily human dose of one drop (approximately 28 mcL) of 0.05% **RESTASIS®** twice daily into each eye of a 60 kg person (0.001 mg/kg/day), assuming that the entire dose is absorbed. No evidence of embryofetal toxicity was observed in rats or rabbits receiving cyclosporine at oral doses up to 17 mg/kg/day or 30 mg/kg/day, respectively, during organogenesis. These doses in rats and rabbits are approximately 3,000 and 10,000 times greater (normalized to body surface area), respectively, than the daily human dose.

Offspring of rats receiving a 45 mg/kg/day oral dose of cyclosporine from Day 15 of pregnancy until Day 21 postpartum, a maternally toxic level, exhibited an increase in postnatal mortality; this dose is 7,000 times greater than the daily human topical dose (0.001 mg/kg/day) normalized to body surface area assuming that the entire dose is absorbed. No adverse events were observed at oral doses up to 15 mg/kg/day (2,000 times greater than the daily human dose).

There are no adequate and well-controlled studies of **RESTASIS®** in pregnant women. **RESTASIS®** should be administered to a pregnant woman only if clearly needed.

Nursing Mothers

Cyclosporine is known to be excreted in human milk following systemic administration, but excretion in human milk after topical treatment has not been investigated. Although blood concentrations are undetectable after topical administration of **RESTASIS®** ophthalmic emulsion, caution should be exercised when **RESTASIS®** is administered to a nursing woman.

Pediatric Use

The safety and efficacy of **RESTASIS®** ophthalmic emulsion have not been established in pediatric patients below the age of 16.

Geriatric Use

No overall difference in safety or effectiveness has been observed between elderly and younger patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenesis: Systemic carcinogenicity studies were carried out in male and female mice and rats. In the 78-week oral (diet) mouse study, at doses of 1, 4, and 16 mg/kg/day, evidence of a statistically significant trend was found for lymphocytic lymphomas in females, and the incidence of hepatocellular carcinomas in mid-dose males significantly exceeded the control value.

In the 24-month oral (diet) rat study, conducted at 0.5, 2, and 8 mg/kg/day, pancreatic islet cell adenomas significantly exceeded the control rate in the low-dose level. The hepatocellular carcinomas and pancreatic islet cell adenomas were not dose related. The low doses in mice and rats are approximately 80 times greater (normalized to body surface area) than the daily human dose of one drop (approximately 28 mcL) of 0.05% **RESTASIS®** twice daily into each eye of a 60 kg person (0.001 mg/kg/day), assuming that the entire dose is absorbed.

Mutagenesis: Cyclosporine has not been found to be mutagenic/genotoxic in the Ames Test, the V79-HGPRT Test, the micronucleus test in mice and Chinese hamsters, the chromosome-aberration tests in Chinese hamster bone-marrow, the mouse dominant lethal assay, and the DNA-repair test in sperm from treated mice. A study analyzing sister chromatid exchange (SCE) induction by cyclosporine using human lymphocytes *in vitro* gave indication of a positive effect (i.e., induction of SCE).

Impairment of Fertility: No impairment in fertility was demonstrated in studies in male and female rats receiving oral doses of cyclosporine up to 15 mg/kg/day (approximately 2,000 times the human daily dose of 0.001 mg/kg/day normalized to body surface area) for 9 weeks (male) and 2 weeks (female) prior to mating.

PATIENT COUNSELING INFORMATION

Handling the Container

Advise patients to not allow the tip of the vial to touch the eye or any surface, as this may contaminate the emulsion. To avoid the potential for injury to the eye, advise patients to not touch the vial tip to their eye.

Use with Contact Lenses

RESTASIS® should not be administered while wearing contact lenses. Patients with decreased tear production typically should not wear contact lenses. Advise patients that if contact lenses are worn, they should be removed prior to the administration of the emulsion. Lenses may be reinserted 15 minutes following administration of **RESTASIS®** ophthalmic emulsion.

Administration

Advise patients that the emulsion from one individual single-use vial is to be used immediately after opening for administration to one or both eyes, and the remaining contents should be discarded immediately after administration.

Rx Only

ALLERGAN

Based on package insert 71876US17

Visit us at Vision Expo West Sep. 18-20 Booth #MS7059

YOUR EXAM... ONLY BETTER!

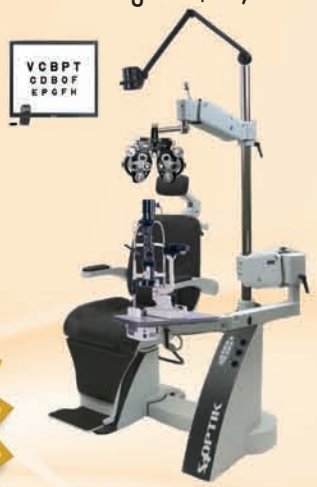
The Veatch Digital Refraction System eliminates costly inefficiencies, enhances your practice, and makes the exam process more enjoyable for you and your patients.

FREE Visual Acuity System* with purchase of Digital Refractor

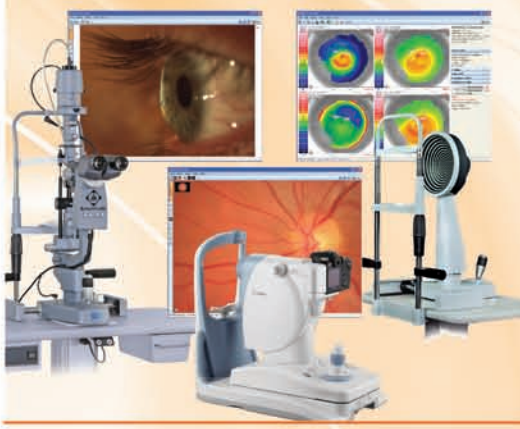
- Increased efficiency • Combat reductions in reimbursements
- Enhanced practice image • EMR integration • Patient referrals
- Increased optical sales • Competitive advantage
- Eliminate back and shoulder pain and bursitis

ADDITIONAL OFFERS FROM VEATCH

Exam Lane Packages
Starting at $12,995



ReSeeVit™
- Anterior Segment Imaging
- Retinal Imaging
- Corneal Wavefront Topography



Insight Visual Acuity System
ONLY $1,295

VEATCH OPHTHALMIC INSTRUMENTS

TOLL FREE: 800.447.7511 E-MAIL: ad@voi2020.com www.voi2020.com

I Know What You Did Last Summer

The 2013 Sunshine Act data is about to go public. Are you ready for the big reveal?

By Jack Persico, Editor-in-Chief

Breaking news: On August 16, 2013, a major pharmaceutical company provided a lunch buffet of some kind—unconfirmed reports indicate deli sandwiches—for the medical staff of a doctor's office. The Diet Coke was described as "flat," sources familiar with the matter reveal. The quality of the corned beef, reputed to be "New York's finest," could not be determined at press time. One high-value target demanded, and was given access to, a brownie. Updates will be posted as the story develops.

Those are the sort of revelations I expect we'll see at the end of this month. That's when the 2013 physician payment data collected as per the so-called Sunshine Act is slated to be released—nine to 14 months after the fact, which is considered breakneck speed for a federal agency but woefully outdated by everyone else.

Is there any value in knowing what happened between doctors and industry a year ago, especially when so much of it is mundane? CMS seems to think so. Fasten your seat belts—I expect this to be about as juicy as the opening of Al Capone's vault.

Fun in the Sun

The Sunshine Act stipulates that payments of $10 or more from industry to a doctor must be disclosed to CMS and, eventually, to the public. But even payments below $10 must still be tracked; if the cumulative annual total is $100 or more, that triggers a report too.

After a few false starts, the law went into effect early last year. Data on transactions from August 1 to December 31, 2013 will be released to the public on September 30, 2014; the site will be updated annually on June 30 thereafter.

I can think of at least two reasons why the Sunshine Act would offend me if I were in your shoes.

First, the fundamental premise of the Act is to assume that you can be bought—that your clinical decisions are susceptible to influence from relationships cultivated with industry, even if there's no direct quid-pro-quo arrangement. Any person with a good code of ethics would find that inherently offensive, but especially a doctor sworn to put patients' best interest first.

Secondly, the Act might interfere with the care you provide, by driving a wedge between industry and practitioners. Manufacturers need doctors to give them feedback on their products, and clinicians need the expertise of a drug or device's creators to make the best possible use of it in practice. Those goals are served by open, unfettered dialog.

If doctors get spooked by the negative connotations that surround a public "outing" of their business dealings, or manufacturers (who face stiff penalties for noncompliance) feel they must walk on eggshells in the presence of doctors, routine communication will dry up. It might also jeopardize research programs and educational initiatives that rely on industry support. Who benefits then?

Lastly, consider how arbitrary implementation seems to be.

If you request data on a drug's performance and the company provides article reprints that have monetary value, those could trigger a report if they cross the $10/$100 threshold. Anything that constitutes a "transfer of value" must be tracked. But professional samples, product rebates and patient education materials don't. Promotional items at an exhibit booth don't need to be tracked because they're made available to all. But if given during a one-on-one visit, they do.

Confused? No worries. CMS has published a handy "user guide" to answer your questions. It weighs in at a svelte 359 pages. Brave souls can find it here: www.cms.gov/Regulations-and-Guidance/Legislation/National-Physician-Payment-Transparency-Program/Downloads/Open-Payments-User-Guide-%5BJune-2014%5D.pdf.

With even the most humdrum physician/industry relations being tracked, you're subject to Sunshine whether you know it or not. And all this assumes that patients will even bother to look for the data.

Good Intentions, Bad Policy

Certainly, there are legitimate concerns about industry influence in medicine, and guidelines regarding disclosure of financial interest are both necessary and welcome. They're also already the norm. Adding another layer of cumbersome oversight and reporting brings clouds, not sunlight. ■

19TH ANNUAL

OPHTHALMIC PRODUCT GUIDE

Innovative products to enhance your practice

The **future** is in your hands. One tap, many possibilities.

Experience the digital edition on your handheld device. Use your smart device to scan the code below or visit:

www.revoptom.com/supplements/

Download a QR scanner app. Launch app and hold your mobile device over the code to view http://www.revoptom.com/supplements

REVIEW® OF OPTOMETRY

Feeding Your Inner Fraidy Cat

Optometrists all seem to be living in a constant state of fear. I'm no different. Just like you, I put on my scaredy pants one leg at a time. **By Montgomery Vickers, OD**

Optometrists always seem a little afraid of things. We're afraid of health care reform, online glasses and contact lenses, our office manager (well, at least *I am* after being married to her for nearly 34 years), the IRS, HIPAA, meaningful use, larger-than-average optic discs, thinner-than-average corneae, antitrust laws, state boards, national boards, surfboards, and on and on.

Dude, we are *supposed* to be scared! Cavemen who weren't scared to death by every little thing were the ones who were eaten by the saber-toothed tiger. Our bravery genes were chewed and swallowed and lost forever.

Studies have proven that the physiological response to fear in the body is the same exact physiological response to excitement. Therefore, if you are excited about your chosen profession, then you are also scared of it. And vice-versa.

Fear Itself

Now, I don't spend a lot of time actually feeling fear in the office, but there are a few things that make me want to run down the hallway screaming and flailing my hands like my hair is on fire. One obvious example is if my hair were, in fact, on fire. But this has only happened a handful of times in my practice, so no big deal.

What else scares me? Well, one example is anything involving plumbing. In 34 years, I've never found a staff member who can be trained properly to jiggle the handle when the toilet keeps running. They're afraid of it, too. They'll interrupt me when I'm in the middle of needling a rust ring to have me deal with that one.

What about a 4-year-old who really, *really* needs cycloplegia? (This is why my son loves being a surgeon—the patients are unconscious.) I used to just muscle the kid into submission. But since I turned 61, the average 4-year-old can now whoop me, so that doesn't work any more.

How about the abject fear produced when a 59-year-old former Air Force pilot who's +1.00-1.25 x 056 shows up and wants contact lenses? I would rather face him in his F-18 than in my chair.

And, *eeeeeoowww*, the "my-last-doctor-just-gave-me-a-bunch-of-contact-lenses-so-why-won't-you?" patient. I try to explain that contact lenses are like meth, and that's not free, right? This kind of patient usually understands this simple analogy, and then tries to pay me with meth IOUs.

I scream at the sight of a juice box in the hands of any 2-year-old. Might as well just take it from the toddler and dump it on my new shirt myself.

How about the "where's my checkbook" fear when an order comes in COD because I'm 90 days behind on the last order of $12.37? Terrifying. (By the way, where *is* my checkbook anyhow?)

I get petrified when we have a lunch staff meeting and they all want food from the healthy vegetarian salad place up the street. I'm afraid I'll need IV glucose by 2 PM.

Speaking of my staff, I live in mortal fear of running out of bottled water and being on the receiving end of *The Look*. You know The Look. It's the one they teach all females to make any man feel like a stupid idiot. My daughter could peel paint off the walls with one glance when she was 7. Now that she's a mom, I'll only look at her through a welder's helmet.

Doctors, what are YOU afraid of? Just pretend the patient is unconscious. Works every time! ■

Don't just meet the needs of your patients– **exceed them.**

New Bausch + Lomb PeroxiClear™– the most advanced* peroxide solution.

	PeroxiClear™	Clear Care
Delivers Exceptional Disinfection	✓	✓
Preservative Free	✓	✓
Provides Superior All-Day Comfort[1,2*]	✓	
Keep Lenses Cleaner and Moister for Longer[1-5*]	✓	
Lenses Are Ready to Wear in Only 4 Hours	✓	

Switch your patients to the most advanced* peroxide solution.
Request your care kits today at Bausch.com/Peroxiclear.

*Compared to Clear Care

REFERENCES: 1. Results from a 22-investigator, multi-site study of PeroxiClear™, with a total of 440 eligible subjects. Subjects were randomized to use either PeroxiClear™ or Clear Care for 3 months. Subjects completed performance surveys at 2-week, 1-month, 2-month, and 3-month visits. **2.** Results from a 21-investigator, multi-site study of PeroxiClear™, with a total of 297 eligible subjects who were habitual Clear Care users. After 7 days of use, subjects completed an online survey. Consumers rated the performance of PeroxiClear™ across a range of attributes and compared the performance to their habitual Clear Care solution. **3.** High-resolution/accurate-mass (HR/AM) mass spectrometry was used to detect and quantitate the relative amounts of surfactant retained on lenses from PeroxiClear™ and Clear Care solutions after 20 hours of wear. PureVision®2, ACUVUE OASYS, and AIR OPTIX AQUA lenses were soaked in solutions for 12 hours prior to patients wearing lenses for 20 hours. **4.** Results of an in vitro study measuring deposits on ACUVUE OASYS lenses. Lenses were subjected to 14 cycles of deposition with a lipid and protein solution mimicking the human tear film followed by a cleaning regimen with either PeroxiClear™ or Clear Care 3% hydrogen peroxide systems. Each deposition/cleaning cycle was representative of one day of patient use. Cycled lenses (n=3) were analyzed for deposits using image analysis. After 14 cycles, lenses cleaned with PeroxiClear™ had only 8.0% surface coverage compared to 33.0% for lenses cleaned with Clear Care. **5.** Results of an ex vivo study measuring deposits on worn contact lenses to compare the clinical performance of PeroxiClear™ and Clear Care solutions. Lenses were worn daily for 1 month (silicone hydrogel and Group IV hydrogel lenses) or 3 months (gas permeable lenses). A total of 374 lenses were randomly selected for image analysis. Lenses were scored for mean density of deposits and percent coverage of deposits.

BAUSCH+LOMB
See better. Live better.

Don't Cry Over Dry Eye

A specialty contacts practice can insulate you from the commodity-based mindset of disposable lenses. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

Keeping our patients' ocular surface healthy and comfortable sounds simple, but in fact can be very challenging. The ocular surface is involved in most things that we do: contact lenses, refractive care, glaucoma, cataracts and refractive surgery.

Though ocular surface disease (OSD) itself can be complicated, coding for ocular surface diagnostic care and ongoing management is very simple and straightforward. It generally consists of a combination of office visits and specific clinical tests. Many of the traditional diagnostic tests performed for "dry eye"—such as Schirmer, phenol red thread, tear film break-up time and tear prism analysis—are usually considered to be part of the office visit and not separately identified by the CPT or HCPCS with their own codes.

However, some recent technologies—separately identified as a new CLIA-waived procedure and a Level III HCPCS code—can be coded in addition to the office visit:

• *83516*. The currently available product is InflammaDry (Rapid Pathogen Screening), and the test is defined by CPT as: "Immunoassay for analyte other than infectious agent antibody or infectious agent antigen; qualitative or semiquantitative, multiple step method," is now a CLIA-waived, in-office test. (So, for each procedure performed, use the modifier –QW, which marks it as a CLIA-waived test.) It must be done in a practice that has a clinical lab designation and with a physician who has been registered as a clinical lab director. The current national reimbursement amount for this lab test is $15.52 per eye.

• *0330T*. "Tear film imaging, unilateral or bilateral, with interpretation and report." This code for tear film interferometry—marketed as LipiView (TearScience)—is a Level III HCPCS code, which means it is used to track utilization of new technology and needs to be reported to the carrier. As such, there is no reimbursement associated with this code, and the patient generally pays for it. Because 0330T now defines this service more explicitly, coordinating rules require that this code must be used instead of CPT code 92285 (anterior segment photography).

CPT for OSD is A-OK

When coding the office visit, remember that there is no difference between dry eye, cataracts or even a retinal problem in the sense that they are all recognized disease processes that require proper anatomical assessment of structure and function. So, whenever you perform medical eye services, the first area of the medical record you should assess is the chief complaint (CC). The CC must reflect the reason for the visit; it comes either from the patient as a complaint or symptoms of an eye disease or injury, or from your directive to return to the office for a very specific reason.

Determining the specific CPT code to use is fairly simple. Of the 920XX codes, most likely only the intermediate codes, 92002 or 92012, would be appropriate; however, keep in mind that if you use a 92012, the patient must present with a new condition or an existing condition complicated with a new diagnostic or management problem not necessarily related to the primary diagnosis. In other words, as most patients in this scenario are established patients, and unless the patient presents with a new condition or additional complications relating to dry eye not previously noted, 92012 is not appropriate to use because the specific definition of this code has not been met.

That leaves the 992XX codes. The beauty of the 992XX codes is that they are structure driven. You, as the physician, determine the clinically relevant and pertinent anatomical areas to examine and evaluate. The level of history and medical decision-making are fairly specific and limited in most cases. For OSD, the codes used in the 992XX system are 99201, 99202, 99212 and 99213. Always code each patient encounter by the individual case presentation and the individual patient you're examining and treating.

Managing the ocular surface is professionally rewarding, a lifesaver for your patients, and a tremendous practice builder. Keep these clinical skills and coding guidelines top of mind and all areas of your practice will benefit. ■

Please send your questions to CodingAbstract@gmail.com.

EXCEED YOUR PROGRESSIVE LENS WEARERS' EXPECTATIONS

Give them the sharpest vision at every distance

Presbyopia is unavoidable, but on the bright side, companies are continually improving ways for us all to deal with it. Case in point: W.A.V.E. Technology: Wavefront Advanced Vision Enhancement™ found in Essilor's Varilux® Physio® and Varilux S Series™ lenses.

Jesse Rossow, OD, has been in practice for a little over a year and is one of four optometrists at Kennedy Vision Health Center in Minnesota. He says the practice stands apart from others in the area simply based on the quality of the procedures they perform and the products they sell.

Dr. Rossow estimates that close to 40% of the practice's patients are presbyopic. Interestingly, roughly 90% of them wear *Varilux Physio* and *Varilux S Series* lenses. Here, he tells why his practice relies almost exclusively on these lenses to meet the needs of its presbyopic patients.

GIVE THE BEST TO BE THE BEST

"We pretty much sell Varilux® lenses exclusively, unless a patient specifically requests something different," says Dr. Rossow. "Basically, we believe these lenses give the best vision possible."

Specifically, *W.A.V.E. Technology* identifies and eliminates wavefront aberrations in the lens. A feature of *Varilux Physio* lenses, it provides patients the sharpest vision at every distance with better contrast—near, far and in between.

Varilux S Series and Varilux Physio Enhanced™ lenses have W.A.V.E. Technology 2™, which not only eliminates lens aberrations, but also customizes the lens based on pupil size. As we know, pupil size changes to adjust to brightness. For example, at night, pupil size increases to allow more light in; however, this in turn decreases sharpness. Thanks to *W.A.V.E. Technology 2*, these lenses provide patients the sharpest vision at every distance with better contrast—near, far and in between—even in low light. The result? Patients experience safer, more confident night driving and less strain during low light reading, television viewing and computer use.

To Dr. Rossow, *W.A.V.E. Technology* means decreased aberrations, an increase in contrast sensitivity and a sharper, clearer, more comfortable field of view. "I think that's important because everyone wants more natural and better vision in all situations," he says.

> *"We believe these lenses give the best vision possible."*
> –Dr. Rossow

PATIENT INTERACTIONS

Dr. Rossow's practice values patient education, so when someone doesn't understand the concept of aberrations, he tries to keep it as simple as possible. "I'll explain that because the eyeglass lens surface is not perfect, it may have some blur-causing areas that affect vision," he says. "I then tell them that *Varilux* lenses are designed to reduce those blur-causing aberrations and allow for better vision."

W.A.V.E. Technology uses a patented design process that analyzes a full beam of light as it passes through each area of the lens to identify and remove small distortions.

In the exam room, Dr. Rossow and the other doctors mention some facts and benefits about the *Varilux* lenses, but he says the opticians themselves do most of the education. "They are very up to date on the technology and our reps have been great at keeping all of us up to date on recent studies, the technology and why it's better," he explains.

"We've had patients wearing lenses that they got elsewhere and aren't happy," he says, noting that patients often complain about how much they have to turn their heads. He always advises them to switch to a *Varilux* lens. "These patients are almost always happier with the performance, so I do talk about the decreased aberrations and the contrast sensitivity of *Varilux* lenses," he says.

HAPPY PATIENTS, HEALTHY PRACTICE

We all know that happy patients make for a healthy practice. Dr. Rossow says that switching progressive wearers to a *Varilux* lens design results in a satisfied customer, but are there other reasons to embrace this brand? Absolutely! According to Dr. Rossow, using *Varilux* lenses almost exclusively in the practice has decreased their nonadapts and returns. And, compared to other lenses, he says the adaptation/transition process is easier and shorter with *Varilux*.

Take all of this together and it results in greater profitability. Dr. Rossow explains, "Patients are very happy with these lenses, which limits their returns, but also keeps them coming back to purchase lenses with us because of the quality."

GO FORTH & PLEASE PRESBYOPES

"People are generally happier with this lens than they are with other designs," says Dr. Rossow. A recent patient told him how impressed she was with the performance of her *Varilux* lenses at one of her favorite restaurants. She used to struggle reading the menu, but ate there so frequently, she would simply order what she knew she liked. With her new *Varilux* lenses, however, she decided to take a look at the menu and immediately noticed a huge difference. She was able to read not only the dishes, but the ingredients and details for each item.

Coincidentally, Essilor's recently launched TV commercial takes place in a restaurant setting. It calls out to progressive lens wearers looking for smooth transitions and sharp vision at any distance, informing them that with the *Varilux* brand, they never have to compromise their vision again.

"Think about your patients needs and desires," advises Dr. Rossow. "Do you think they want the best vision possible? I think so. Essilor makes the choice easy with its *W.A.V.E. Technology* and *Varilux* lenses."

Dr. Rossow is in group practice at Kennedy Vision Health Center in Minnesota and is a member of the American Optometric Association.

A Lesson Plan for Pediatric Practice Building

Here's a five-step plan to help you to expand your practice with pediatric eye care services. **By Nathan Bonilla-Warford, OD, and Beth Knighton, OD**

Are you not seeing as many patients you'd like? Many practices aren't. These same practices are feeling the squeeze of reduced reimbursements. What many optometrists may not realize is that there is a steady stream of potential patients already in your office: the kids that tag along with mom, dad, grandma or granddad.

While some practitioners think that pediatric service is not a profitable niche in optometry, many doctors (like us) have found the opposite to be true. Here's how you can analyze your practice to determine if adding pediatric care is right for you.

For pediatric exams, turn tests into games, says Stephanie Lyons, OD. Kids make up half of the patient base at her Chicago-area practice.

'A Crying Need'

There are four main reasons why optometrists are now expanding their pediatric care, either within their existing office or by opening a second office:

- Medical reimbursements and retail revenue are largely declining.
- Increased competition requires optometrists to do more to stand

ELEVATE

A whole new slit lamp experience is coming.

THE PROBLEM: The most challenging of patients to examine are the ones in greatest need of care.

THE SOLUTION: Introducing the HAI SL-5000e, a new slit lamp by HAI Labs. Developed through years of input from doctors, the SL-5000e's streamlined foundation is ergonomically adapted to fit plus-sized patients and patients with limited mobility without compromising on features or image quality.

SCAN or visit
www.hailabs.com/elevate
to pre-order and save.

HAI Laboratories, Inc.

LAUNCHING AT AAO 2014.

YEAR-END 2014 SHOW SPECIAL PRICES

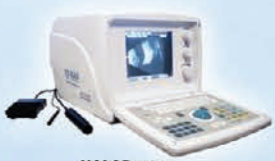
	LIST PRICE	SHOW PRICE
Basic Exam Lane Package*	~~$12,998~~	$11,500
SL-5000p Plus Slit Lamp	~~$6,300~~	$4,850
SL-5000s Standard Lamp	~~$4,800~~	$2,800
SL-5000b Basic Slit Lamp	~~$3,800~~	$1,950
SL-5000h Handheld Slit Lamp	~~$3,150~~	$1,825
VC-170 17" LED Vision Chart	~~$2,500~~	$1,500
AB-6100 A/B Ultrasonic Scan	~~$12,000~~	$8,800

* **Includes:** HAI SL-5000b Basic Slit Lamp, HAI VC-170 17" LED Vision Chart, S4OPTIK CB-1600 Chair & Stand Combo and SL-Y100 Refractor

Visit Booth #615 at AAO 2014 Denver for these great deals on clearance.

Basic Exam Lane Package

HAI SL-5000h Handheld Slit Lamp

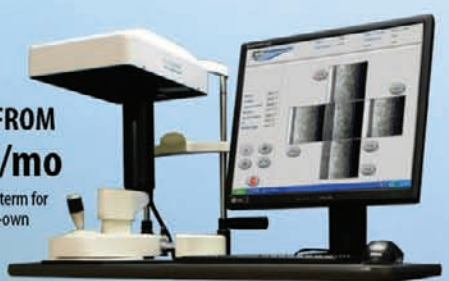


HAI AB-6100 A/B Ultrasonic Scan

FREE SCLERAL DEPRESSOR FOR THE FIRST 50 VISITORS TO OUR BOOTH

HAI Ophthalmic

RENT FROM $495/mo
39-month term for rent-to-own



HAI CL-1000nc Non-Contact Specular Microscope

HAI Ophthalmic is a division of HAI Laboratories, Inc., an ISO 13485-certified US manufacturer.

Create a separate waiting area for kids, but make it comfortable and appealing for adults also, as in the Little Eyes satellite office of Katherine Schuetz, OD, and Jeremy Ciano, OD, of Carmel, Ind. (Classic arcade games are optional.)

out from the crowd.

- The inclusion of the pediatric vision benefit of the Affordable Care Act has drawn a lot of attention to children's eye care.
- There are simply more kids due to a "baby boomlet" that occurred in the late 2000s.

As Mary Lou French, OD, of Children's Eyecare in Orland Park, Ill., puts it, "There is a crying need, in my opinion, for pediatric eye care, which I am afraid a lot of our colleagues do not address."

To respond to that need, follow this a five-step lesson plan to help you to strengthen your practice with pediatric eye care services.

Step 1: Decide Whether Pediatric Care is Right for Your Practice

Many factors come into play for a successful pediatric practice. Most obviously, there must be children. Look around the neighborhood for pediatricians, day care centers, karate studios and other businesses that cater to kids. If so, there are parents in your area who want the best for their kids, and that includes eye care. Unless a large number of practices already cater to kids in your area, this demographic is almost certainly underserved.

It's not necessary to have specific academic training to expand into pediatric vision care. Katherine Schuetz, OD, of Carmel, Ind., did not complete a pediatric residency, but she did have an affinity for kids and attended lectures and read journals. She and her partner, Jeremy Ciano, OD, noticed many pediatric dentists in their area, but no pediatric eye care. So they opened Little Eyes, a purely pediatric satellite office of their Revolution Eyes practice.

Before you do the same, decide if you or your associate doctors enjoy the company of children. If not, hire someone who does before you proceed. The same goes for staff. Optometric care of kids is easy and fun if the patient is relaxed and comfortable; otherwise, it becomes very difficult to get useful clinical data from them.

Dr. Schuetz recommends that the entire office needs to be committed to the same vision early on. If half of the staff disagrees about how to implement the new pediatric focus, there will be problems. Before launching the pediatric service, have full staff meetings so that everyone from the senior doctor to the newest tech understands this new and important direction for the practice.

Practice consultant Gary Gerber, OD, puts it this way, "We have many clients who have been financially successful with pediatric practices. What they all have in common is that none of them are dabblers. They have a singular focus on growing that segment of their practice. That sense of purpose and consistent work towards concrete goals are the reason they are doing so well."

You must also alter your office environment. Ideally, you should create a separate waiting area with colorful, kid-size furniture and toys, such as a LEGO table or coloring books, to keep little ones occupied and away from delicate frames. Make this part of the practice look kid-friendly and colorful, but be sure it's comfortable for adults, as well. You want it to be consistent with your overall decor of the practice, and not necessarily look like The Wiggles work there.

Importantly, you'll need to decide if the influx of younger patients will affect the mix of third-party plans you accept. If you take primarily medical plans, many of these plans will not cover specialty services and you'll have to explain this to parents. If you don't take medical plans, you may need to accept lower-reimbursing vision

ALLERGAN

We Bring More to Your World

- AllerganOptometry.com
- OPTOMETRY JUMPSTART™
- National and Regional Meeting Support
- Dedicated Sales Team
- Teaching and Residency Support
- Practice Management Resources

The Allergan Commitment to Optometry Is Stronger Than Ever. With new programs designed for doctors at every phase of their career, there are more ways for us to work together than ever before.

Visit AllerganOptometry.com to access a world of possibilities for your practice.

ALLERGAN

How We Built a Purely Pediatric Practice

By Nathan Bonilla-Warford, OD, and Beth Knighton, OD

My primary practice, Bright Eyes Family Vision Care, is about 40% pediatric. My associate, Beth Knighton, OD, and I both have completed residencies in pediatric vision and we see infants and children, including those with developmental disabilities and traumatic brain injury. Over the years, with virtually no external marketing, we grew our vision therapy practice to capacity. We wanted to expand, and decided to build a purely pediatric practice from scratch called Bright Eyes Kids.

We made a big event out of the grand opening of our Bright Eyes Kids office. A magician helped bring in a crowd and made it fun.

Opened in mid-March 2014, Bright Eyes Kids centers on specialty services for children, including vision therapy and ortho-k. This new office is located 17 miles away from our original office, and is closer to main highways for the convenience of our therapy patients who may travel one hour or more for regular therapy sessions. There are few vision therapy optometrists in our area, so easy access from the highway will make therapy more feasible for a lot of patients.

At Bright Eyes Kids, we have one exam room, two vision therapy rooms, a doctor's office, an optical area and a separate waiting area that we share with a music teacher in the same building. The optical has a large selection of pediatric frames, from infant to teen sizes, although we don't expect it to be a major profit center (besides covering the cost of optical staff). We have kid-created artwork on the walls as well as other cute glasses-related art (from http://eyepowerkidswear.com). In the waiting area, we have kid-themed reading material, such as *American Girl* and *Disney* magazines, a LEGO play table and coloring activity center.

We've received a good response from the limited advertising we've placed in local parenting magazines. We also send info to potential referral partners, such as occupational therapists and educational specialists. Additionally, we've been visiting local optometrists, pediatric ophthalmologists and pediatricians to see how our services can be helpful to them. We started off seeing one to two patients a day and now we're seeing eight to 10, including vision therapy sessions. We're serving an important patient population, building up our practice and having fun doing it.

plans to attract families.

Additionally, much has been made about the pediatric vision benefit of the Affordable Care Act, which took effect January 1, 2014. We haven't yet seen children flood into our offices because of this benefit, but we're ready for them if they do.

Step 2: Provide Primary Pediatric Care

If you decide to expand your pediatric care, primary eye care is a good place to start. While every optometrist has completed pediatric training, it's a good idea to review some basics. Start with the AOA Optometric Clinical Practice Guidelines, specifically the guidelines on pediatric care, amblyopia and accommodative and vergence disorders.

Modify your exam based on the age of patient. Children older than eight are examined essentially as adults, just with a special emphasis on the nearpoint visual skills needed for reading and writing in school.

Some equipment you'll need for the pediatric eye exam:

- Retinoscope and direct ophthalmoscope.
- Digital acuity chart that uses pediatric symbols and is capable of playing videos to hold their attention.
- Lang stereocards.
- Color Vision Testing Made Easy.
- Teller acuity cards or LEA grating paddles, Cardiff cards, LEA symbols or broken wheel cards.
- Toys, puppets and lights to use as fixation targets.
- Dilation spray or drops that can be applied with eyes closed to minimize patient anxiety.

What do you not want in the exam room? A white clinic coat. Kids tend to associate white coats with specialists, shots and other unpleasant things. Even pediatricians don't wear white coats. Also, move fragile items out of reach, and take down or hide medical educational material that might be too graphic or "gross."

Put kiddos at ease by starting off with jokes and questions about their favorite toys and movies. If you're a parent of little tykes, mine them for material and basic knowledge of what's popular at the moment.

The younger the patient is, the more you have to entertain and play with them, and even sing to them to keep their attention. Don't get too bogged down getting every measurement precise; keep moving and

UNMATCHED ACCURACY. **ANYWHERE.**

THE ONE & **ONLY RETINOMAX**

Whether in your office or on the road, the Retinomax hand-held autorefractor / keratometer provides unmatched accuracy and convenience in any testing environment.

Pretest with the gold standard portable autorefractor / keratometer, Retinomax K-Plus and ensure confidence in your test data with measurements that are acquired quickly, easily and accurately. See for yourself why we are called RIGHTon.

Righton

Contact S4OPTIK for more information.

S4OPTIK

5325 Cleveland Street, Suite 303 Virginia Beach, VA 23462
CALL NOW 1-888-224-6012 I www.s4optik.com

As attitudes about wearing glasses have evolved, so have lenses and frames. Although the parent will have the final call, kids now have many stylish options.

come back to a particular test if you need to complete the exam.

"We often describe the testing we perform on kids as games," says optometrist Stephanie Lyons, whose Chicago-based practice is 50% pediatric. Such games can be as simple as doing a high five every time the child gets a line correct on the acuity chart. Use toys for fixation targets that they "catch" by looking at. Turn Wirt circles into a whack-a-mole game; have the child press down—or "whack"—the circles as they pop up.

Optometrists who've never performed an infant or toddler eye exam are usually more afraid than the patient. Remember that you're looking for problems that will interfere with visual development. See how they respond to toys, momentary occlusion and light. Use preferential looking, retinoscopy, cover test and similar tests to quickly pick up on any problems.

Step 3: Stock Kids Glasses and Contacts

Pediatric frames and lenses are not as profitable as high-end adult frames, but they can be a money-maker. And these are not the plain frames that were available when you were a kid. They are fashionable, look like adult frames and some are nearly indestructible.

Kids are much more likely to accept glasses if they feel like they are part of the decision-making process. For instance, ask the child what their favorite color is and then have the parent pick two or three appropriately colored frames that suit their needs. Fortunately, glasses are extremely popular right now among children of all ages; picking out glasses for kids has never been more fun. Of course, their age and size will dictate the dimension and style of the frame, so have lines for all ages. (*See "Children's Optical: Little Tykes, Large Opportunity," September 2013.*)

Don't forget the important elements: polycarbonate or Trivex lens materials for safety, scratch protection and UV protection. When offered, many children choose photochromic lenses to reduce sensitivity to bright sun. Others prefer separate sunglasses with greater frame wrap and style. Also offer additional options that children may need, such as sports eyewear and prescription swim goggles.

As with frames, contact lenses have changed dramatically; they are safer, more convenient and cover more prescriptions than ever. While we used to think that children shouldn't use contacts until their teenage years, the CLIP study has shown that children as young as eight years old tolerate contacts well.[1] Older kids want contacts for activities such as sports, but contacts are important for any age in cases of high anisometropia.

For kids' contacts, patient motivation is absolutely key. If the parent wants contacts more than the child does, there is a poor chance of success. Demonstrating the comfort and handling of today's daily disposable contacts is easy to do in the exam chair, and will help make the child less apprehensive. Having the patient practice controlling blink reflex and handling the eyelids at home prior to the initial dispense helps greatly.

Step 4: Consider Specialty Pediatric Services

Any optometrist who sees many pediatric patients will encounter children with special needs, such as those with Down syndrome or autism spectrum disorders. Over time, you'll develop a comfort level but, in the beginning, by doing as much of the exam as possible and taking into account each patient's individual needs and challenges, you can help a great many of them, even without special training. Simply prescribing a lens that benefits them, such as bifocals (when indicated), may go a long way in improving their quality of life.

Other patients require additional care in the form of vision therapy to help with visual skills that they either did not develop or lost. While

Advanced Genotyping Reveals Future AMD Risk

RetnaGene™

Now, visionary science puts the power of prediction in your hands

RetnaGene™ is a portfolio of laboratory-developed genetic tests that provides a highly accurate and comprehensive risk assessment for advanced AMD.

For more information about the RetnaGene™ tests, please call a *my*Nicox concierge professional at **1.855.MY.NICOX** (1.855.696.4269), email **concierge@myNicox.com**, or visit **myNicox.com/RetnaGene**.

nicox Ophthalmic Diagnostics

Testing performed exclusively by: sequenom Laboratories

Sequenom and RetnaGene are trademarks of Sequenom, Inc. and are used with permission by Sequenom Center for Molecular Medicine, LLC, dba Sequenom Laboratories.

No test is perfect. The RetnaGene™ AMD and RetnaGene™ LR laboratory-developed tests are highly accurate, genetic tests. However, erroneous results may occur in rare cases.

The RetnaGene™ AMD and RetnaGene™ LR laboratory-developed tests were validated under federal laboratory guidelines by Sequenom Center for Molecular Medicine, dba Sequenom Laboratories, a wholly owned subsidiary of Sequenom, Inc. The tests have not been cleared or approved by the US Food and Drug Administration (FDA). Although laboratory-developed tests to date have not been subject to US FDA regulation, certification of the laboratory is required under CLIA to ensure the quality and validity of the test. Sequenom Laboratories is CAP-accredited and CLIA-certified to perform high-complexity clinical laboratory tests.

AMD=age-related macular degeneration; CAP=College of American Pathologists; CLIA=Clinical Laboratory Improvement Amendments.

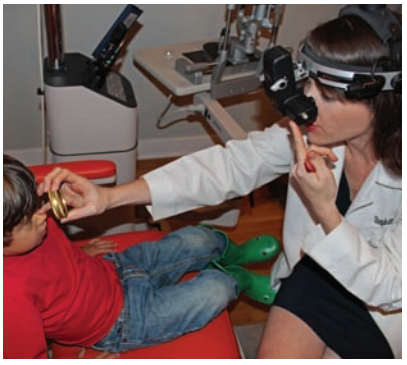
vision therapy can certainly benefit patients of all ages, the majority of vision therapy patients are kids, and it can be a major component of a pediatric practice.

While there are many avenues for post-doctoral training in vision therapy (most notably the College of Vision Development and the Optometric Extension Program Foundation), you can start growing the program with the most common and easily treated cases such as convergence insufficiency and amblyopia, for which there is a wealth of clinical research. As you gain comfort, you can take on more complicated cases, such as strabismus.

It's true that vision therapy is time consuming, but it is often provided on a fee-for-service basis and can be quite profitable once vision therapists are trained. Additionally, vision therapy patients tend to be the most loyal and the loudest word-of-mouth marketers for your practice. Many doctors maintain a separate practice for vision therapy. Vision therapy does require additional office space, but can be performed in a spare exam room.

Orthokeratology, also called ortho-k or corneal reshaping, is another profitable specialty service that patients will seek out. Ortho-k corrects refractive error in the short term so kids can be active without glasses or contacts. In the long term, it has been shown to reduce myopia progression.[2]

Myopia is a significant source of concern for many parents and doctors. But our understanding of myopiagenesis has improved greatly and, in addition to ortho-k, bifocal glasses, multifocal soft contacts and low-dose atropine have also been shown to reduce myopia progression.[3,4] "I don't think you can have a pediatric practice without offering some, if not all, of those services," says Paul Levine, OD, of Vision Care Specialists in Southborough, Mass.



Pediatric vision care is rewarding in more ways than one. "There's no better feeling than watching a child's eyes light up when they put on their first pair of glasses," Dr. Lyons says.

Also, if you invest in special diagnostic equipment, you'll not only serve your pediatric patients more effectively, you may also receive referrals for testing from fellow ODs and other professionals. Examples include visual evoked potential for objectively evaluating the vision pathway in cases of amblyopia and glaucoma, the Test of Variables of Attention (TOVA Company) for evaluating visual attention, and the Visagraph (Reading Plus) or ReadAlyzer (Compevo AB) for documenting eye movements.

Step 5: Get the Word Out

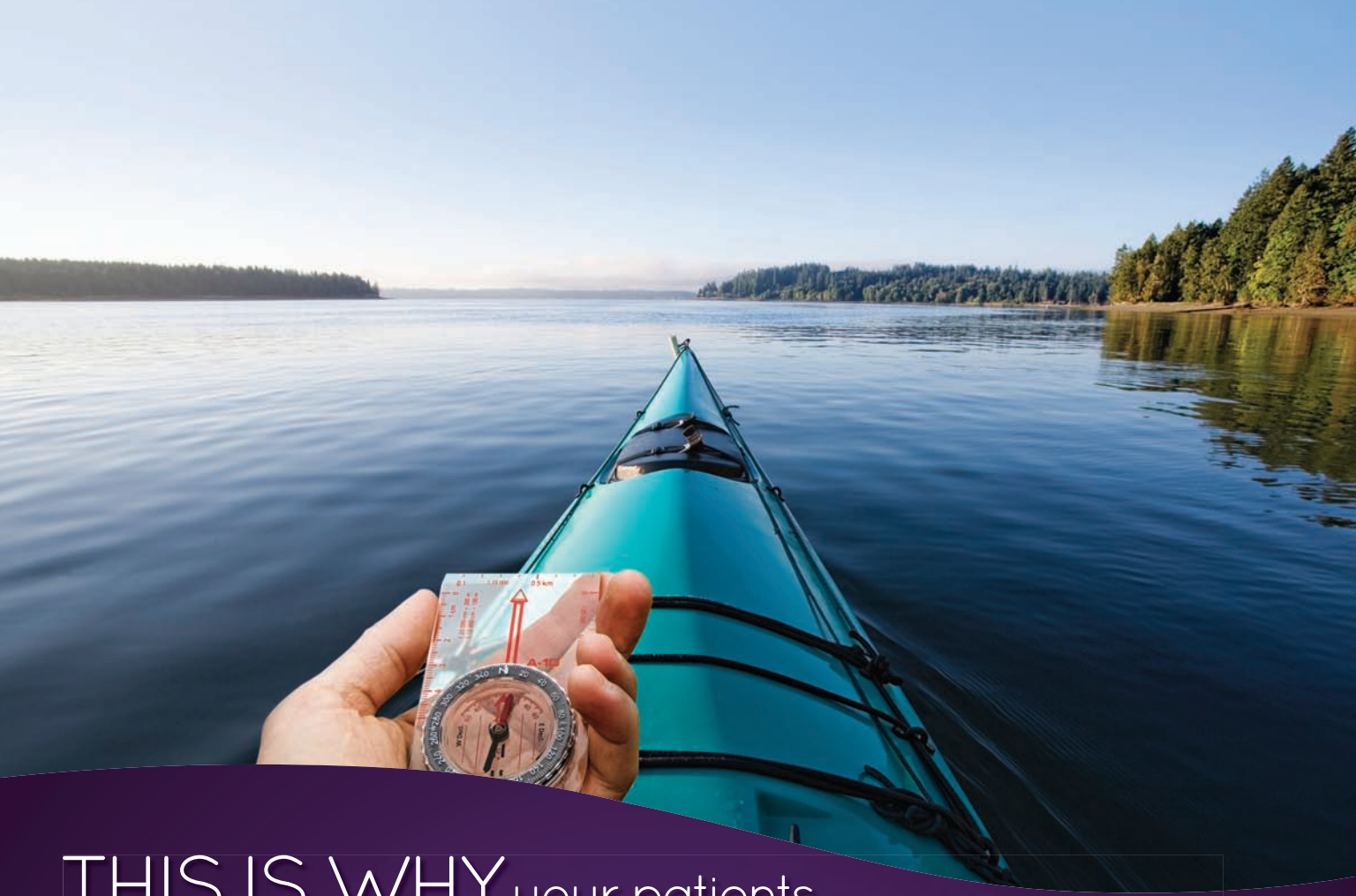
Promoting a pediatric practice or clinic within a larger practice is not much different than your current marketing approach. Start with internal marketing. As you know, many parents mistakenly think that if their kids have passed a school vision screening then they'll have no problem. So, place signs and literature educating parents about children's vision. Discuss InfantSee with pregnant patients. Make pediatric care highly visible on your website and include pediatric intake forms for parents to fill out at home.

Finally, don't forget networking. Drs. Schuetz has had success working with the local schools. Sending a follow-up letter to the pediatrician after a child fails their vision screening is a good way to initiate a relationship. Visit potential referral sources and find out how you can be helpful to them. Leonard Press, OD, of Fair Lawn, NJ, agrees: "If you can establish a working relationship with at least one pediatric ophthalmologist in your area where there is mutual respect, that can be helpful."

One of the most powerful aspects of expanding a practice into pediatric care is the statement that your practice is different from others. As a specialist, you are dedicated to the most important thing in a parent's life: their children. This can be very profitable as well as rewarding. "There's no better feeling than watching a child's eyes light up when they put on their first pair of glasses or seeing the patient with convergence insufficiency become the most avid reader in his class," Dr. Lyons says. "Those moments make the daily hassles of working with kids well worth the extra time and effort." ■

Dr. Bonilla-Warford is in private practice in Tampa, Fla., specializing in vision therapy and orthokeratology. Dr. Knighton is in private practice in Tampa and Clearwater, Fla., specializing in pediatrics and patients with special needs.

1. Walline JJ, Jones LA, Rah MJ, et al; CLIP Study Group. Contact Lenses in Pediatrics (CLIP) Study: chair time and ocular health. Optom Vis Sci. 2007 Sep;84(9):896-902.
2. Walline JJ, Jones LA, Sinnott LT. Corneal reshaping and myopia progression. Br J Ophthalmol. 2009 Sep;93(9):1181-5.
3. Walline JJ, Lindsley K, Vedula SS, et al. Interventions to slow progression of myopia in children. Cochrane Database Syst Rev. 2011 Dec 7;(12):CD004916.
4. Walline JJ, Greiner KL, McVey ME, Jones-Jordan LA. Multifocal contact lens myopia control. Optom Vis Sci. 2013 Nov;90(11):1207-14.



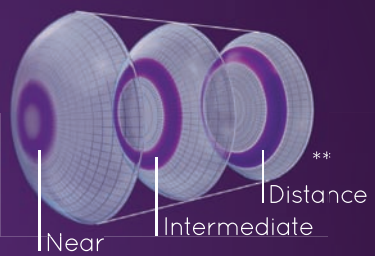
THIS IS WHY your patients will never lose sight of the big picture.

The **#1 multifocal contact lens design**, Precision Profile Design, is now available on today's market-leading monthly replacement and daily disposable contact lenses[1]—for clear binocular vision, near through far.

- **AIR OPTIX® AQUA Multifocal** contact lenses have a unique plasma surface technology for consistent comfort from day 1 to day 30.[2]
- **DAILIES® AquaComfort Plus® Multifocal** contact lenses deliver refreshing comfort with every blink and have blink-activated moisture technology.

Visit MYALCON.COM for additional information.

Precision Profile Design



PERFORMANCE DRIVEN BY SCIENCE™

Daily Disposable Lens

Monthly Replacement Lens

*AIR OPTIX® AQUA Multifocal (lotrafilcon B) contact lenses: Dk/t = 138 @ -3.00D. Other factors may impact eye health. **Image is for illustrative purposes and not an exact representation.

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

References: 1. Based on third-party industry report, 12 months ending March 2014, Alcon data on file. **2.** Eiden SB, Davis R, Bergenske P. Prospective study of lotrafilcon B lenses comparing 2 versus 4 weeks of wear for objective and subjective measures of health, comfort and vision. *Eye & Contact Lens.* 2013;39(4):290-294.

See product instructions for complete wear, care, and safety information.

Alcon
a Novartis company

 5/14 MIX14193JAD

Rx only

PEER • REVIEWED • RRA • ARTICLE

A Detailed Look at Optic Nerve Anomalies

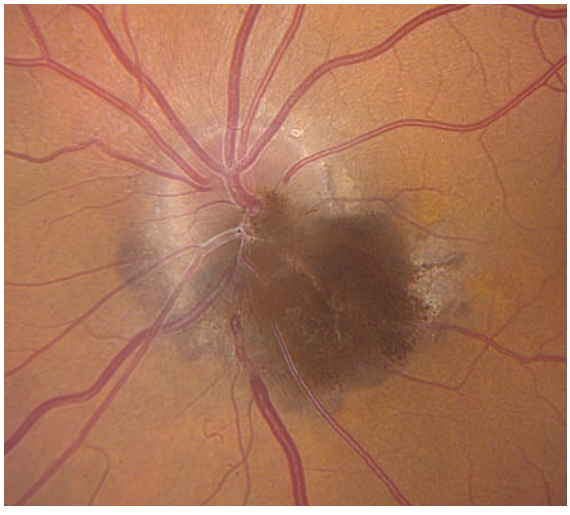
Here, we review diagnostic strategies for a wide variety of optic nerve irregularities that may be observed in clinical practice. **By Anu Laul, OD, and Marta Fabrykowski, OD**

Examining the optic nerve can be a daunting task. Its potential permutations are diverse, with individualized variations in color, size and even vascular supply.

A normal optic nerve head (ONH) usually is round or oval, mildly elevated and pink in color, with a centralized depression known as the cup. The horizontal diameter of a typical optic nerve is approximately 1.5mm.[1]

When optic nerve abnormalities are detected, it is essential to differentiate between anatomical and pathological causes. This is because certain irregularities may require additional testing and/or intervention.

Optic nerve anomalies can be categorized as *congenital* or *acquired*. Congenital anomalies can be further subdivided into *benign* or *pathologic*. Acquired abnormalities are assumed to be pathologic, and generally are described with respect to optic nerve's reaction to a given insult (i.e., cupping, swelling or atrophy).[1]



1. Melanocytoma with adjacent choroidal, retinal components and mild yellow exudation.

Benign Congenital Anomalies

• ***Melanocytomas*** are characteristically unilateral, very deeply pigmented black or dark brown lesions that obscure all or part of the optic disc (*figure 1*).[2] Their mean thickness is approximately 1.0mm; however, they may be noticeably elevated.[2]

Melanocytomas usually are located inferiorly on the optic nerve head, and up to 60% of cases involve either the retina or choroid.[2] From a demographic standpoint, approximately two-thirds of all presentations are documented in whites, with just one-third seen in blacks.[2] Although considered benign neoplasms, 1% to 2% of melanocytomas can transform into malignant melanoma.

• ***Tilted discs*** are characterized by an elevation of the superotemporal disc, posterior displacement of the inferonasal disc and situs inversus of the retinal vessels.[1] They present bilaterally in 80% of patients.[1]

One Plug
One Size

SnugPlug™

That's *all* you need!

One-size-fits-all punctum plug
eliminates sizing and **simplifies stocking**

Pre-stretched shape (on inserter)
avoids dilation and **facilitates insertion**

Expanded shape (once inserted)
assures snug fit and **virtually eliminates pop-out**

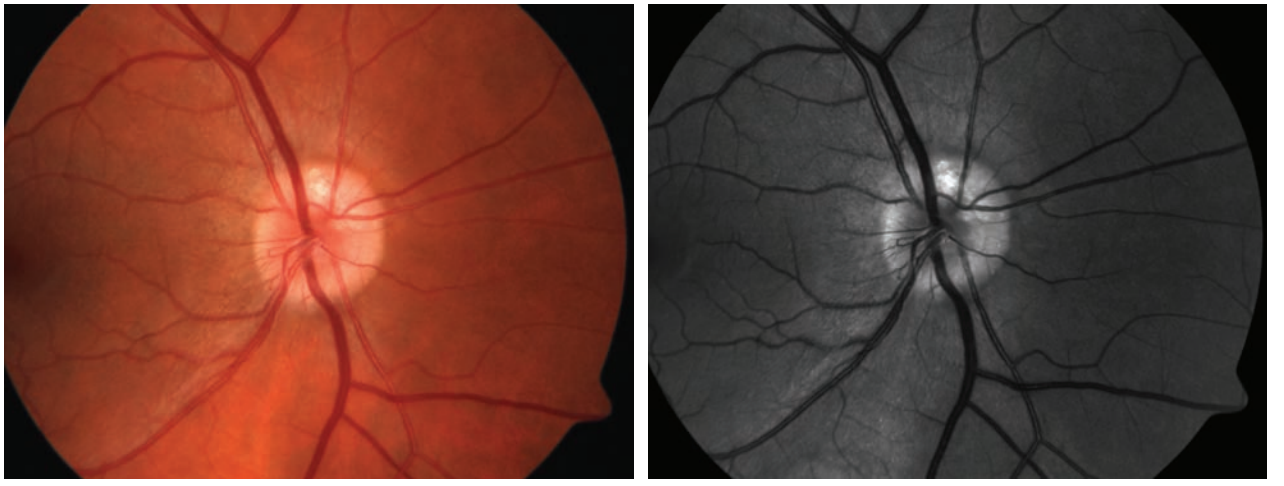
Soft collar prevents migration
and **provides patient comfort**

Ophthalmics
FCI

FCI-Ophthalmics.com
800.932.4202

Plug into FCI for Dry Eye Treatment and watch the SnugPlug insertion video.

Visit PlugintoFCI.com for promotional pricing



2, 3. Optic nerve head drusen located at approximately 12 o'clock, with a highlighted appearance on fundus autofluorescence (right).

• ***Optic disc colobomas*** appear as sharply defined, white, bowl-shaped, inferiorly decentered excavations. Rarely is the entire disc affected. They can occur either unilaterally or bilaterally, with equal frequency.[1]

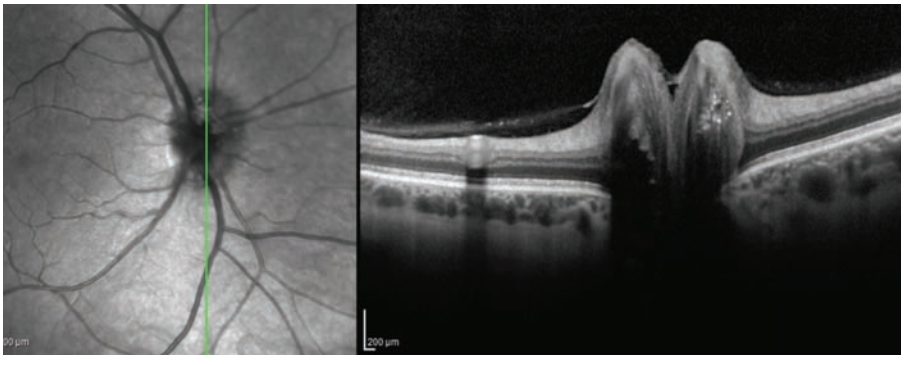
Often, there can be associated colobomas of the iris and/or ciliary body. Further, patients with optic disc colobomas commonly experience significant refractive error and anisometropia.[3]

It is important to note the potential association of optic disc colobomas with renal coloboma syndrome, which may cause devastating kidney problems. Because associated kidney diseases are potentially life threatening, individuals diagnosed with optic nerve colobomas should have a renal ultrasound.[1,3]

• ***Optic nerve head drusen*** are multilobed, globular concretions of calcium, amino and nucleic acids, mucopolysaccharides and sometimes iron.[4] Ophthalmoscopically, they appear as multiple, round to irregular, whitish-yellow dots within the surface of the nerve.[1] Anomalous blood vessel patterns frequently are visible. The nerve itself is characteristically small and appears crowded. Such drusen are not usually visible at an early age, are said to be "buried" and appear to enlarge throughout life as they move closer to the surface and become more visible.

Optic nerve head drusen are often bilateral and have no gender predilection. Adjunctive photographing techniques, such as fundus autofluorescence, may make the drusen appear more pronounced (*figures 2 and 3*). And, because optic nerve head drusen also may exhibit considerable elevation, they are best visualized with optical coherence tomography (*figure 4*).

It is important to differentiate optic nerve head drusen from acquired bilateral disc edema or papilledema. Key clinical findings here include defined refractile aggregates, the presence of spontaneous venous pulsation and the absence of Paton's lines.

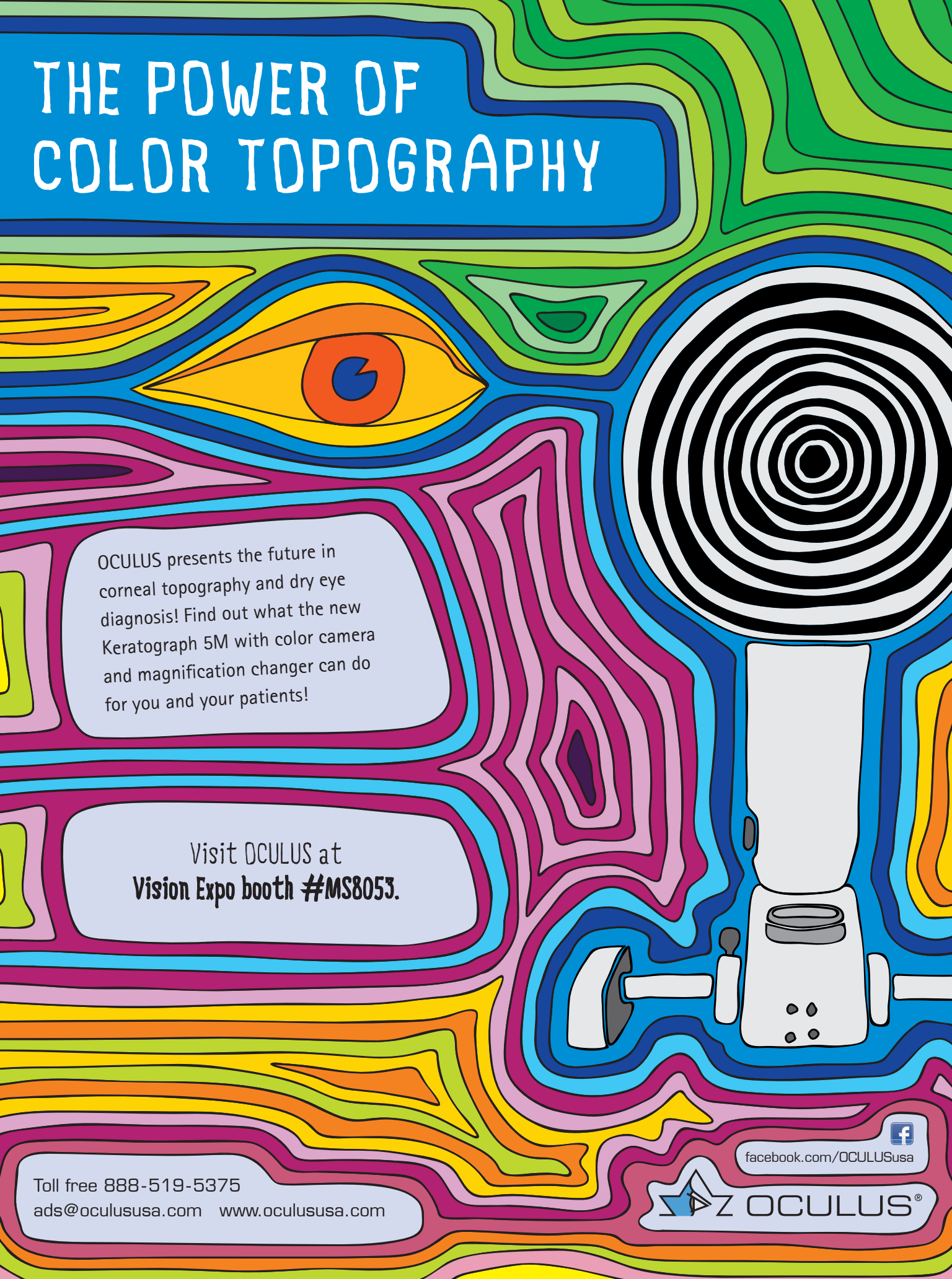


4. Multiple focal, round, hyper-reflective masses with significant elevation.

Pathologic Congenital Anomalies

• ***Optic nerve pits*** are congenital abnormalities of the optic nerve due to incomplete closure of the fetal fissure. They are believed to occur during the first trimester of gestation, are usually less than one-half disc diameter in size and are more commonly located temporally.[5] There are no known systemic or gender associations; however, a higher incidence of optic nerve

THE POWER OF COLOR TOPOGRAPHY



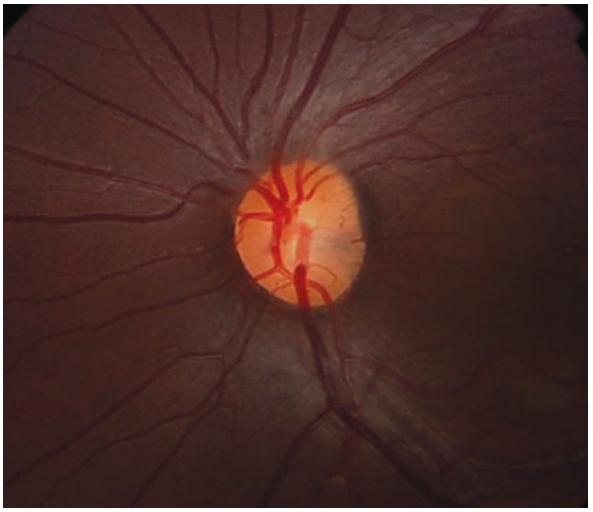
OCULUS presents the future in corneal topography and dry eye diagnosis! Find out what the new Keratograph 5M with color camera and magnification changer can do for you and your patients!

Visit OCULUS at
Vision Expo booth #MS8053.

facebook.com/OCULUSusa

OCULUS®

Toll free 888-519-5375
ads@oculususa.com www.oculususa.com



5. Optic nerve pit. Note the subtle gray area located nasally to the optic nerve head's center.

pits is observed in those with basal encephalocele.[5] Optic nerve pits are relatively rare, with an overall incidence of one in 11,000.

Circumpapillary chorioretinal atrophy with associated retinal pigment epithelium changes are commonly seen in those with optic nerve pits—especially if the pit is located near the disc margin.[5] This can lead to visual field defects, particularly if the anomalies displace nerve fibers.

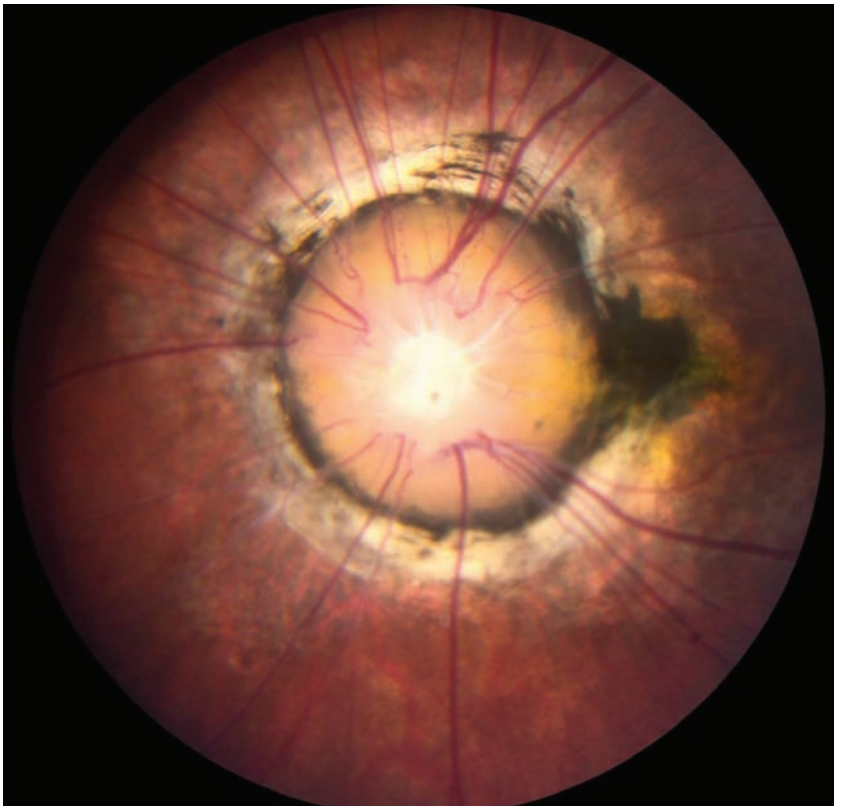
Optic pits are best seen clinically on dilated fundus evaluation and OCT imaging. They appear as grey, yellow or black excavations in the optic disc and are unilateral 95% of the time (*figure 5*).[5] In 85% of cases, the optic nerve with the pit is larger than the fellow optic nerve.[5]

Optic nerve pits rarely affect visual acuity, unless the patient develops a serous macular detachment.[5] The origin of the serous fluid is not completely understood, however.[6] The most widely accepted theories are liquefied vitreous material gaining access to the subretinal space via the optic pit, or cerebrospinal fluid from the optic nerve leaking through the optic pit into the subretinal space.[5] Histological studies involving dogs showed a connection between the vitreous and subretinal space; however, that finding has yet to be shown in human histopathological studies.

• ***Morning glory syndrome*** is a rare congenital dysplasia of the optic nerve.[3] It almost always presents unilaterally and is more common in females.[3] The condition was first described in 1970, and was named for its resemblance to the morning glory flower (*figure 6*).[3] Upon physical examination, affected individuals frequently exhibit a large, excavated disc with glial tissue that is centrally surrounded by peripapillary pigment changes (which may include the macula).[3] There is debate on the pathophysiology of this disc abnormality; however, many authorities now believe that it is a dysgenesis of mesodermal and ectoderm tissues.[3]

Like optic nerve pits, morning glory syndrome may be associated with basal encephalocele—so, MRI often is indicated.

Unfortunately, visual acuity tends to range from 20/200 to finger counting in patients with morning glory syndrome; however, entering acuity can range anywhere from 20/20 to no light



6. Morning glory disc. Note the dark pigment ring around the nerve, as well as centralized elevation where the glial tissue remains.

The **world's first** and only silicone hydrogel, daily disposable family.

Affordable Innovation

FREE TRIAL

Why not try it for yourself?

Go to: **sauflonusa.com/freetrial**
to try clariti®1day today*.

*Offer available only to Eye Care Practitioners.

SINGLE VISION | ASTIGMATISM | PRESBYOPIA

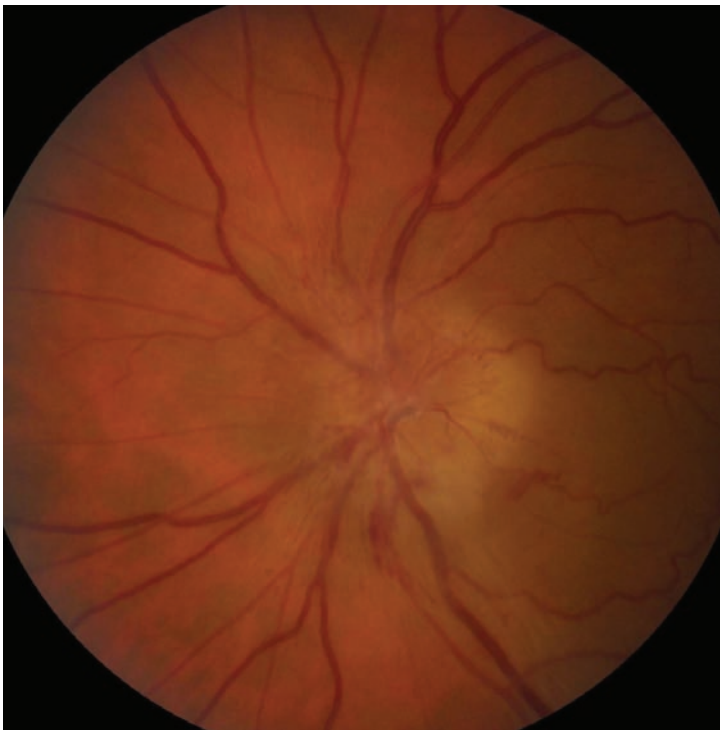
SAUFLON
Exclusively for Eye Care Professionals

Exclusively for Eye Care Professionals

For more information contact Sauflon at:
T: 800-682-3240 E: info@sauflon.com www.sauflonusa.com

perception (NLP).[3] While secondary vision loss is not progressive, 30% of morning glory patients develop serous retinal detachments secondary to small breaks in the peripapillary region.[3]

• ***Optic nerve hypoplasia*** is usually associated with an uncharacteristically small optic nerve head in which the number of axons is significantly reduced.[3] It often occurs bilaterally (in 65% to 75% of cases), and is the third leading cause of blindness of children in the US (after cerebral damage during birth and retinopathy of prematurity).[3]



7. This patient presented with anterior ischemic optic neuropathy (AION). Note the hyperemic swelling of the optic nerve associated with many flame-shaped, peripapillary hemorrhages.

Optic nerve hypoplasia is characterized by a regression of axons due to apoptotic mechanisms catalyzed by the incomplete formation of other axons.[3] Although causation can be spontaneous (especially in unilateral cases), some reports have indicated that premature birth, young maternal age, maternal type 1 diabetes or gestational diabetes, fetal infection, fetal alcohol syndrome, illicit drug abuse by the mother or the use of certain medications during pregnancy can precipitate optic nerve hypoplasia.[3]

Clinically, the most recognizable sign of optic nerve head hypoplasia is the "double ring sign," in which a peripapillary ring sometimes surrounds the small nerve.[3] Further, if the distance from the disc to macula is equal to or greater than 3DD, optic nerve hypoplasia is likely.[3] Finally, most patients with optic nerve hypoplasia exhibit vessel tortuosity in the affected eye.[3]

Depending on the extent of axonal loss, visual acuity can be as good as 20/20 or as poor as NLP.[3] Visual field defects are common, and clinicians often detect an afferent pupillary defect in patients with asymmetrical presentations.[3] Visual evoked potential (VEP) testing can be helpful when the diagnosis is unclear.[3] Patients with optic nerve hypoplasia would show a decreased amplitude in VEP, but a normal electroretinogram.[3]

• ***Leber's hereditary optic neuropathy*** was first described in 1871 by Theodore Leber, when he documented a terrifying, non-Mendelian pattern of progressive vision loss in members of four families.[7] Subsequent research indicated that the condition was caused by mutations to mitochondrial DNA.[7] The prevalence of Leber's hereditary optic neuropathy is as high as one in 25,000 in some populations, and it is by far the most common mitochondrial genetic disease.[7] The condition commonly affects males in the second to forth decades of life.[7]

Leber's presents in two phases: acute and chronic.[7] In the acute phase, circumpapillary telangiectasia, nerve fiber layer swelling and vessel tortuosity are often noted; however, in 20% of cases, the optic nerve looks completely unremarkable.[7]

In the chronic phase, the nerve fiber layer degenerates, which causes optic atrophy.[7] The acute phase typically persists for four to eight weeks after initial presentation. Then, after approximately six months, patients transition into the chronic phase.[7]

Patients with Leber's hereditary optic neuropathy characteristically experience significant vision loss during the acute phase, frequently declining to 20/200 or worse before transitioning into the chronic disease phase.[7]

Acquired Pathologic Anomalies

• ***Ischemic optic neuropathy (ION)*** is the result of decreased blood flow to the optic nerve's ganglion cells.[8] ION may be either arteritic (AION) or non-arteritic (NAION).[8,9] AION is caused by giant cell arteritis (GCA), while

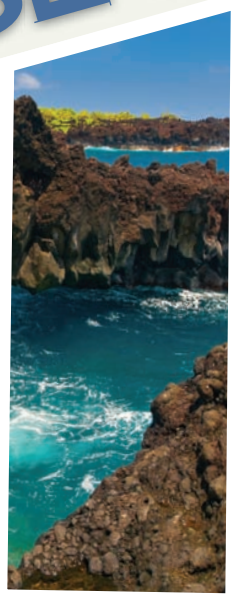
REVIEW OF OPTOMETRY
EDUCATIONAL MEETINGS OF CLINICAL EXCELLENCE

New Technologies and Treatments
NT&T
IN VISION CARE
2015

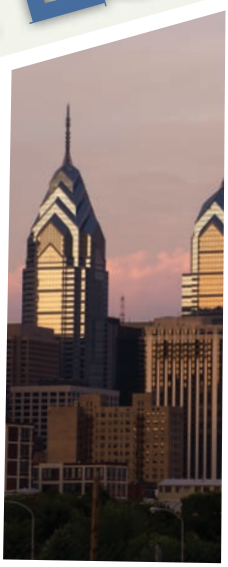
SAVE THESE DATES

SAN DIEGO, CA
APRIL 10-12

CHARLESTON, SC
JUNE 12-14



MAUI, HI
JULY 23-26



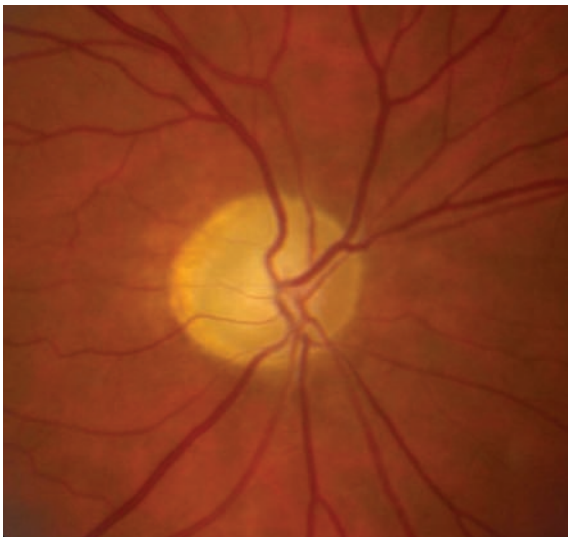
PHILADELPHIA, PA
NOVEMBER 6-8

For more information visit www.revoptom.com

REVIEW OF OPTOMETRY

cope®
*Approval pending

Up to
19 CE
Credits
(COPE approval pending)



8. Optic nerve appearance following AION, which features disc pallor located more temporally than nasally.

NAION may result from a variety of underlying vascular disorders.

AION is characterized by a loss of blood flow to the most anterior portion of the optic nerve, which is supplied by the posterior ciliary artery.[9] Visual acuity varies in patients with AION; however, all forms of ischemic optic neuropathy can potentially cause visual field defects, typically altitudinal (*figure 7*).[8]

NAION is the most common form of ischemic optic neuropathy.[9] Risk factors include hypertension, diabetes, atherosclerosis, sleep apnea, nocturnal hypotension, small discs and certain medications.[9] NAION patients often present with painless, unilateral vision loss that has persisted for a few hours to several days.[8]

Upon examination, NAION patients show evidence of an afferent pupillary defect, sectoral disc edema and corresponding visual field loss.[6] Over the course of a few weeks to months, the disc swelling subsides, resulting in sectoral nerve head pallor (*figure 8*).[6]

The clinical presentation of anterior arteritic ischemic optic neuropathy (AAION), is similar to that of AION; however, patients typically experience weight loss, headache, scalp tenderness, fever and malaise.[6,8] These patients tend to be older and, if not treated within five to 10 days, will also develop vision loss in the contralateral eye up to 50% of the time.

The collateral branches of the long posterior and central arteries supply the posterior portion of the optic nerve. Thus, poor or compromised blood flow to this area results in a posterior ischemic optic neuropathy (PION).[9] Like anterior ischemic optic neuropathy, PION can be arteritic or non-arteritic. Also, PION may be caused by significant blood loss, hypotension or general anesthesia during surgery.[9] In these cases, resultant visual field loss tends to be both pronounced and bilateral, and can even result in complete blindness.[9]

• ***Papilledema*** is defined as disc swelling secondary to increased intracranial pressure, and most often presents bilaterally.[10] In the US, the most common cause of papilledema is idiopathic intracranial hypertension (also called pseudotumor cerebri).[11] Most prevalent in obese women of childbearing age, chronic papilledema can lead to progressive optic atrophy.[11] Although the exact etiology is unknown, overweight or obese individuals are at increased risk for papilledema.[11]

The clinical presentation includes bilateral disc swelling with obscuration of blood vessels, blurry disc margins, vessel tortuosity, hemorrhages and Paton's lines. Patients often present with symptoms of blurred vision, transient vision loss, dizziness, tinnitus, headaches and visual field compromise.[11] Visual prognosis tends to be quite good, with less than 25% of patients experiencing significant permanent vision loss.[11]

• ***Optic neuritis (ON)*** is inflammation of the optic nerve secondary to demyelinating diseases, such as multiple sclerosis (MS) and neuromyelitis optica.[12] It tends to present in the second to fifth decades of life and has the highest incidence in white females.[12] Although MS is the most common cause, ON also may be due to orbital or systemic infection (e.g., HIV or syphilis).[5]

Clinical signs and symptoms include acute unilateral vision loss that persists for hours or days before improving, pain upon eye movement, afferent pupillary defect and visual field defects.[5,13] Because the optic nerve inflammation is retrobulbar in most cases, funduscopic examination often appears unremarkable. However, the optic nerve may start to show signs of pallor within four to six weeks after an acute event.[5,6]

Visual prognosis tends to be quite good after the first occurrence, as 75% of patients recover to 20/40 or better.[6] That said, with each subsequent occurrence of ON, further visual decline is likely.

• ***Glaucoma*** is a progressive neuropathy of the optic nerve secondary to apoptosis, which eventually causes visual field defects. While the condition often is acquired, glaucoma also may be congenital or primary.[6] Historically, glaucoma was thought to be a disease of increased eye pressure, and

2CE Credits
cope
*Cope Approval Pending

Save the Date

JOIN THE OCULAR NUTRITION SPECIAL INTEREST GROUP OF THE AMERICAN ACADEMY OF OPTOMETRY FOR A CE DINNER IN DENVER

Thursday, November 13th

Current Nutrition Controversies affecting ECP recommendations to protect against ocular disease

FACULTY

Dennis Ruskin, OD, FAAO (Chair)

Paul Chous, MA, OD, FAAO

Kim Reed, OD, FAAO

Stuart Richer, OD, PhD, FAAO

Register through the American Academy of Optometry meeting registration site below!

LOCATION

Hyatt Regency
Meeting Room: Centennial Ballroom F-H
650 15th Street
Denver, CO 80202

TIME

7:15pm – 7:30pm Registration Check-in

7:30pm – 9:30pm **Dinner and CE Seminar**

CE TOPICS

- Review the legacy of information from AREDS1 and AREDS2 and the unique genetic relationship that genotype has upon AMD risk and potential supplement recommendations.
- Discuss the role that Omega 3 has upon ocular and systemic health.
- Examine the importance of Vitamin D for ocular and systemic health.
- Demystify the controversy of the three carotenoids Lutein, Zeaxanthin, and Mesozeaxanthin with recommendation for risk to AMD.

Jointly sponsored by

REVIEW OF OPTOMETRY

AMERICAN ACADEMY of OPTOMETRY

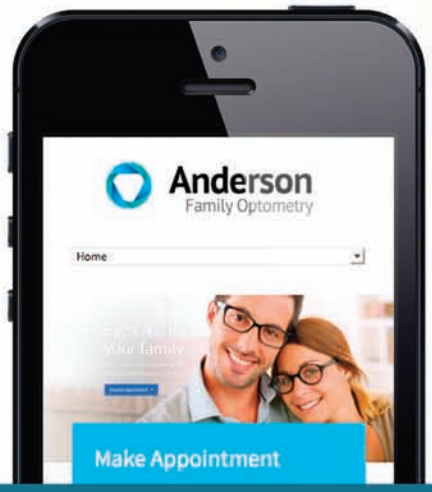
Supported by an educational grant from BAUSCH + LOMB

You must be registered for Academy 2014 Denver and for this dinner program to attend this CE Program.

To register for Academy 2014 Denver visit http://www.aaopt.org/regsite/2014Registration.

You Help Patients See. We Help New Patients See You.

- A strong web presence is essential for new patients to find your practice.



Get the solution you need to create a powerful web presence.

- Appointment requests make it easy for new patients to contact you.
- Online forms streamline first time visits.
- The Frame Selector makes buying frames efficient.
- An interactive Google map gets patients to your door.
- An online store increases revenue and convenience.
- Blogs, social media, and video improve patient retention and referrals.

imatrix™
Your Internet Marketing Partner

Web solutions increase **efficiency and revenue.**
Get the solution that's right for you.

Call **855.976.3008** or visit
optometry.imatrix.com.

although increased intraocular pressure is a significant risk factor, it is no longer considered a defining characteristic.

Glaucomatous optic neuropathy differs from other diseases of the optic nerve, because it does not cause swelling or pallor.[13] In glaucoma, localized areas of thinning (notches) most often occur in the inferior and superior quadrants of the optic nerve.[5,6,14] Over time, we observe vertical enlargement of the cup-to-disc ratio, with corresponding ganglion cell loss.[5,6] In some cases, disc hemorrhages precede ganglion cell loss.[6] Initially, vision tends to be quite good; however, as the disease progresses, peripheral vision loss can eventually encompass the central field.[6]

Optic nerve disorders often have characteristic features, but in some cases may present similarly to other disorders. It is imperative to differentiate benign from pathologic conditions, as different cases may require extensive follow-up or pharmaceutical/surgical intervention. Regardless of the severity or associated risk, careful examination and documentation is necessary to properly identify and manage optic nerve anomalies. ■

Dr. Laul is an instructor of ophthalmology at the Wilmer Eye Institute, Johns Hopkins School of Medicine, in Baltimore.

Dr. Fabrykowski is on staff at the Manhattan Eye, Ear and Throat Hospital Faculty Ophthalmology Practice, operated by Lenox Hill Hospital, in New York.

1. Golnik KC, Payesse EA, Torchia MM. Congenital Anomalies and Acquired Abnormalities of the Optic Nerve. Philadelphia: Wolters Kluwer; 2014.
2. Shields JA, Demirci H, Mashayekhi A, et al. Melanocytoma of the optic disc: a review. Surv Ophthalmol. 2006 Mar-Apr;51(2):93-104.
3. Dutton GN. Congenital disorders of the optic nerve: excavations and hypoplasia. Eye (Lond). 2004 Nov;18(11):1038-48.
4. Grippo TM, Rogers SW, Tsai JC, Lewis RA. Optic disc drusen: practical implications and management. Glaucoma Today. Available at: http://bmctoday.net/glaucomatoday/2012/02/article.asp?f=optic-disc-drusen. Accessed August 13, 2014.
5. Yanoff M, Duker J. Ophthalmology, 2nd ed. St. Louis: Mosby; 2004.
6. Kanski J. Clinical Ophthalmology: A Systematic Approach, 5th ed. London: Butterworth-Heinemann; 2003.
7. Man PY, Turnbull DM, Chinnery PF. Leber hereditary optic neuropathy. J Med Genet. 2002 Mar;39(3):162-9.
8. Athappilly G, Pelak VS, Mandava N, Bennett JL. Ischemic optic neuropathy. Neurol Res. 2008 Oct;30(8):794-800.
9. Hayreh S. Management of ischemic optic neuropathies. Indian J Ophthalmol. 2011 Mar-Apr;59(2):123-36.
10. Goosman M. Medscape. Papilledema. Available at: http://emedicine.medscape.com/article/1217204-overview. Accessed August 13, 2014.
11. Gans M. Medscape: Idiopathic intracranial hypertension. Available at: http:emedicine.medscape.com/article/1214410-overview. Accessed August 15, 2014.
12. Ergene E. Medscape: Adult optic neuritis. Available at: http://emedicine.medscape.com/article/1217083-overview. Accessed August 15, 2014.
13. Goodwin D. The differential diagnosis of an optic nerve disorder. Rev Optom. 2010 Apr;147(4):78-87.
14. Susanna R, Weinreb R. Answers in Glaucoma, 1st ed. Rio de Janerio, Brazil: Cultura Medica; 2005.

15th Annual Dry Eye Report

How to Build a Dry Eye Center of Excellence

From technology to education to the latest clinical research, experts weigh in on how to create a practice specialty that's patient-focused and can boost your bottom line.

By Jane Cole, Contributing Editor

Ohio optometrist Mile Brujic recalls the patient who made him change the way he practiced. In 2006, a female patient visited Dr. Brujic's office for the first time, complaining she couldn't see up close in the -1.00D single-vision glasses she was prescribed by another doctor. Dr. Brujic did a refraction and found she needed an add.

A relatively simple exam, he thought as she went out the door, until a month later when the woman returned and informed Dr. Brujic she couldn't see out of her new prescription either. As Dr. Brujic checked her vision a second time, he noticed the patient continued to blink as she tried to focus. "I could actually see her try to sweep a fresh set of tears over her eyes," he recalls.

It was then Dr. Brujic realized he missed the initial diagnosis. This patient, who had already gone to several doctors but was continuing to be misdiagnosed, had dry eye. He resolved to never again give a patient cause to question his clinical decisions, particularly when it involved paying out of pocket for new corrective lenses. "I made it a point then to become much more aggressive about understanding dry eye and to find the tools to help me diagnose these patients and manage them better."

As the prevalence of dry eye continues to rise and clinical research sheds more light on the disease, Dr. Brujic and many other practitioners have invested the time and resources to become dry eye experts and build that distinction into their practices. In so doing, they offer a haven to patients who've gone from doctor to doctor either incorrectly diagnosed or prescribed ineffective treatments: the so-called dry eye center of excellence.

With a proliferation of such centers opening across the country, we look at the philosophy of these centers, technology needed to serve these patients, and what such dry eye centers can ultimately do for patients and the practice.

The Practice Behind the Name

The term "dry eye center of excellence" is a bit misused today, says Chuck Aldridge, OD, of Burnsville, NC. "It was originally established by one of the pharmacy companies to assist doctors in setting up dry eye centers within

their practice," he says. They had a full set of tools and guidelines, and a practice could only use that designation if certain minimum standards were met. The program became a casualty of increased pharma regulations and was abandoned. Currently, there are no real standards for use of the phrase, he adds.

As such, any practice can call itself a dry eye center of excellence. What truly sets the practice apart, however, is not the name but rather what happens inside the office, says Phoenix optometrist Art Epstein, who recently opened a dry-eye focused practice called the Dry Eye Center of Arizona. "To me, a center of excellence is a practice that devotes itself to dealing with patient problems in a specific area and serves as the last stop, the final answer, that solves the problems that in many cases have gone on for quite some time," Dr. Epstein says.

For Dr. Brujic, a center of excellence is about a shift in philosophy, where a practitioner takes extra steps and does everything possible, starting with a thorough examination from the front to the back of the eye. "The front of the eye isn't just the cornea," Dr. Brujic says. "It's the tear film and how it supports the surface of the eye."

When a dry eye center of excellence first opens its doors, a few interesting things are going to happen, says Paul Karpecki, OD, who runs one of the largest dry eye centers in the country, at Koffler Vision Group, in Lexington, Ky.

"One, you're going to get a good amount of new dry eye patients at your center, but you'll also see a great deal of patients who have severe diseases—including some that aren't dry eye per se and have confused doctors, including myself, for many years," Dr. Karpecki says. *Demodex*, skin cancers of the eyelids, Sjögren's syndrome, convergence insufficiency, conjunctivochalasis and Salzmann's nodular degeneration are a few examples, he says.

"Working at a dry eye center of excellence really tests *all* your skills as an optometrist, not just your ocular surface expertise, which I think is really exciting and rewarding," Dr. Karpecki says.

Benefit to Patients and Bottom Lines

Because dry eye is one of the most prevalent conditions an optometrist will encounter, incorporating a dry eye specialization is very important for those practitioners who want to develop a medically-oriented practice, says Jack Schaeffer, OD, of Schaeffer Eye Center in Birmingham, Ala.

And the need is obviously there. Dry eye affects an estimated 14.4% of the US population over age 40 as well as 50% of all contact lens wearers.[1,2] Those numbers will only continue to increase. "If you look at it from simple demographics, the population is aging," Dr. Epstein says. "Since we know that dry eye is prevalent in an aging population, it's unavoidable that more of your patients will have ocular surface disease."

Another driver of patient volume affects a younger demographic:

Comanagement: Optometry Takes the Lead

Dry eye center experts say they are seeing a new trend: instead of referring a patient to a corneal specialist, the majority of patients are being referred in to them—and not just by fellow ODs, but by ophthalmologists as well.

"We have all the tools now to really manage these patients," Dr. Brujic says. "The days of comanaging patients for ocular surface conditions are dwindling. What ophthalmology is really taking on is tertiary surgical care, and we are really owning everything else."

Dr. Epstein agrees, saying that he "can't remember the last time we referred a patient for ocular surface disease," but now receives a rapidly increasing number of referrals from colleagues. "If you asked me this question 10 years ago, I'd say this would not be the case, but at this point, optometry has reached a level where we are managing many more complex cases because of advances in technology and growing confidence in our abilities. Optometrists are exquisitely patient-centric, and doing what's best for our patients gives us a decided advantage."

Dr. Karpecki estimates that 80% of his dry eye patients are referrals and he comanages them with a wide array of other doctors. "I comanage with optometrists, ophthalmologists, rheumatologists, endocrinologists, you name it," he says. A large, cataract-oriented practice in his area sends him four or five new referrals a week. "A dry eye center can create a wonderful secondary comanagement tier involving almost every kind of specialty."

Dr. Schaeffer considers comanagement an integral part of optometry's future, with optometrists referring back and forth to each other. "If you are a dry eye practice that doesn't fit scleral contact lenses, find a practitioner that does," he advises. "If you don't want to buy a LipiFlow machine, find a practitioner that has one. If you don't want to have a dry eye practice, find a practitioner who is good at dry eye and you can take care of all the other patient's needs. Working together by referring patients to each other is a sign of a higher level of optometric care."

"Dry eye disease is something optometry should own and manage start to finish," Dr. Aldridge says. "For optometrists who don't want to work with the more difficult cases, this is an excellent opportunity to refer to their colleagues."

Basic Dry Eye Questionnaire

CHUCK ALDRIDGE, O.D., M.B.A.
Fellow American Academy of Optometry
Email: ccaldridge@yahoo.com • P.O. Box 218 • 419 • East Main Street • Burnsville, NC 28714 • (828) 682-2104

Office Hours By Appointment

Do you ever experience:

Gritty or sandy sensation?	Never	Slight	Moderate	Severe
Pain or soreness?	Never	Slight	Moderate	Severe
Fluctuating vision?	Never	Slight	Moderate	Severe
Occasional tearing?	Never	Slight	Moderate	Severe
Blurred vision while reading?	Never	Slight	Moderate	Severe
Discomfort in windy conditions?	Never	Slight	Moderate	Severe
Discomfort in air conditioned areas?	Never	Slight	Moderate	Severe
Itching?	Never	Slight	Moderate	Severe

mobile device use. Smartphones and tablets, ubiquitous among teens and 20-somethings with otherwise healthy eyes, "have an impact on blink rate," Dr. Epstein says. Fewer blinks per minute limit the ability of meibomian glands to release oil and the lids to properly disperse it, he says, creating a cascade that results in tear film instability and, ultimately, dry eye symptoms.

This growth in patient ranks has been matched by greater attention from both researchers and clinicians. "Our understanding of dry eye and the ocular surface has increased dramatically," Dr. Epstein says, particularly in the pathogenesis of dry eye and tear dysfunction, allowing industry to develop more targeted therapeutics.

Clinical protocols have matured as well. "Some things I do today I didn't do routinely just three or four years ago, like meibomian gland expression," Dr. Karpecki says. "There's so much more we've learned, and it's starting to make a difference." There is also a sense of great accomplishment "because of the gratitude patients have" for your efforts, he says.

Of course, done effectively, a center of excellence can also generate significant revenue from exams, procedures and sales of materials such as warm compress masks, artificial tears and ocular nutritional supplements, Dr. Karpecki adds.

Increased scrutiny and regulation from insurers and the trend of declining reimbursements pose challenges to many optometrists who need to find ways to ensure patients are properly cared for and their practices can also be compensated appropriately. says Dr. Epstein. A specialization in dry eye, including some services that aren't covered by insurance, can fill that void, he suggests.

Dry Eye Goes High Tech

Recently, new technology has offered the potential to diagnose dry eye with greater precision, and experts familiar with such tools say they have changed the way they practice and offered greater opportunities for dry eye centers.

However, even though these new technologies may help to set a practice apart, tried-and-true diagnostic tools remain an integral part of the dry eye clinic and are generally already a mainstay—though perhaps underused—in a typical practice, Dr. Brujic says. Fluorescein dye with a cobalt-blue slit lamp assessment, lissamine green and phenol red thread are just a few of the "old school" techniques that still serve ODs well in dry eye care, he says. "This is all available, and every single optometrist has this in their practice, but only a fraction may be using them regularly"

The experts agree: None of the new technologies, no matter how intuitive or sensitive, replace a good exam, starting with a patient discussion and a survey or questionnaire (*see "Basic Dry Eye Questionnaire," above*). "If you listen carefully to patients, they will often tell you what their diagnosis is," Dr. Epstein says. "For example, the patient whose dry eye or OSD is worsened by exposure at night will often say, 'I keep drops by the side of the bed.' One patient said if he gets up in the night, he has to feel his way to the bathroom with his eyes closed and then splash water on his face before he can open his eyes," he elaborates. "So, the patient discussion is extremely important, just as is the slit-lamp exam and tear and ocular surface assessment."

If a practitioner truly wants to create a dry eye center of excellence, there is an amazing amount of advanced technology available today, Dr. Schaeffer says. "We can now qualify dry eye not just by objective and subjective signs observed through the biomicroscope, but we can quantify the

grading levels with the help of high tech equipment. So, now we can grade the severity of dry eye when we begin treatment, and we can more closely measure the results of our treatment."

And, this new technology can help make a differential diagnosis. "These new devices on the market now offer the clinician a means to validate what they feel is the correct diagnosis. 'I think this is dry eye disease, but could it be allergy?'" Dr. Aldridge says. "The new tests help confirm the diagnosis. And from a patient's perspective, they can see and understand, for example, a picture of the meibomian glands or an osmolarity number, and for doctors, new technology can help them look for changes during their treatment."

Here's how some of the latest innovations are used in practice to refine dry eye care:

• ***TearLab Osmolarity System*** (TearLab Corporation): Tear osmolarity was recognized as an indicator of dry eye in the Dry Eye Workshop of 2007 and other studies.[3-5] This device measures osmolarity of tear samples and categorizes the severity of the results. Clinicians say the device can confirm the findings of a slit-lamp exam, provide a baseline of disease severity and track the progress of treatment. "I've used TearLab since the start, and it's been an awesome test," Dr. Aldridge says. "Patients understand numbers, such as their blood pressure, blood sugar levels and cholesterol." Sharing their tear osmolarity with them helps them to put it in perspective and understand its significance, he says.

One clinical pearl he offers is the need for doctors to understand the inherent fluctuation in the findings, particularly for abnormally high results. "This is the nature of DED," Dr. Aldridge says. "The severity goes up and down through the day." As the condition is treated and brought under control, the numbers begin to fall and the degree of fluctuation becomes less and less, he adds.

• ***RPS InflammaDry Detector*** (Rapid Pathogen Screening): Increased matrix metalloproteinase (MMP)-9 activity has been observed in the tear fluid of dry eye patients. As such, MMP-9, a cytokine produced by epithelial cells experiencing inflammation, appears to be a reliable marker for the presence of early ocular surface

A New Dry Eye Resource Just One Click Away

This month, doctors will be able to add a new dry eye tool to their practice by simply logging onto their computer.

The web-based service Ophthalmic Resources (www.ophthalmicresources.com), which will officially be launched this fall for doctor enrollment, is an online database that will feature educational information on dry eye and ocular surface disease, in addition to providing an order and delivery service for dry eye products such as artificial tears, warm compresses, lid cleansers and ocular nutrition supplements. It essentially allows the physician to profit from the sale of OTC products based on ocular surface disease protocols, without inventory hassles or patients being confused by numerous options at a pharmacy, says Paul Karpecki, OD, the service's clinical advisor and founder.

Doctors can custom order the items from a distributor and the products are then shipped to a patient's home or business, which can reduce in-house stock and ensure a patient gets the exact product their doctor recommends. Stay tuned for further details.

disease and dry eye.[6,7]

The InflammaDry test detects the presence of MMP-9 in tear fluid, which can help confirm a diagnosis. Normal levels of MMP-9 in human tears range from 3ng/ml to 40ng/ml. InflammaDry identifies a level above 40ml, a critical threshold for dry eye patients, says Dr. Brujic, who incorporated the technology in his practice earlier this year. He says the test has given him better insight into what's happening on the ocular surface, and the results have provided guidance on potential treatment options.

Meibomian gland dysfunction is now considered the leading cause of dry eye.[8] MGD can result in inadequate release of oil needed to form the lipid layer of the tear film.[8] Obstructive MGD is the most common form of MGD that results in dry eye.[9] With this in mind, several products analyze the state of the meibomian gland and provide treatment to restore function.

• ***Oculas Keratograph 5M*** (Oculus): This corneal topographer adds a high-resolution infrared camera to allow for meibography, and includes a suite of dry eye tests, including non-invasive tear film break-up time, ocular redness, tear meniscus height measurement and the ability to evaluate the lipid layer of the tear film. "New technology such as this has allowed me to see things I never could before," Dr. Epstein says. "Even more importantly, it allows us to share the findings with our patients so they are seeing exactly what we are seeing, and we can document it." Additionally, the device can objectively measure tear break-up time down to a fraction of a second, he adds.

• ***LipiView/LipiFlow:*** (TearScience): LipiView offers an objective measure of meibomian gland function. "We know that almost 90% of patients who have dry eye have primary or contributory meibomian gland dysfunction," Dr. Epstein says. Being able to measure the thickness of the lipid layer and also the completeness of the blink is extremely important, he says. "Every time we blink, we express the glands." He says the device measures lipid layer thickness "down to the nanometer" and is useful for documentation of baseline status and response to treatment. Patient education, too—"I show this to patients and they love getting a number, and it allows us to track their progress. It helps them understand the value of LipiFlow, a treatment for MGD that has been nothing short of

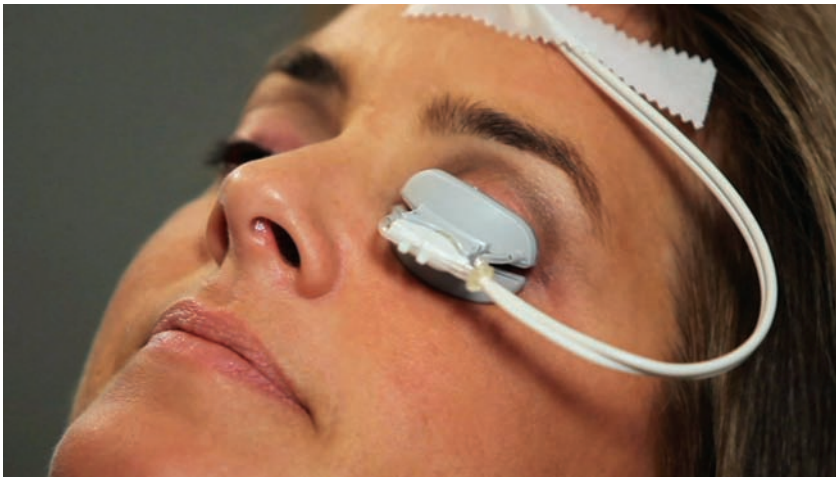
Putting Dry Eye Education Into Practice at SCO

Dry Eye Centers of Excellence aren't exclusive to private practice. The Southern College of Optometry recently launched TearWell Advanced Dry Eye Treatment Center, which opened its doors on July 1, 2014.

"TearWell provides our students a unique view into a premium, or concierge, practice," says SCO associate professor Whitney Hauser, OD, TearWell's clinical development consultant, in which the patient's needs and satisfaction are the focus from initial greeting through the conclusion of the examination. "Our dry eye patients have often been seen by many practitioners and found little relief from their symptoms. These patients demand and deserve an individualized approach that focuses exclusively on their complicated and chronic condition."

Dr. Alan Kabat, SCO professor and clinical care consultant states, "We pride ourselves on offering the newest and most cutting edge technology in patient care. Ours is a practice dedicated to ocular surface wellness, identifying the primary etiologies of our patients' symptoms and outlining a progressive strategy to correct those issues." Among the center's core battery of testing: LipiView interferometry, tear osmolarity, infrared meibography, InflammaDry, Sjö (for those with suspected Sjögren's syndrome), microscopic evaluation for *Demodex* and in-office testing for regionally specific ocular allergies. TearWell is also the first practice in the greater Memphis area to offer such innovative therapies as BlephEx and LipiFlow.

SCO's Alan Kabat, OD, and Whitney Hauser, OD.



The LipiFlow (TearScience Inc.) device used during a 12-minute, in-office dry eye treatment. It simultaneously applies heat and pulsatile pressure to the eyelids.

disruptive," Dr. Epstein says. "LipiFlow has been life changing for many of my patients who have suffered for years."

The LipiFlow device allows treatment of obstructed meibomian glands by heating the eyelid surfaces and applying a pulsating force to express the glands. "The only way to treat meibomian gland disease is to get the glands working again," Dr. Schaeffer says. "The glands need to be expressed, as they have been blocked and are not functioning." Manual expression works to some extent, he says, but heating the glands improves the result. "LipiFlow is a procedure that places a device into the eye, heats the glands to 108°, massages the glands therefore allowing the glands to function again."

Routine meibomian gland expression may allow optometrists to adopt a mode of care akin to the dental model of routine cleaning, Dr. Karpecki says. Patients may be open to direct payments for such care if price points reach an appropriate level, he says.

• *OCT:* This ever-versatile techology can assist in noninvasive assessment of tear volume by measuring tear meniscus dimensions, helping clinicians determine whether the patient experience aqueous deficient dry eye. Some OCT devices employ software specifically designed to measure the tear meniscus dimensions.

Don't Skimp on the Education

As an optometrist who has studied dry eye and emphasized its care in his practice for the past two decades, Dr. Karpecki admits the first 17 years were challenging. "Patients often weren't getting much better. It was another tough day at clinic." Now, he says, it's just the opposite. "It's rare not to have a happy patient."

Education was a key driver of that, he says. Over the past few years, the amount of knowledge that has been discovered about dry eye, combined with new technologies and therapies, have made a tremendous improvement in diagnosis and treatment. Understanding meibomian gland function and dysfunction, learning more about osmolarity and tear film constituents and how they correlate with real-world symptomology, researching and applying new

treatments—all stem from diligent education.

That means there's even more to learn for those who want to start a dry eye center of excellence, but myriad educational opportunities are available to do so:

• ***Hit the Books:*** Dr. Brujic suggests these three seminal points of reference to build your dry eye clinical skills: the Dry Eye Workshop 2007 (www.tearfilm.org/dewsreport/pdfs/TOS-0502-DEWS-noAds.pdf), the Meibomian Gland Dysfunction Workshop (www.tearfilm.org/mgdworkshop/) and the TFOS International Workshop on Contact Lens Discomfort (www.iovs.org/content/54/11/TFOS7.full).

• ***Learn From the Experts:*** With a wealth of information available—some of it conflicting—Dr. Karpecki suggests studying "the masters," i.e., following the work of those whose careers have been solely or primarily centered on dry eye. "Find the doctors who've gone through the school of hard knocks and spent the majority of their careers in this area. Learn from what they did and look at what they're doing now."

• ***Take a Field Trip:*** Dr. Karpecki suggests visiting dry eye clinics to get a first-hand view of a dry eye center of excellence.

• ***Join a Dry Eye Organization:*** Dr. Schaeffer recommends becoming a part of a dry eye group, such as the Ocular Surface Society of Optometry (http://ossopt.net), which works to increase the awareness and advance the understanding and management of dry eye and ocular surface disease.

• ***Take a Course:*** All the large optometric meetings typically feature dry eye courses ranging from clinical research to practice management. Adds Dr. Schaeffer: "You need to go to the major meetings and meet with the best practitioners who will educate how to develop a dry eye center, how to improve your skills, and how to become an expert."

"I think to be a true center of excellence, you have to have a portion of your clinic time dedicated to dry eye. It can't just 'work in' to your general patient schedule for a given day."
—Dr. Karpecki

Timing and Workflow

"I think to be a true center of excellence, you have to have a portion of your clinic time dedicated to dry eye," Dr. Karpecki says. "It can't just 'work in' to your general patient schedule for a given day." Reserve a dedicated block of time to see dry eye patients exclusively, he says.

For those practitioners beginning to build a dry eye center, Dr. Karpecki suggests starting out with a half-day each week and then transitioning to a full day at the practice when you only see patients with dry eye or other ocular surface disease conditions.

Another key to building a successful dry eye clinic is staffing. Dr. Karpecki has a seasoned lead tech he refers to as his clinic's "quarterback." She works directly with him as his scribe when she's not overseeing the patient flow and work-up of patients. And his lead tech will jump in and help with the work-ups if she notices the patient flow beginning to lag.

"You've got to have a dedicated person who takes on the role of coordinator. They too will develop expertise, same as you," Dr. Karpecki says, but in the areas of productivity, logistics, patient education, clinical anticipation and overall problem-solving.

Eventually, you may want to have other techs whose job responsibilities shift to dry eye work-up on clinic days devoted to such care. On your dry eye day at your practice, you may need to pull a tech from your contact lens side to help as needed, he suggests.

Once you have a dedicated day to see dry eye patients and your staffing is in place, Dr. Karpecki stresses that staff needs to keep a strict eye on the sequencing of care. "Testing has to be systematic," he says. "Once you instill the staining agent, you can no longer do osmolarity or meibomian gland expression," for instance.

At Dr. Karpecki's practice, a patient work-up is done in the following order:

- *Patient questionnaire and history*
- *Osmolarity testing*
- *Diagnostic imaging*
- *Meibomian gland expression*
- *Staining*

Although he hasn't fully incorporated this yet, a new test for MMP-9 measurement would follow osmolarity testing in the lineup order, Dr. Karpecki adds.

Once the testing is complete, he reinforces the patient education by showing patients images of their eyes reflecting their dry eye or other ocular surface disease conditions, as well as animations (from Eyemaginations) that explain the condition.

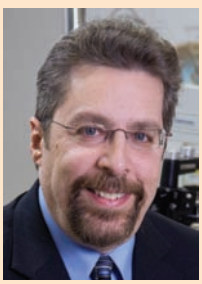
The lead tech will then explain to the patient Dr. Karpecki's tear treatment recommendation, instructions, and when they should schedule their next visit. Handouts are provided to each patient that further discuss their condition or how to appropriately apply warm compresses or take certain medications.

While the prevalence of dry eye increases and research continues to shed new light on the disease, dry eye centers of excellence may be in demand more than ever before. "The opportunities have never been greater, and the need is clearly there," Dr. Epstein says. ■

1. Moss SE, Klein R, Klein BE. Prevalance of and risk factors for dry eye syndrome. Arch Ophthalmol. 2000;118(9):1264-68
2. Scaffidi RC, Korb DR. Comparison of the efficacy of two lipid emulsion eye drops in increasing tear film lipid layer thickness. Eye Contact Lens. 2007;33:38-44.
3. The definition and classification of dry eye disease: report of the Definition and Classification Subcommittee of the International Dry Eye WorkShop (2007). Ocul Surf. 2007 Apr;5(2):75-92.
4. Lemp MA, Bron AJ, et al. Tear osmolarity in the diagnosis and management of dry eye disease. Am J Ophthalmol. 2011 May;151(5):792-798.e1. doi: 10.1016/j.ajo.2010.10.032.
5. Suzuki M, Massingale ML. Tear osmolarity as a biomarker for dry eye disease severity Invest Ophthalmol Vis Sci. 2010 Sep;51(9):4557-61. doi: 10.1167/iovs.09-4596.
6. Chotikavanich S, de Paiva CS, De-Quan L, et al. Production and activity of matrix metalloproteinase-9 on the ocular surface increase in dysfunctional tear syndrome. Invest Ophthal Vis Sci. 2009, Feb 28.
7. Lam H, Bleiden L, De Paiva CS, et al. Tear cytokine profiles in dysfunctional tear syndrome. Am J Ophthalmol. 2009;147:198-206.
8. Nichols KK, Foulks GN, Bron AJ, et al. The international workshop on meibomian gland dysfunction: executive summary. Invest Ophthalmol Vis Sci. 2011 Mar 30;52(4):1922-9.
9. Nelson JD, Shimazaki J, Benitez-del-Castillo JM, et al. The international workshop on meibomian gland dysfunction: report of the definition and classification subcommittee. Invest Ophthalmol Vis Sci. 2011 Mar 30;52(4):1930-7.

I Started With "Why?"

By Arthur Epstein, OD



In truth, opening a brand new practice in the current health care environment is not for the faint of heart or the insecure. Even more so, opening a practice narrowly focused on dry eye—at least from what many of my friends and colleagues have shared—bordered on downright lunacy. So why did I open a dry eye practice "cold"?

Here are the reasons why I launched the Dry Eye Center of Arizona:

• ***Need.*** Arizona is one of the driest places on earth and, with an aging population, the need for dry eye care was evident. As a result, we have literally changed people's lives.

• ***Opportunity.*** An extensive search on the web and among local practices showed a dearth of sites that advertised dry eye care or seemed interested in building that aspect of their practice.

• ***Ability.*** When I left school, I could not even use a dilating agent or topical anesthetic. Times have changed. More than a dozen years devoted to attending every cornea clinic at a university hospital ophthalmology department, plus many years of hands-on experience and learning from some of the brightest minds in eye care, has transformed the way I practice and my skills and abilities, much as it has transformed our profession. Today, optometry owns the ocular surface!

• ***Passion.*** My interest in anterior segment disease has increasingly focused on the ocular surface. I have been immersed in dry eye and ocular surface disease and believe that the Dry Eye Center of Arizona could serve as a local and regional center for dry eye and ocular surface disease care.

• ***Technology.*** Just as the microscope expanded our knowledge and understanding of the world around us, advances in technology have elevated dry eye and ocular surface disease management to new levels. I spend more time explaining test results, treatments and prognoses than I do actual testing.

• ***Confidence.*** The Dry Eye Center of Arizona is part of Phoenix Eye Care, PLLC. I knew my wife and partner, Dr. Shannon Steinhäuser, would quickly build a large and successful primary care optometry practice, which would support the Dry Eye Center. She did, and it has.

In the end, I opened the Dry Eye Center of Arizona for all the right reasons, and I believed I could do it successfully. If your "why" is similar to mine, I encourage you to go for it.

15th Annual Dry Eye Report

When It's More Than Dry Eye

Don't overlook or dismiss these concomitant, confounding conditions that can instigate and exacerbate dry eye. **By Alex Kabiri OD, Michael Cooper OD, Devin Singh, OD, and Matt Burkart, OD**

There are substantially more mechanisms and connections in play within dry eye disease (DED) than just a lack of tears. As defined in 2007 by the Dry Eye WorkShop, DED is a multifactorial disease afflicting millions of individuals worldwide.[1-6] Various intrinsic and extrinsic risk factors, to name just a few, include gender, genetics, tear quality, age, hormones, immune status, nutrition, pathogens, contact lenses/refractive surgery and environmental stress, any of which can alter the harmonious balance within the ocular surface.

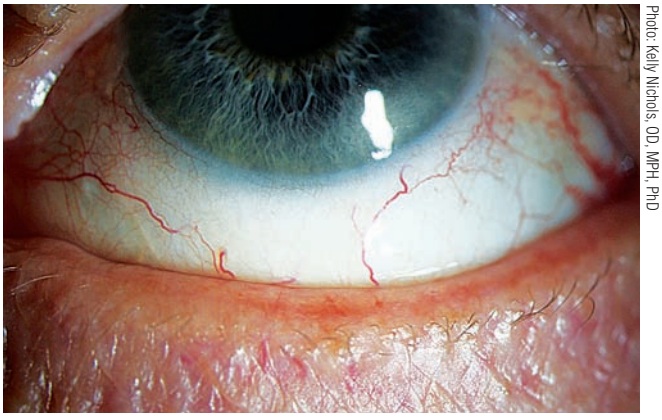


Photo: Kelly Nichols, OD, MPH, PhD

Meibomian gland dysfunction is notoriously intertwined with dry eye disease. Indeed, the majority of dry eye patients are likely to have an underlying meibomian gland disorder.

The pathophysiology of this condition is far reaching—sometimes the outward ocular surface signs can pale in comparison to the chronic internalized damage. Subsequently, complications can increase the potential susceptibility to desiccation and epithelial injury. In turn, a vicious cycle develops in which inflammation amplifies and fosters further damage to the ocular surface by chronic deregulation of the lacrimal functional unit and tear composition.

In this article, both new and recognized clinical insights illustrate how even a small change from a concomitant disease state can have a big impact on ocular surface anatomy.

Identifying the Players

The lacrimal functional unit is a self-contained integrated system comprising the ocular surface (cornea, conjunctiva and meibomian glands) and the lids, which is linked directly to primary and accessory lacrimal glands with a feedback connective loop that includes both motor and sensory nerves.[7-10] Furthermore, when DED exerts its influence on the lacrimal function unit, there is a chronic inflammatory echo, similar to a sound wave, that ripples through the ecosystem, creating an inherent

Do **two-week lenses** give your patients **AMNESIA?**

Replacement Schedule Amnesia

Amnesia (RSA) refers to the loss of memory as a result of a 2-week lens replacement regimen. Symptoms include confusion and disorientation, often marked by patients staring into space wondering if their lenses were replaced "last Tuesday or the Tuesday before?" RSA is easily cured by switching patients to the **simple, comfortable, affordable** 1-Week Lens, Icuity H_2O.

Contact: hydrogelvision.com | 877-336-2482

Think **Easy to Remember**
Think **1-Week**
Think **Icuity H_2O®**

breakdown in tear chemistry. The piercing or triggering effect causing the ocular surface destabilization can be connected to endogenous and microbial stress, antigen localization and epigenetic factors.[11,12]

Tear integrity issues—the chipping away of the epithelial/stromal corneal barrier—can be devastating to the lacrimal function unit. Consequently, exposure of these underlying basal corneal layers creates a vacuum-like effect that increases the risk of ulcerative infection and collagenous scar deposition. In addition, a vast degree of conjunctival goblet cell dropout reduces efficiency of mucin production and causes scarring and wrinkling that can lead to conditions such conjunctival chalasis.[13]

Finally, lid disease cannot be underestimated in its relation to tear quality.[14,15] It has been established that, like glaucoma, there is an inflow (internal) and outflow (external) pathway in the meibomian gland anatomy. For the internal component, meibomian glands contain several acini for which secretory cells called meibocytes produce meibomian oils (meibum). For the external component, the meibum fluid is transported through a ductal system where it is shuttled to the anterior external lid margin surface by the contractile forces of the orbicularis and the muscle of Riolan.

However exquisite this system might seem, the deficiency in the model can be traced back to androgens, blink reflex and, as more recent reports have shown, to *Demodex* mites. Consequently, the pathophysiological breakdown in the lid structure initiates signaling cascades of tumor necrosis factor-alpha and other pro-inflammatory mediators within these tissues manifesting in cytolysis and hyperkeratinization.[14,16,17]

Now that we understand the anatomy, let's take a close look at three common disease states that are often concomitant with DED.

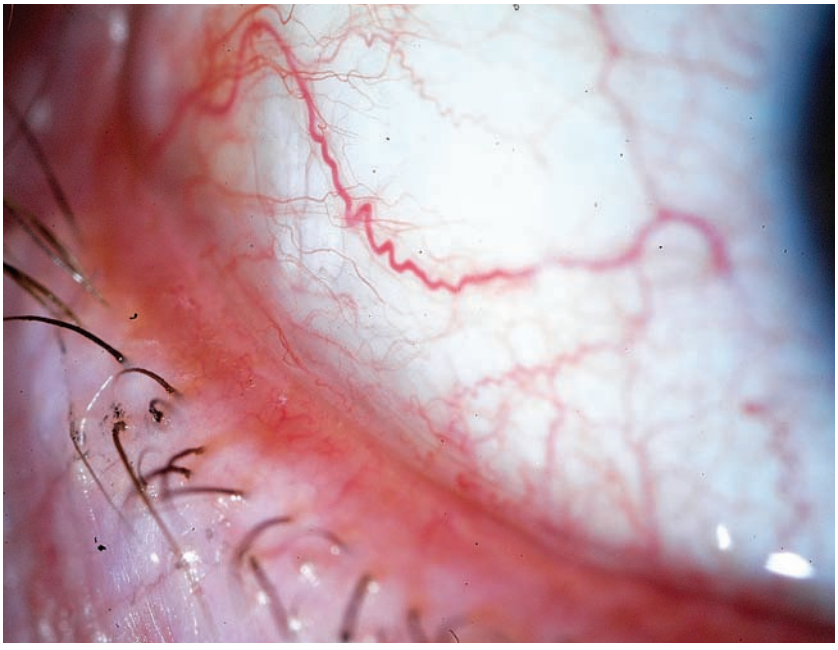


Photo: John P. Herman, OD

With symptoms that are easily mistaken for dry eye disease, conjunctival chalasis is characterized by loose or redundant conjunctival tissue.

Consider Conjunctival Chalasis

Conjunctival chalasis (CChal) is a very common and frustrating ocular surface condition—a study in Japan found that it occurs in more than 98% of individuals over the age of 60 (1,388 patients).[18]

Although it is easily mistaken for DED, CChal's chief distinction is that patients usually present with pain and discomfort at the chalasis site due to redundant conjunctiva (typically temporal) that becomes loose because of the absence of Tenon's fascia.

Symptoms are associated with age greater than 50 and dry eye history, and it has a tendency to occur after cataract surgery, blepharoplasty or other surgical procedure that involved peribulbar or retrobulbar anesthesia.[19] Also of interest, a prospective study found the prevalence of CChal in autoimmune thyroid disease to be as high as 88%.[20]

Even though pain and foreign body sensation are differentiating characteristics, conjunctival chalasis is linked to evaporative dry eye by the dynamic interference in the normal tear film. Clinically, it can be observed as a distinctly elevated tear meniscus centrally, with a broken up nasal and temporal tear film.[21] In addition, check for tear clearance with fluorescein or lissamine green; these stains will quickly show "bunching" of the conjunctival tissue apposed to the inferior lid margin for which the lack of tear flow on the blink reflex is akin to the frictional forces associated with lid wiper epitheliolopathy.[22-24]

Inflammation may have a role in the pathogenesis of conjunctival chalasis. Significant amounts of selective pro-inflammatory markers—matrix metalloproteinase-9

IT'S BECAUSE THEY ARE.

As an independent, you're up against market forces beyond your control. Fortunately, you can partner with companies that empower you to be more competitive, and don't compete against you. Companies that **only succeed when you do**. Companies like HOYA.

HOYA

ALLY OF THE INDEPENDENT ECP

SEE WHY HOYA IS THE RIGHT CHOICE FOR YOUR PATIENTS AND YOUR PRACTICE AT **ecpALLY.com**

(MMP-9), interleukin-1 beta, interleukin-6 and interleukin-8—have been found in the tear film of patients with CChal.[25,26]

A useful pearl when attempting to localize the chalasis site: Ask the patient to point to the affected area. Once identified, place your thumb on the external eyelid and apply gentle pressure to the site while the patient looks up and down. To properly assess the CChal severity, the technique must be done without anesthetic in the eye.

Treat CChal with palliative remedies such as artificial tears, short-term use of a corticosteroid (such as loteprednol) or with amniotic membrane transplantation.[27-30]

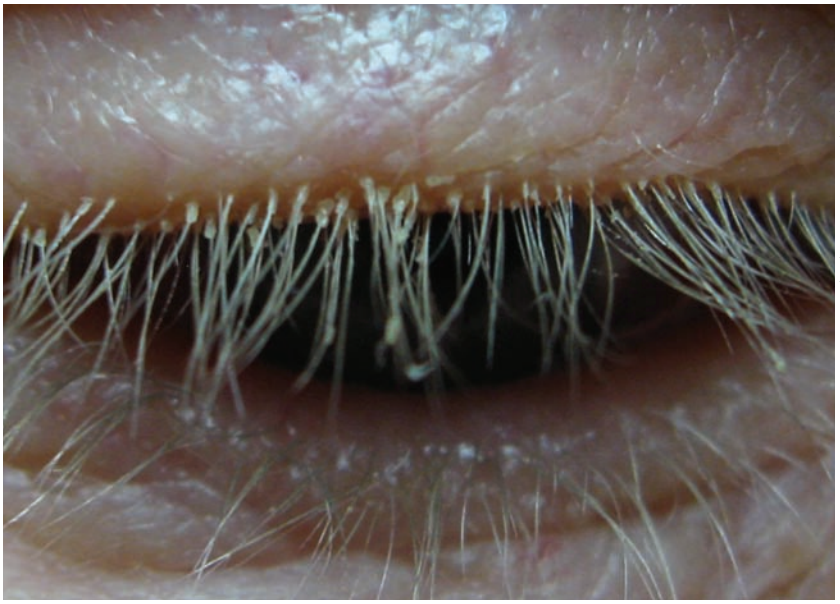


Photo: Ron Melton, OD, and Randall Thomas, OD, MPH

Cylindrical sleeves on the eyelash are characteristic of *Demodex* eyelid infestation.

Demodex: A 'Mitey' Masquerader

Perhaps one of the most confounding conditions in your chair today, tomorrow or the next day will simply be a *Demodex* mite lid infestation. These particular mites are a significant culprit in many cases of blepharitis and can be concomitant with DED due to their chronic nature. Patients with an ocular *Demodex* infestation often complain of itching, burning, redness, crusting at the base of the lashes, blurry vision and dry eye.[31,32] Additionally, some patients will anecdotally report they have more symptoms in the morning, especially after taking a hot shower.

Many of us overlook this small ectoparasite (class *Arachnid*, order *Acarina*) in search of a "better" differential diagnostic solution to why all of our conventional treatment pathways have failed.

To further complicate matters, there are two distinct species:[33,34]

• ***Demodex folliculorum*** has a comparatively long body and proliferates at the base of the lashes (cylindrical dandruff) causing anterior blepharitis. Specifically, *Demodex folliculorum* consume epithelial cells at the hair follicle causing lash distention, hypoplasia and reactive hyperkeratinization, which is commonly observed as trichiasis and madarosis.

• ***Demodex brevis***, as its name implies, has shorter size and burrows deep into the sebaceous and meibomian glands, causing posterior blepharitis. *Demodex brevis* mechanically blocks meibomian gland orifices, leading to insufficient tear lipid secretion, which can be an inflammatory trigger for dry eye.

Interestingly, *Demodex* mites carry their own bacterial reservoirs that can contribute to ocular surface inflammation. The bacteria on the surface of the mite skin has been shown to compete with known staphylococcal species, producing a human host immune response leading to an inflammatory eyelid and periorbital epidermal to subepidermal reaction.[35]

If the disease is in a chronic and progressive phase, the inflammation will spread to the conjunctiva and cornea, potentially leading to infiltrative keratoconjunctivitis, nodular scar deposition and corneal neovascularization.

The good news is that there are now specific treatments available to mediate *Demodex* infestation. Recent to the ophthalmic marketplace is Cliradex (Bio-Tissue), a concentrated tea tree oil derivative based on the potency of the active ingredient 4-terpineol to kill *Demodex* mites.[36] Other available treatments include BlephEx (Rysurg) and the Demodex Convenience Kit (Ocusoft), as well as compounded off-label oral and topical ivermectin, which is currently used in the veterinary community and now being studied in human subjects.[37,38]

Recurrent Corneal Erosion

Recurrent corneal erosion (RCE) is a painful and sometimes even incapacitating condition for many patients. The majority of cases

MANUFACTURED
IN THE **USA**

Power at Your Fingertips

The Smart System® Tablet for Testing Patients at Near

The M&S Smart System Tablet can be used as more than just a customizable controller for the Smart System. We've developed five tablet protocols for testing patients at near: Near Acuity testing, ColorCheck color screening, Letter Contrast Sensitivity testing, Stereo testing, and the Amsler Grid.

All the tests are comprehensive, easy-to-use, produce accurate results, and come standard on each tablet when purchased with a system. You can also purchase them individually as apps (for Android) through our online web store. Please visit **mstech-eyes.com/store** for info and pricing.

Call us toll-free to learn more about the Smart System Tablet and all of our Smart System products.

****Demo the Tablet at Vision Expo West, Booth MS11039****

M&S holds US Patents 7,354,155; 7,926,948; 8,425,040; 8,167,429; 8,419,184 & 8,550,631. Other Patents Pending.

M&S® TECHNOLOGIES

The First Choice in Vision Testing Systems

www.mstech-eyes.com
1-877-225-6101 08P_REV0914

occur secondary to a superficial traumatic injury, yet the pathophysiology of RCE is only partially understood.[39]

In the last several years, researchers have used confocal laser technologies to further investigate the etiology of recurrent erosions and were able to establish that there was altered epithelial cell types coupled with inflamed stromal keratocytes compared to those cells found within normal corneal physiology. The hypotheses from these studies indicate the origins of the trauma—whether from a glancing fingernail abrasion, edge piece of paper, leaf or branch, DED, previous herpetic keratitis, or other epithelial/stromal dystrophies—are rooted in a smoldering inflammatory cascade of MMP-9 and MMP-2. The expression of these particular enzymes causes an upregulation of pro-inflammatory mediators, leading to basement membrane degradation and poor epithelial membrane adhesion. Consequently, the process of re-epithelialization to recurrent sloughing off of these same cells could be directly related to the preexisting events.[40-42]

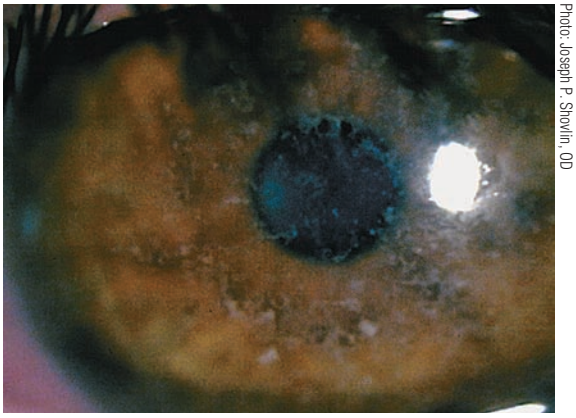


Photo: Joseph P. Shovlin, OD

Deposits in the epithelial basement membrane, shown here, may be partly to blame for recurrent corneal erosion, which is sometimes mistaken for dry eye disease.

The link between RCE and DED is mechanical in nature. Logically, when the ocular surface is desiccated via a dry eye mechanism from contact lens wear, previous surgery, autoimmune disease or other anterior basement/stromal dystrophy condition, the dominos are set into motion priming the epithelium to dismount by the shearing force of the lids sliding over the weakened

Pardon the Interruption: How Refractive Surgery Disrupts the Ocular Surface

By Matthew A. Burkart, OD

Despite great advances in refractive surgery, patients still suffer post-surgically from chronic dry eye.

Much of it has to do with the nerves. The cornea is one of the most densely innervated physiological structures of the body. A vast majority of these nerves branch off from the posterior corneal nerves and form three key networks throughout the cornea.[1] One network is located in the mid-stroma, another in the anterior stroma and the third is located throughout the epithelium.[1] The cornea also contains a high density of sympathetic nerve fibers that aid in epithelial metabolism, proliferation and wound healing.[1] Given that refractive surgery of all types affects one or all of these networks, it's no surprise that these patients can have postoperative chronic dry eye symptoms.[2]

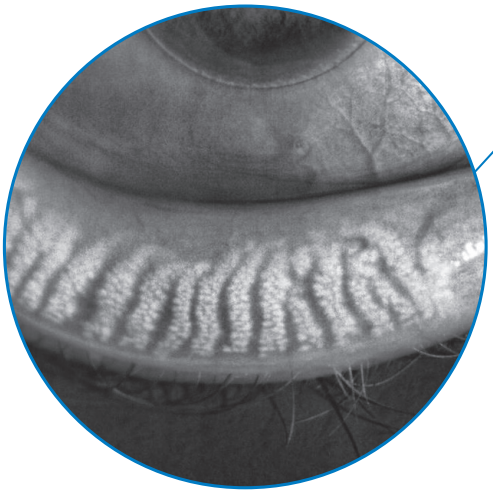
Evidence of this dry eye phenomena can be observed at the one-week, one-month, three-month and six-month follow-up visits using tear break-up time, Schirmer's test and corneal sensitivity as measures.[3,4,8,9] Patients who have the most issues postoperatively tend to have a greater attempted refractive error correction, a greater ablation depth, are older and often are female.[4,6,7]

In addition, multiple retrospective studies have shown that patients with tear film instability before surgery are more likely to experience greater and more persistent symptoms postoperatively.[2,5,7,8] One of the most significant measures of postoperative dry eye symptoms is a value less than 10mm from a preoperative Schirmer's test.[8]

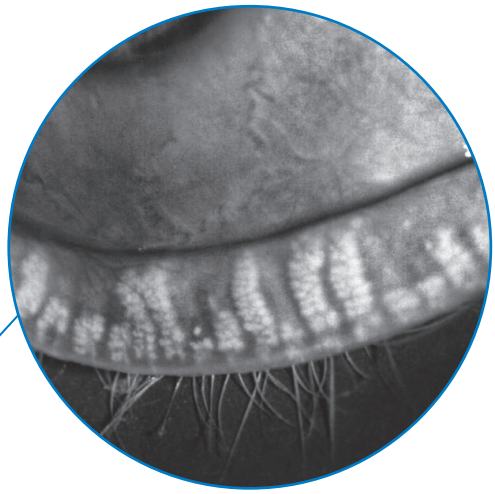
While DED is commonly linked to many other disease states—thyroid disease, Sjögren's syndrome, hormonal changes, rheumatoid arthritis and diabetes—the fact that it's also present in our refractive surgery patients cannot be dismissed. To truly serve our patients, we must take care to screen appropriately and treat our patients who suffer from dry eye preoperatively. By taking this extra proactive step, treatment outcomes will improve to ensure a more successful experience for our surgical patients.

1. Remington LA. Clinical Anatomy of the Visual System, 2nd ed. St. Louis: Elsevier/Butterworth-Heinemann; 2005.
2. Ambrósio R Jr, Tervo T. Wilson SE. LASIK-associated dry eye and neurotrophic epitheliopathy: Pathophysiology and strategies for prevention and treatment. J Refract Surg. 2008 Apr;24(4):396-407.
3. Donnenfeld ED, Solomon K, Perry HD, et al. The effect of hinge position on corneal sensation and dry eye after LASIK. Ophthalmology. 2003 May;110(5):1023-9.
4. Shoja MR, Besharati MR, Sadoughi S. Dry eye after LASIK for myopia: Incidence and risk factors. Eur J Ophthalmol. 2007 Jan-Feb;17(1):1-6.
5. Albietz JM, Lenton LM. Management of the ocular surface and tear film before, during, and after laser in situ keratomileusis. J Refract Surg. 2004 Jan-Feb;20(1):62-71.
6. De Paiva CS, Chen Z, Koch DD, et al. The incidence and risk factors for developing dry eye after myopic LASIK. Am J Ophthalmol. 2006 Mar;141(3):438-45.
7. Albietz JM, Lenton LM, McLennan SG. Chronic dry eye and regression after laser in situ keratomileusis for myopia. J Cataract Refract Surg. 2004 Mar;30(3):675-84.
8. Yu EY, Leung A, Rao S, Lam DS. Effect of laser in situ keratomileusis on tear stability. Ophthalmology. 2000 Dec;107(12):2131-5.
9. Battat L, Macri A, Dursun D, Pflugfelder SC. Effects of laser in situ keratomileusis on tear production, clearance, and the ocular surface. Ophthalmology. 2001 Jul;108(7):1230-5.

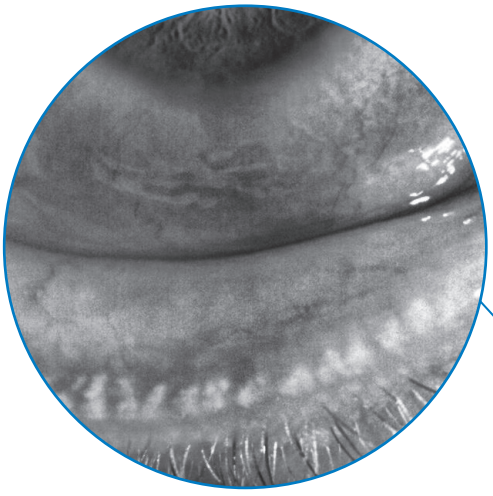
Give MGD a Second Glance



normal meibomian gland structure



structural change (duct dilation,
gland atrophy and drop out)



structural change
(severe gland atrophy and drop out)

Meibomian Gland Dysfunction isn't always visible.

Identify, diagnose and treat MGD without all the dry eye complexity.
Visit us at MS2053, Vision Expo West to learn more about the TearScience® Solution for MGD, featuring LipiView® and LipiFlow®.

TearScience®

tearscience.com **919-459-4880** **sales@tearscience.com**

tissue. As a result, the patient tends to awaken in the middle of the night or morning with a neuropathic shockwave of corneal pain.[43]

From a clinical perspective, be sure to clearly communicate the visual expectations and prognosis. Furthermore, when managing recurrent corneal erosion, more than one treatment will be necessary, especially in severe cases. Fortunately, multiple modalities are available, such as oral doxycycline, autologous serum, topical loteprednol, cyclosporine, phototherapeutic keratectomy and amniotic corneal transplantation.

By discussing the various therapeutic options and surgical techniques, the optometrist can ensure a preventive strategy that will make the experience easier and more tolerable for the patient.[44]

For ocular surface disease management, today is the day to effect change. Take the opportunity to grasp that "carpe diem moment" and delve deeper into a patient's history. You could make their day, or even change their life. ■

Drs. Kabiri and Singh practice at Malik Eye Care, a multi-location MD/OD group setting in Queens, New York.

Dr. Cooper practices at an MD/OD group setting in Willimantic, Conn.

Dr. Burkart practices at a multi-location OD setting in Zionsville, Ind.

1. The definition and classification of dry eye disease: report of the Definition and Classification Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5(2):75-92.
2. The epidemiology of dry eye disease: report of the Epidemiology Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5(2):93-107.
3. Methodologies to diagnose and monitor dry eye disease: report of the Diagnostic Methodology Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5:108-152.
4. Design and conduct of clinical trials: report of the Clinical Trials Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5(2):153-162.
5. Management and therapy of dry eye disease: report of the Management and Therapy Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5(2):163-178.
6. Research in dry eye: report of the Research Subcommittee of the International Dry Eye WorkShop. Ocul Surf. 2007 Apr;5(2):179-93.
7. Beuerman RW, Mircheff A, Pflugfelder SC, Stern ME: The lacrimal functional unit. In: Pflugfelder SC, Beuerman RW, Stern ME, ed. Dry eye and ocular surface disorders, New York: Marcel Dekker; 2004.
8. Stern ME, Beuerman RW, Fox RI, et al. The pathology of dry eye: the interaction between the ocular surface and lacrimal glands. Cornea. 1998 Nov;17(6):584-9.
9. Stern ME, Beuerman RW, Fox RI, et al. A unified theory of the role of the ocular surface in dry eye. Adv Exp Med Biol. 1998;438:643-51.
10. Beuerman RW, Maitchouk DY, Varnell RJ, Pedroza-Schmidt L. Interactions between lacrimal function and the ocular surface. In: Kinoshita S, Ohashi Y, eds. Proceedings of the 2nd Annual Meeting of the Kyoto Cornea Club. The Hague, the Netherlands: Kugler Publications; 1998:1-10.
11. Zierhut, M, Stern ME, Sullivan DA. Immunology of the Lacrimal Gland, Tear Film and Ocular Surface. Abingdon, UK: Taylor and Francis; 2005.
12. McDermott AM, Perez V, Huang AJ. et al. Pathways of corneal and ocular surface inflammation: a perspective from the Cullen Symposium. Ocul Surf. 2005 Oct;3(4 Suppl):S131-8.
13. Behrens A, Doyle JJ, Stern L, et al; Dysfunctional tear

REVIEW® OF OPTOMETRY

CONTINUE YOUR EDUCATION WITH REVIEW

OUR FLAGSHIP TITLE, *REVIEW OF OPTOMETRY*, IS THE MARKET'S LEADING RESOURCE FOR ALL OF YOUR OPTOMETRY NEEDS.

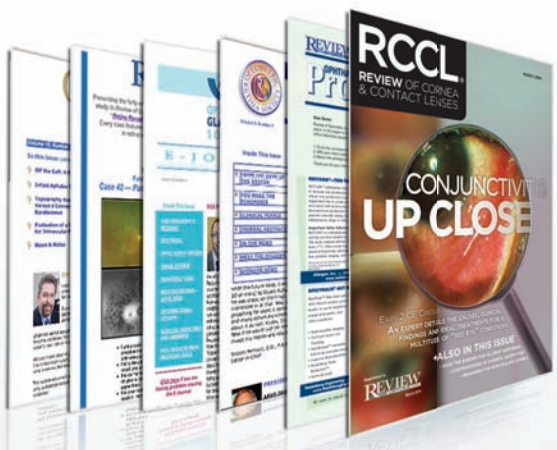
Review of Optometry is your primary source for ground-breaking clinical information as well as timely news, market trend information and continuing education programs.

Review of Cornea & Contact Lenses serves as a valuable resource for all practitioners and features detailed articles focusing on various fitting methods, solutions and corneal cases. Also available is the ***Review of Cornea & Contact Lenses "Annual Contact Lenses & Lens Care" Guide***, a yearly publication detailing the newest lenses and lens care products.

The ***Review*** Group's ***Ophthalmic Product Guide*** brings you the newest and most innovative products on the market. Published every February and July, the guide provides concise information about new literature, drugs and equipment designed to help your practice thrive.

The ***Review*** Group also offers valuable **continuing education** sessions in both print and online formats, allowing a convenient way for you to earn **CE credits**. In addition, *Review* also offers an impressive fleet of ***free e-newsletters***, such as Optometric Physician, the Optometric Retina Society quarterly e-newsletter and the Optometric Glaucoma Society E-Journal, so you can keep up to date on breaking news and the latest research online.

The *Review* Group is dedicated to the constant growth and education of the profession. *Review* offers many different publications and services to help enhance your practice and patient care.



On top of these products, the *Review* Group also spearheads meetings and conferences, bringing together experts in the field and providing a forum for practitioners to earn CE credits and learn from others in the profession.

www.revoptom.com

jmi

Jobson Medical Information LLC
The *Review* Group

syndrome study group. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. Cornea. 2006 Sep;25(8):900-7.
14. Korb DR, Henriquez AS. Meibomian gland dysfunction and contact lens intolerance. J Am Optom Assoc. 1980 Mar;51(3):243-51.
15. Nichols KK, Foulks GN, Bron AJ, et al. The international workshop on meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2011 Mar 30;52(4):1917-2064.
16. Smith KR, Thiboutot DM. Thematic review series: skin lipids. Sebaceous gland lipids: friend or foe? J Lipid Res. Feb 2008;49(2):271-81.
17. Yano M, Kishida E, Iwasaki M. Docosahexaenoic acid and vitamin E can reduce human monocytic U937 cell apoptosis induced by tumor necrosis factor. J Nutri. May 2000;130(5):1095-1101.
18. Mimura T, Yamagami S, Usui T, et al. Changes of conjunctivochalasis with age in a hospital-based study. Am J Ophthalmol. 2009 Jan;147(1):171-7.
19. Hovanesian JA. Diagnosis and surgical treatment of conjunctival chalasis. Ocular Surgery News. May 5, 2009. Available at: www.healio.com/ophthalmology/blogs/hovanesian/diagnosis-and-surgical-treatment-of-conjunctival-chalasis. Accessed July 21, 2014.
20. de Almeida SF, de Sousa LB, Vieira LA, et al. Clinic-cytologic study of conjunctivochalasis and its relation to thyroid autoimmune diseases: prospective cohort study. Cornea. 2006 Aug;25(7):789-93.
21. Yeu E. Strategies for managing difficult-to-treat DED. Cataract & Refractive Surgery Today. 2013 March;13(3):44-5.
22. Korb DR, Greiner JV, Herman JP, et al. Lid-wiper epitheliopathy and dry-eye symptoms in contact lens wearers. CLAO J. 2002 Oct;28(4):211-6.
23. Korb DR, Herman JP, Greiner JV, et al. Lid wiper epitheliopathy and dry eye symptoms. Eye Contact Lens. 2005 Jan;31(1):2-8.
24. Korb DR, Herman JP, Finnemore VM, et al. An evaluation of the efficacy of fluorescein, rose bengal, lissamine green, and a new dye mixture for ocular surface staining. Eye Contact Lens. 2008 Jan;34(1):61-4.
25. Acera A, Rocha G, Vecino E, et al. Inflammatory markers in the tears of patients with ocular surface disease. Ophthalmic Res. 2008 Oct;40(6):315-21.
26. Erdogan-Poyraz C, Mocan MC, Bozkurt B, et al. Elevated tear interleukin-6 and interleukin-8 levels in patients with conjunctivochalasis. Cornea. 2009 Feb;28(2):189-93.
27. Kruse FE, Meller D. Amniotic membrane transplantation for reconstruction of the ocular surface. Ophthalmologe. 2001 Sep;98(9):801-10.
28. Meller D, Tseng SC. Reconstruction of the conjunctival and corneal surface. Transplantation of amniotic membrane. Ophthalmologe. 1998 Dec;95(12):805-13.
29. Meller D, Tseng SC. Conjunctivochalasis: literature review and possible pathophysiology. Surv Ophthalmol. 1998 Nov-Dec;43(3):225-32.
30. Huang Y, Sheha H, Tseng SC. Conjunctivochalasis interferes with tear flow from fornix to tear meniscus. Ophthalmology. 2013 Aug;120(8):1681-7.
31. Basta-Juzbasic A, Subic JS, Ljubojevic S. Demodex folliculorum in development of dermatitis rosaceiformis steroidica and rosacea-related diseases. Clin Dermatol. 2002 Mar-Apr;20(2):135-40.
32. English FP,Nutting WB. Demodicosis of ophthalmic concern. Am J Ophthalmol. 1981 Mar;91(3):362-72.
33. Gao YY, Di Pascuale MA, Li W, et al. High prevalence of Demodex in eyelashes with cylindrical dandruff. Invest Ophthalmol Vis Sci. 2005;46:3089-3094. Invest Ophthalmol Vis Sci. 2005 Sep;46(9):3089-94.
34. Bevins CL, Liu FT. Rosacea: skin innate immunity gone awry? Nat Med. 2007 Aug;13(8):904-6.
35. Wolf R, Ophir J, Avigad J, et al. The hair follicle mites (Demodex spp.). Could they be vectors of pathogenic microorganisms? Acta Derm Venereol. 1988;68(6):535-7.
36. Tighe S, Gao YY, Tseng SC, et al. Terpinen-4-ol is the most active ingredient of tea tree oil to kill Demodex mites. Transl Vis Sci Technol. 2013 Nov;2(7):2.
37. Salem DA, El-Shazly A, Nabih N, et al. Evaluation of the efficacy of oral ivermectin in comparison with ivermectin-metronidazole combined therapy in the treatment of ocular and skin lesions of Demodex folliculorum. Int J Infect Dis. 2013;17(5):e343-7.
38. Schachter S. A different approach to treating Demodex blepharitis. Jan 3, 2014, Available at: http://optometrytimes.modernmedicine.com/optometrytimes/news/different-approach-treating-demodex-blepharitis. Accessed July 29, 2014.
39. Trobe JD, Laibson PR. Dystrophic changes in the anterior cornea. Arch Ophthalmol. 1972 Apr;87(4):378-82.
40. Labbe A, Nicola RD, Dupas B, et al. Epithelial basement membrane dystrophy: evaluation with the HRT II Rostock Cornea Module. Ophthalmology. 2006 Aug;113(8):1301-8.
41. Chikama T, Takahashi N, Wakuta M, et al. In vivo biopsy by laser confocal microscopy for evaluation of traumatic recurrent corneal erosion. Mol Vis. 2008;14:2333-9.
42. Sakimoto T, Shoji J, Yamada A, et al. Upregulation of matrix metalloproteinase in tear fluid of patients with recurrent corneal erosion. Jpn J Ophthalmol. 2007 Sep-Oct;51(5):343-6.
43. Mandell RB, Fatt I. Thinning of the human cornea on awakening. Nature. 1965;208:292.
44. Friedman NJ. Treating recurrent corneal erosion syndrome. Advanced Ocular Care. 2010 Jul/Aug;1(5):38-40.

15th Annual Dry Eye Report

Sizing Up Ocular Surface Therapies

Optometrists now have a wide array of products and procedures to get chronic dry eye symptoms under control. By James A. Williamson, OD

Decades ago, eye care providers didn't have many over-the-counter (OTC) dry eye treatments, let alone pharmaceutical or surgical options. That's not the case today, however. Now, a plethora of viable treatments are available for dry eye and meibomian gland dysfunction patients.

This contemporary surge in effective choices largely is due to an improved understanding of ocular surface disease and patient care.

The multifactorial etiology of dry eye warrants a detailed diagnostic approach. Thus, the prudent management course may not involve ocular surface treatments at all, but rather environmental, functional or pharmacological modifications. Focused and individualized therapy should only be determined following a thorough history and in-office work-up.

Conventional Treatments

Lid scrubs, compresses and digital massage still comprise the first line of dry eye treatment. Single-use lid scrubs offer convenience to those who desire an alternative to traditional methods, such as cleansing with baby shampoo. Hot compresses should be prescribed with the awareness that meibomian secretions from severely obstructed glands possess a higher melting point.[1] Digital lid massage effectively targets the meibomian glands, but may cause warping if performed over a heated cornea.[2]

OTC: "Oh, the Choices"

Many dry eye patients self-treat before seeking professional help.[3] And given a choice, cost-conscious dry eye sufferers likely will lean toward less expensive, store-brand, artificial tears. Although generally well tolerated when dosed up to six times per day, these BAK-preserved lubricants can lead to corneal toxicity and increased inflammation.[4,5] Prescribing beyond this interval, however, merits a switch to either a preservative-free option or one that breaks down upon ocular surface contact (e.g., sodium perborate). However, increasing the viscosity will permit a less frequent dosing schedule.

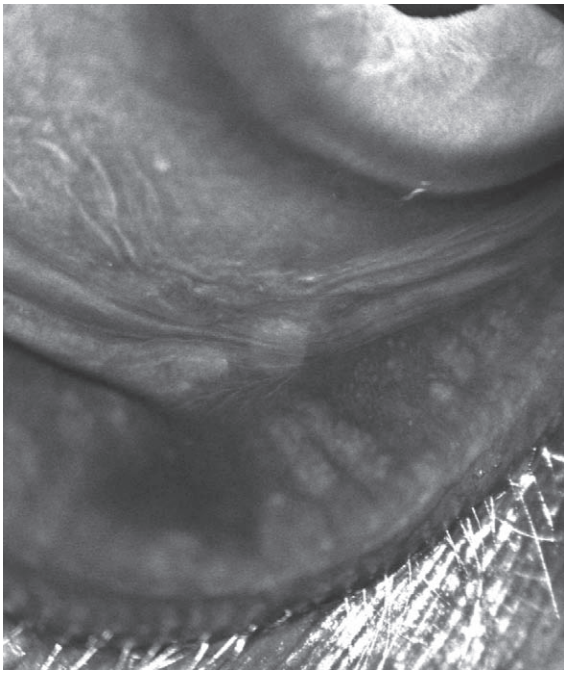
As evaporative dry eye accounts for the majority of cases, lipid-based products can provide symptomatic relief in many (though not necessarily all) patients.[6] These include FreshKote (Focus Laboratories), Refresh Optive Advanced (Allergan), Retaine MGD (Ocusoft), Systane Balance (Alcon) and Soothe XP (Bausch + Lomb). Specific brand selection generally is left up to the eye care provider's discretion. Single-dose, unpreserved artificial tears also are also available, such as

LACRISERT®

(hydroxypropyl cellulose ophthalmic insert)

BAUSCH + LOMB

US/LAC/14/0008



On infrared meibography, gland dropout appears as dark areas vs. light areas in meiboscopy. Glands exhibit multiple morphological changes, which may include dilation, truncation or complete atrophy.

Refresh Optive Sensitive (Allergan).

Fish oil supplementation is a potential alternative to topical dry eye treatments. Long-chain omega-3 fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]) present in fish oil capsules provide an anti-inflammatory effect, which often helps improve the signs and symptoms of meibomian gland dysfunction (MGD).[7] Common side effects of fish oil supplementation include gastrointestinal complications and "fishy" burps.

To complement any of the aforementioned dry eye treatments, you might consider the use of moisture goggles. Some technologies, such as Tranquileyes (Eyeeco), have been specifically designed for dry eye treatment; however, simple swimming goggles may be used, as well.[8]

Stick to the Script

In recent years, optometrists have written more prescriptions for topical steroids and nonsteroidal inflammatory agents than ever before.[9] And, without question, underlying inflammation is one of the primary causes of dry eye.[10]

When considering prescription treatments for dry eye, most eye care providers start with Restasis (cyclosporine, Allergan) BID in conjunction with a topical steroid for several weeks or months to rapidly suppress inflammation (*see "A Word About Steroids," below*). After a year of Restasis treatment, dosing may be reduced to QD in order to maintain a therapeutic benefit.[11]

The broad-spectrum topical macrolide, azithromycin, also has been shown to decrease inflammation and improve lipid alteration in patients with dry eye disease and meibomian gland dysfunction (MGD).[12,13] A four-week course of the sustained delivery drop, dosed BID for two days then once daily, may effectively improve foreign body sensation and meibomian gland obstruction.[14]

Tetracyclines offer an oral alternative to inhibit meibomian gland inflammation.[12] Of these agents, doxycycline appears to be the common selection.[12] Dosing varies depending upon presentation severity, and may be as high as 100mg BID (although doses as low as 40mg QD can be effective).[15] When prescribing tetracyclines, be sure to educate patients about the potential effect on oral contraception.

Secretagogues represent an additional oral treatment for dry eye, though with undesirable parasympathomimetic side effects. In a systematic review of published trials, both cevimeline and pilocarpine demonstrated symptomatic improvement in dry eye.[16]

Outside of topical and oral treatments, once-a-day Lacrisert (hydroxypropyl cellulose ophthalmic insert, Valeant) may be a potential option. These inserts work well for dry eye patients who have mental or physical conditions that prohibit topical drop instillation, or if convenience is a priority.[17]

Should the aforementioned therapeutic options fail, compounded topical tacrolimus—an immunosuppressive agent used after organ transplant—has been shown to improve tear stability and ocular surface status.[18] It is also available as an ointment, making overnight coverage possible.

Additionally, compounded 3% testosterone cream applied at bedtime has been shown to increase

A Word About Steroids

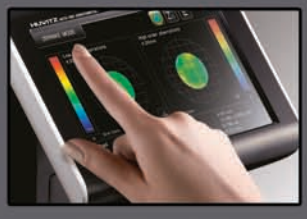
Most ophthalmic steroids are ketone-based, and therefore put patients at risk for intraocular pressure increase. So, an ester-based agent, such as Lotemax (loteprednol etabonate, Bausch + Lomb) may be preferable.[33] For increased potency, consider the steroidal emulsion Durezol (difluprednate, Alcon), which does not require vigorous bottle shaking.

Once the inflammation is controlled, continue adjunctive therapy with artificial tears, fish oil supplementation and/or hot compresses. Keep in mind that some patients subsequently require periodic steroid dosing to control recalcitrant inflammation.

Complete Patient Luxury

DIGITAL REFRACTION SYSTEM

Huvitz
Pacing Progress toward People



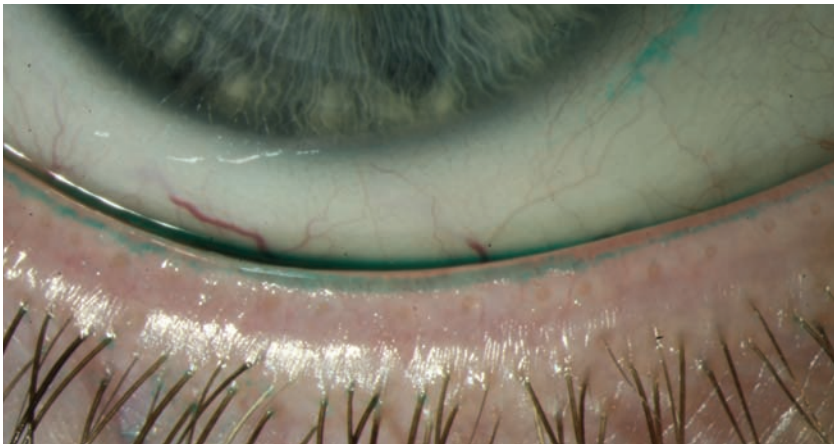
COBURN TECHNOLOGIES

YOU DON'T HAVE TO WAIT FOR EXPO PRICES
CALL FOR DETAILS AND SPECIAL OFFERS

1-800-COBURN-1 (800-262-8761)
www.coburntechnologies.com

VISION EXPO WEST LP 6075
LAS VEGAS, NV - SEP 18-20

AAO 2014 MEETING BOOTH 930
CHICAGO, IL - OCT 18-21



Lissamine green staining on the line of Marx. An irregular, thickened or anteriorly placed line of Marx can produce ocular surface symptoms and could serve as an indication for the debridement-scaling technique.

tear osmolarity and improve evaporative dry eye.[19,20] Exogenous testosterone use, however, may be an independent risk factor for central serous chorioretinopathy.[20]

Plugs, Probes and Power Tools

When punctal plugs first gained popularity more than 20 years ago, many of us simply used the devices when patients complained of dryness. However, today we know that any inflammatory component must be addressed before punctal plugs may be considered. Also, depending upon the type and size of plug selected, extrusion may occur.

Before the widespread acceptance of punctal plugs, thermal cautery of the puncta was a frequently employed treatment option for recurrent dry eye. A study published in 1998 indicated that this technique yielded better subjective improvement in dry eye symptoms than argon laser punctal ablation.[21] To minimize tissue destruction, some surgeons prefer a low-temperature/high-frequency cautery device.[22]

For meibomian glands, the debridement-scaling technique promoted by Donald Korb, OD, and Caroline Blackie, OD, PhD, may be used to remove accumulated tissue and debris from the line of Marx (LOM) and keratinized lid margin.[23] To accomplish this, apply lissamine green dye and use a lateral motion with the golf club spud along the LOM to remove the stained cells.

If the meibomian glands are obstructed, several methods exist. Intraductal probing, for example, allows the eye care provider to penetrate and clear the meibomian orifice. First described by ophthalmologist Steven L. Maskin, this technique yields symptomatic improvement in all patients tested.[24,25]

Additionally, an automated device, the LipiFlow Thermal Pulsation System (TearScience), could be considered. The technology includes a lid warmer (which resembles a larger scleral lens) that heats to 42.5°C and an inflatable/deflatable cup that rests over top of the closed eyelids and "milks" the meibomian glands. One study suggested that the LipiFlow system improved both signs and symptoms of dry in after 12 minutes of use.[26]

Patient, Heal Thyself

Severe dry eye or persistent epithelial defects may warrant the use of autologous serum (AS) drops. This preservative-free, 20% to 100% dilution is formulated using the patient's own nutrient-rich blood (*see "How and Why to Make Autologous Serum," March 2012*). These drops contain growth factors, fibronectin and vitamins that help to promote ocular surface healing.[27,28]

AS drops generally are dispensed in single-use vials, which can be preserved for at least one month if kept refrigerated and three months if kept frozen. The typical dosing frequency of AS ranges from Q1H to QID, depending upon disease severity.[27]

Take note that patients with HIV, hepatitis C or syphilis are not candidates for AS formulation, and instead must use ready-made, ABO-matched serum.[29]

Additionally, commercially available amniotic membrane grafts also promote ocular surface healing via anti-scarring, antimicrobial and anti-inflammatory effects.[30] Practitioners may choose choose from three different graft sizes, depending on the level of inflammation. They also may be employed after conjunctivochalasis surgery to prevent tear meniscus changes and delayed tear clearence.[31,32]

Today, eye care providers are in an excellent position to treat dry eye disease. Research into novel, even more advanced treatments remains ongoing, and new options will continue to trickle down the pipeline.

However, finding the right treatment—or combination of treatments—remains the key to successful, individualized dry eye care. ■

retaine® MGD™

Retention Starts with Superior Coverage

Triple Action Tear Film Enhancement

Lipid Restoration | Epithelial Healing | Osmocorrection

For FREE samples and more information, call (800) 233-5469 or visit VEW Booth #MS10049

OCuSOFT®

Stop, Look and Listen

Pinpointing the underlying cause of dry eye will help you most effectively manage patient symptoms. Here's how to do it:

• ***History***. To start, take a detailed history. Assess functional issues (e.g., prolonged reading, computer use and monitor height), visual fluctuations, environmental factors (e.g., working conditions, smoking status, driving commute duration, use of household humidifiers, contact lens wear, ceiling fan use), current medications and any history of ocular surgery or trauma. For contact lens patients, document both wearing schedules and the solutions used. Also, be sure to offer dry eye questionnaires, such as the Ocular Surface Disease Index (OSDI) or the Standard Patient Evaluation of Eye Dryness (SPEED, TearScience).

• ***Testing***. Next, perform a variety of diagnostic tests. Start with fluorescein dye instillation to assess tear film break-up time (TFBUT), corneal staining and the inferior meniscus for tear clearance, volume and debris. Avoid meibomian gland manipulation prior to TFBUT assessment, as this may negatively affect results. Add lissamine green to evaluate conjunctival staining, lid wiper epitheliopathy and anterior displacement of the line of Marx. Use grading scales for both dyes, if desired. Less than 10mm of strip wetting on the five-minute Schirmer test with anesthesia signals aqueous deficiency.

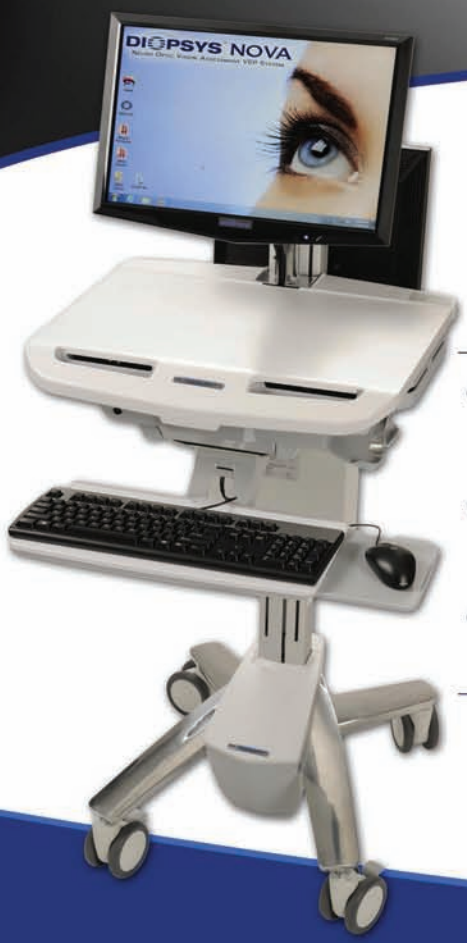
• ***Work-up***. During the lid evaluation, look for blepharitis, floppy eyelid syndrome and meibomian gland dysfunction with expression and transillumination. Take note of lid apposition, lagophthalmos and blink rate. Calculating the Ocular Protection Index, or OPI (defined as TFBUT divided by the time between blink), via a standardized approached also may help. An OPI <1 is indicative of an elevated risk for dry eye signs and symptoms.

Further, during the work-up, don't forget to slow down and give the patient some face time. In optometry school, I can recall a fellow intern examining a patient with a heavy accent who presented with a complaint of "eye crust." Dumbfounded, after an unremarkable slit lamp evaluation, the student's heart dropped when the staff doctor simply looked at the patient and noted his "eye-crossed" appearance. So, take your time, talk to the patient and answer any questions they may have.

• ***Education***. Because anxiety and depression frequently are associated with dry eye, you should educate the patient about the chronic nature of the condition and that—even with appropriate management—the symptoms may never be completely eliminated.[34] The more dry eye patients subscribe to our evidence-based recommendations, the less likely they will be to deviate from or forthrightly abandon prescribed treatment regimens.

Dr. Williamson is the residency supervisor at the Memphis VA Medical Center in Tennessee. He has no direct financial interests in any of the products mentioned.

1. Blackie CA, Solomon JD, Greiner JV, et al. Inner eyelid surface temperature as a function of warm compress methodology. Optom Vis Sci. 2008 Aug;85(8):675-83.
2. McMonnies CW, Korb DR, Blackie CA. The role of heat in rubbing and massage-related corneal deformation. Cont Lens Anterior Eye. 2012 Aug;35(4):148-54.
3. Clegg JP, Guest JF, Lehman A, Smith AF. The annual cost of dry eye syndrome in France, Germany, Italy, Spain, Sweden and the United Kingdom among patients managed by ophthalmologists. Ophthalmic Epidemiol. 2006;13(4):263-74.
4. Subcommittee T. Management and Therapy of Dry Eye Disease : Report of the Management and Therapy Subcommittee. 2007;5(2):163-78.
5. Sarkar J, Chaudhary S, Namavari A, et al. Corneal neurotoxicity due to topical benzalkonium chloride. Invest Ophthalmol Vis Sci. 2012 Apr 6;53(4):1792-802
6. Kaercher T, Thelen U, Brief G, et al. A prospective, multicenter, noninterventional study of Optive Plus in the treatment of patients with dry eye: the prolipid study. Clin Ophthalmol. 2014 Jun 17;8:1147-55.
7. Oleñik A, Jiménez-Alfaro I, Alejandre-Alba N, Mahillo-Fernández I. A randomized, double-masked study to evaluate the effect of omega-3 fatty acids supplementation in meibomian gland dysfunction. Clin Interv Aging. 2013;8:1133-8.
8. Eyeeco. Tranquileyes. Available at: www.eyeeco.com/category/icategory_id/37/tranquileyes_eye_hydrating_therapy.html. Accessed July 5, 2014.
9. Gonzalez A, Lakhani R, Bennett N, Paz C. A twelve-quarter quantitative analysis of ophthalmic drugs prescription-writing by optometrists in the United States. Clin Optom. 2014:5-10. Available at: www.dovepress.com/articles.php?article_id=16006. Accessed June 29, 2014.
10. Gumus K, Cavanagh DH. The role of inflammation and antiinflammation therapies in keratoconjunctivitis sicca. Clin Ophthalmol. 2009;3:57-67. Epub 2009 Jun 2
.
11. Su MY, Perry HD. The effect of decreasing the dosage of cyclosporine A 0.05% on dry eye disease after 1 year of twice-daily therapy. Cornea. 2011 Oct;30(10):1098-104.
12. Foulks GN, Borchman D, Yappert M, et al. Topical azithromycin therapy for meibomian gland dysfunction: clinical response and lipid alterations. Cornea. 2010 Jul;29(7):781-8.
13. Sobolewska B, Doycheva D, Deuter C, et al. Treatment of ocular rosacea with once-daily low-dose doxycycline. Cornea. 2014 Mar;33(3):257-60.
14. Qiao J, Yan X. Emerging treatment options for meibomian gland dysfunction. Clin Ophthalmol. 2013;7:1797-803.
15. Foulks GN, Borchman D, Yappert M, Kakar S. Topical azithromycin and oral doxycycline therapy of meibomian gland dysfunction: a comparative clinical and spectroscopic pilot study. Cornea. 2013 Jan;32(1):44-53.
16. Alves M, Fonseca EC, Alves MF, et al. Dry eye disease treatment: a systematic review of published trials and a critical appraisal of therapeutic strategies. Ocul Surf. 2013 Jul;11(3):181-92.
17. Lacrisert [package insert]. Lawrenceville, NJ: Aton Pharma, Inc; 2007.
18. Moscovici BK, Holzchuh R, Chiacchio BB, et al. Clinical treatment of dry eye using 0.03% tacrolimus eye drops. Cornea. 2012 Aug;31(8):945-9.
19. Truong S, Cole N, Stapleton F, Golebiowski B. Sex hormones and the dry eye. Clin Exp Optom. 2014 Jul;97(4):324-36.
20. Gagliano C, Caruso S, Napolitano G, et al. Low levels of 17- -oestradiol, oestrone and testosterone correlate with severe evaporative dysfunctional tear syndrome in postmenopausal women: a case-control study. Br J Ophthalmol. 2014 Mar;98(3):371-6.
21. Hutnik C, Probst L. Argon laser punctal therapy versus thermal cautery for the treatment of aqueous deficiency dry eye syndrome. Can J Ophthalmol. 1998 Dec;33(7):365-72.
22. Law RW, Li RT, Lam DS, Lai JS. Efficacy of pressure topical anaesthesia in punctal occlusion by diathermy. Br J Ophthalmol. 2005 Nov;89(11):1449-52.
23. Korb DR, Blackie CA. Debridement-scaling: a new procedure that increases Meibomian gland function and reduces dry eye symptoms. Cornea. 2013 Dec;32(12):1554-7.
24. Maskin SL. Intraductal meibomian gland probing relieves symptoms of obstructive meibomian gland dysfunction. Cornea. 2010 Oct;29(10):1145-52.
25. Wladis EJ. Intraductal meibomian gland probing in the management of ocular rosacea. Ophthal Plast Reconstr Surg. 2012 Nov-Dec;28(6):416-8.
26. Friedland BR, Fleming CP, Blackie CA, Korb DR. A novel thermodynamic treatment for meibomian gland dysfunction. Curr Eye Res. 2011 Feb;36(2):79-87.
27. Tsubota K, Goto E, Fujita H, et al. Treatment of dry eye by autologous serum application in Sjögren's syndrome. Br J Ophthalmol. 1999 Apr;83(4):390-5.
28. Celebi ARC, Ulusoy C, Mirza GE. The efficacy of autologous serum eye drops for severe dry eye syndrome: a randomized double-blind crossover study. Graefes Arch Clin Exp Ophthalmol. 2014 Apr;252(4):619-26
29. Harritshøj LH, Nielsen C, Ullum H, et al. Ready-made allogeneic ABO-specific serum eye drops: production from regular male blood donors, clinical routine, safety and efficacy. Acta Ophthalmol. 2014 Mar 16. [Epub ahead of print]
30. Pachigolla G, Prasher P. Evaluation of the role of ProKera in the management of ocular surface and orbital disorders. Eye Contact Lens. 2009 Jul;35(4):172-5.
31. Meller D, Tseng S. Conjunctivochalasis: Literature review and possible pathophysiology. Surv Ophthalmol. 1998 Nov-Dec;43(3):225-32.
32. Huang Y, Sheha H, Tseng SC. Conjunctivochalasis interferes with tear flow from fornix to tear meniscus. Ophthalmology. 2013 Aug;120(8):1681-7.
33. The Loteprednol Etabonate US Uveitis Study Group. Controlled evaluation of loteprednol etabonate and prednisolone acetate in the treatment of acute anterior uveitis. Am J Ophthalmol. 1999 May;127(5):537-44.
34. Li M, Gong L, Sun X, Chapin WJ. Anxiety and depression in patients with dry eye syndrome. Curr Eye Res. 2011 Jan;36(1):1-7.



The company that brought VEP into the eye care practice now leads the way to office-based pattern ERG.

- The Diopsys® NOVA-ERG test helps doctors assess retinal function while VEP helps assess the entire visual pathway.
- Easy-to-use ERG Lid Sensors are comfortable for the patient and convenient for the doctor.
- Reimbursement for CPT Codes 95930 (VEP) & 92275 (ERG) can each exceed $100.*

To learn more, visit *www.PatternERG.com*

DIOPSYS® NOVA-ERG
OFFICE BASED PATTERN ELECTRORETINOGRAPHY TESTING

*Insurance coverage may vary by contract. Please contact Diopsys at 973-244-0622 for more detail on coverage.

2 CE Credits
(COPE approval pending)

15th Annual Dry Eye Report

Beneath the Surface: Dry Eye's Link to Systemic Disease

A thorough history and a team approach can help simplify the process of managing ocular surface compromise in these patients.

By Alexander J. Kabiri, OD, Devin Singh, OD, and Michael S. Cooper, OD

We have a tremendous opportunity to counsel our patients on how systemic conditions contribute to dry eye. Although we routinely explain the impact of systemic disease on retinal health, its role is sometimes overlooked when evaluating the ocular surface. The conundrum: To determine whether ocular surface compromise is a local or system-wide phenomenon. How do we best apply our knowledge of these conditions to a constantly changing health care environment?

This article illustrates how you can most effectively identify treatment strategies and develop a team-based approach with other medical specialties when managing ocular surface disease secondary to underlying systemic conditions.

Homeostasis and History

Changes in physical chemistry due to aging, gender, systemic disease and medication use inherently affect the ocular surface, as well as tear secretion and composition, in a multitude of ways. If not treated appropriately, dry eye disease (DED) can evolve—causing visual disturbances, ocular surface scarring and secondary bacterial infection.[1] Hormonal changes during pregnancy are believed to contribute to the inflammatory response in dry eye by decreasing androgen production and disrupting lacrimal gland function.[2]

Release Date: September 2014
Expiration Date: September 1, 2017
Goal Statement: With a thorough history and examination, a targeted treatment strategy can be instituted to prevent further damage from concurrent systemic disease and dry eye. To most effectively prevent severe ocular surface compromise, a rudimentary knowledge of dry-eye inducing disease states—and the ocular side effects of the drugs commonly used to manage them—is essential.
Faculty/Editorial Board: Alexander J. Kabiri, OD, Devin Singh, OD, and Michael S. Cooper, OD
Credit Statement: COPE approval for 2 hours of CE credit is pending for this course. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.
Joint-Sponsorship Statement: This continuing education course is joint-sponsored by the Pennsylvania College of Optometry.
Disclosure Statement: Dr. Kabiri is a consultant to and speaker for Allergan. Dr. Singh has no relationships to disclose. Dr. Cooper is a consultant to Allergan, TearLab and BioTissue.

Any good investigation begins with a thorough patient history. It is of utmost importance to elicit details of symptomatic involvement, such as ocular discomfort, grittiness, itching, redness, vision fluctuation and excessive tearing.[1] In addition, the history should include questions about possible underlying systemic etiologies of dry eye.

Although many systemic diseases are associated with dry eye, the conditions seen most frequently by eye care providers include diabetes, thyroid imbalance, hypertension, lupus, rheumatoid arthritis (both adult and juvenile) and Sjögren's syndrome.[3,4] If you determine that one or more of the aforementioned conditions is precipitating DED in a patient, it is your responsibility to help address and comanage the underlying problem to prevent persistent flare ups.

Finally, cases of DED that do not respond to conventional treatment (either in the adult or pediatric population) usually have a systemic component that warrants a complete work-up.[3] Given the long list of potential differential diagnoses, consider comanagement with other specialists to gain a broader scope of the situation when faced with such multifarious presentations. It is certainly appropriate and advisable to reach out if you believe the expertise of a subspecialist is nescessary.

Hormones and Aging

Hormonal factors, such as androgens, glucagon and adenocorticotropic hormone (ACTH), all affect lacrimal secretion. The posterior layer of the tear film contains the anchoring glycoprotein (known as mucin) secreted by the goblet cells of the conjunctiva. Goblet cells lack innervation and, in other parts of body, respond to hormonal stimulation by prostaglandins, serotonin and secretin, which are believed to be under humoral control in the bulbar conjunctiva. In women, this may indicate an association of DED with post-menopausal status, hormone replacement therapy or contraceptive use.[5]

Photo: Milton M. Hom, OD

Lid debris observed in a type 2 diabetes patient who was diagnosed with evaporative dry eye and blepharitis.

Levels of the major tear proteins are known to decline with age. At the same time, the volume of tears also tends to deteriorate, while the concentration of added serum proteins remains constant or increases.[6,7] Several constituents of the tear film function to prevent microbial and oxidative damage to the ocular surface:

- *Lysozyme*, a plasma cell comprising one third of all proteins, works in conjunction with immunoglobulins G and A to target microbes.
- *Lactoferrin* modulates polymorphonuclear leukocytes that, in turn, mediate lysozyme activity.
- *Ceruloplasmin*, a copper-binding serum protein enzyme that acts as a free radical scavenger.

It is well documented that as ceruloplasmin levels increase in the tear film, lysozyme and lactoferrin measurements proportionately decrease.[6,7] This solute imbalance suggests loss of acinar tissue from either the lacrimal or accessory glands with increased age. The effective result is an age-related decline in aqueous tear production and subsequent increase in dry eye prevalence.[6]

Diabetes Mellitus and the Eye

In diabetes, glucose levels can rise dangerously high in the bloodstream.[8] As a brief review, the pancreas functions as both an exocrine and endocrine gland. Most importantly, however, the pancreas produces insulin—a peptide hormone essential for the conversion of glucose into energy at the cellular level.

Normally, insulin is regulated to allow cellular absorption of sugar molecules; when there is a malfunction, it may be produced in insufficient amounts or used in an inefficient manner, leaving cells in an energy-depleted state. As time progresses, consistently uncontrolled and elevated blood glucose levels may damage any—and all—organ systems and nerves.

A number of factors predispose diabetes patients to dry eye. One example would be microvascular damage secondary to hyperglycemia, resulting in peripheral neuropathy.[8] Tear secretion is controlled via a

feedback loop involving the corneal nerves and lacrimal gland; this mechanism can become dysfunctional in diabetes patients. When corneal innervation is disrupted due to neuropathy, tear output from the lacrimal gland is reduced. Some studies suggest involvement of the enzyme aldose reductase in the sorbitol pathway and oxidative stress-induced changes in the lacrimal gland and corneal epithelium.[8]

These patients suffer from a variety of corneal complications, including superficial punctate keratopathy, trophic ulceration, persistent epithelial defects and chronic DED. Additionally, patients with increased glycosylated hemoglobin (HbA_{1c}) levels and a longer duration of diabetes are more likely to experience a high incidence of dry eye.[8]

The roles of aqueous deficiency and tear film evaporation can be observed when there is a measurable decrease in both tear secretion and reduced tear film break-up time. Subsequently, vascular abnormalities and a heightened inflammatory response can negatively affect lacrimal gland output and goblet cell density.

Dry Eye's Relationship with Diabetes

Dry eye disease typically is categorized as either evaporative or aqueous deficient. The evaporative form comprises approximately 65% to 86% of all DED cases in the United States.[9,10] This particular variant occurs when the lipid layer of the tear film disperses in a rapid fashion secondary to inflammatory manifestation of meibomian gland dysfunction, which reduces lipid secretions.

Numerous studies illustrate the connection between these conditions; the most conclusive and collaborative effort thus far has been the International Workshop on Meibomian Gland Dysfunction.[11] The field of study connecting blepharitis and dry eye has become increasingly popular in the research community, which will help eye care providers to better serve their patients. Lid architecture and meibomian gland function is crucial to lipid layer application. Therefore, disruption of the lids due to chronic inflammation can impact evaporative DED. Literature supports the association of diabetes and chronic blepharitis (e.g., secondary to *Staphylococcus aureus* or *Demodex* mites).[12,13]

Although less common as a primary presentation of DED, the aqueous-deficient form is related to decreased tear volume as a result of lacrimal gland dysfunction. Insufficient aqueous secretion by the lacrimal glands can result in a concentrated (i.e., hyperosmolar) and unstable tear film, with desiccation of the ocular surface.[14,15] Subsequently, reduced aqueous production correlates with an elevated inflammatory response that shifts the focus to neuropathic autoimmune conditions, such as diabetes.

A link between uncontrolled diabetes and dry eye is evident in the literature.[8] Additionally, clinical experience suggests that increased fasting blood sugar (FBS) or HbA_{1c} level corresponds with both the severity of superficial keratitis and patient symptoms.

Efforts to better quantify and qualitatively measure these findings may aid the clinical decision-making process. A notable improvement is the advent of enzyme-directed tests, such as the InflammaDry (Rapid Pathogen Screening) for rapid, in-office use.[16] We believe that many other devices that measure the qualitative level of matrix metalloproteinase-9 will be coming to market in the near future, and will assist the eye care community with rapid specificity in correlating these disease states.

Dry Eye Management in Diabetes Patients

Proper management of diabetic eye disease starts with good glycemic control and clear communication with the patient. Additionally, comanagement with other physicians is essential. The inflammation and damage to the lacrimal gland and ocular surface stems from metabolic, neurotrophic and vascular compromise. Thus, our treatment options include nutritional guidance, anti-inflammatory therapy (topical corticosteroids and NSAIDs), immunomodulators (topical cyclosporine and tacrolimus) and tear supplementation (artificial tears). While lubrication, punctal occlusion and topical cyclosporine are mainstays of ocular therapy, omega-3 and omega-6 triglyceride supplementation also may be useful in these patients.[17]

Finally, it is important to note that aggressive topical corticosteroid use may cause a transient increase in blood glucose levels in patients with controlled diabetes mellitus.[18] Nevertheless, if deemed medically necessary, use of these agents must be considered in such patients.

Perhaps the most powerful tool for the eye care provider, however, is clear communication with the patient and comanaging physicians (e.g., endocrinologists). This empowers each individual to play his or her role correctly for the ultimate benefit of the patient. An excellent example is when an endocrinologist or primary care physician prescribes a new medication that may induce or modulate DED. Once noted, you should contact the respective care provider and inform them of these new signs and symptoms.

Hypertension

In the United States alone, 67 to 75 million adults have been diagnosed with hypertension. It is a

disease with few symptoms early in its course that has fatal consequences, particularly when left untreated. The primary and contributing factors for elevated blood pressure are heart attack, heart failure, stroke, kidney failure and peripheral vascular disease.[19]

The connection between aqueous deficient dry eye and systemic hypertension involves the therapeutic agents used rather than the disease process itself. The classes of antihypertensive agents that contribute to DED include diuretics and beta blockers.[20] It is well established that diuretics (e.g., hydrochlorothiazide) exert a decrease in lacrimation that affects the dynamic solute chemistry of the tear film.[21,22] In addition, beta blockers, such as atenolol, reduce lysozyme levels and immunoglobulin A, causing low aqueous production and subsequently leading to symptoms of dry eye.[23] Patients using beta blockers also exhibit corneal hypoesthesia, decreased tear film break-up time and ocular irritation.

As with any medication side effect, it is prudent to alert both the patient's primary care doctor and cardiologist. By communicating appropriately within the patient's health care team, we can foster greater awareness of the ocular complications of hypertensive treatment. We all want to believe our colleagues in other medical specialties know the many ways that drugs interact with the eye; however, we must be realistic and understand this assumption is flawed.

With new research on therapeutic agents arriving daily, it is our ability to recognize these ocular consequences that will better serve patients and inform the medical community of our continually burgeoning skill set.

There are challenges to the therapeutic management of DED in hypertensives who also experience comorbidities such as diabetes, thyroid disease and anxiety/depression. It is imperative to tease out these linkages in the history and tailor the treatment plans accordingly. Early detection of dry eye disease and appropriate management to prevent progression are essential when you suspect medication(s) may be contributing to the patient's dry eye.

Lupus

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that exhibits a wide range of symptoms due to its impact on virtually every organ system; dry eye disease is its most common ocular manifestation.[24] More than 90% of cases occur in women of childbearing age.

The common triad of symptoms includes fever, joint pain and rash. Autoantibodies produced in the bloodstream of lupus patients can affect the skin, heart, lungs, kidneys, joints and/or nervous system. The incidence is higher in females, and androgen hormones (e.g., estrogen) may play a role in the expression of SLE. Furthermore, researchers have proposed that there are myriad potential etiologies—including genetics, viruses, drug induction and ultraviolet light exposure.[24] Blood testing and tissue biopsy may support the diagnosis.

The goals of primary systemic therapy are centered on alleviating symptoms and protecting the organs by controlling inflammation. Oral NSAIDs and corticosteroids are potent mediators of inflammation, but carry their own risks, including elevated blood sugar levels and steroid-induced glaucoma with prolonged use.

Plaquenil, an anti-malarial agent, prevents flare-ups of lupus with consistent use. Toxicity rarely occurs at doses lower than 6.5mg/kg/day; when complications have occurred, they typically manifest after at least five years of drug exposure.[24]

As optometrists, it is within our purview to assist rheumatology by performing a yearly spectral-domain optical coherence tomography (SD-OCT) scan.[25] Technological advances such as SD-OCT assist in providing improved patient care by early detection of subclinical retinal pathology, such as subtle depigmentation of the retinal pigment epithelium.[26]

Shifting focus to the anterior segment, proposed pathophysiological mechanisms include formation of autoimmune antibodies and immune complex deposition in various ocular tissues. The deposition of these immune complexes in the conjunctiva, cornea and lacrimal gland reduces aqueous tear production and inhibits mucin layer adhesion.[27] These mechanisms are central to lacrimal gland involvement due to antibody dependent cytotoxicity, pathogenic circulating antibodies and anti-neuronal antibodies, which may result in secondary Sjögren's syndrome with resultant aqueous deficient dry eye disease due to decreased tear secretion.[24] In addition, vasculitis and uveitis may impact the viability of the conjunctival space.

It is paramount to control the inflammatory response via therapeutic agents, aggressive lubrication and punctal plugs in order to protect the normal homeostatic balance of the ocular surface and surrounding lacrimal tissues.

Of note, SLE is highly chronic and can be elusive to diagnose at times, which can make the ocular treatment more difficult to manage in a timely manner. Allowing ample time to explain that customized (i.e., compounded) therapeutic drugs may be indicated depending on the severity of the condition is vital

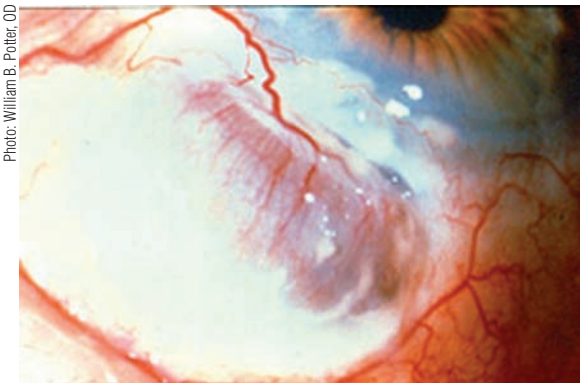


Photo: William B. Potter, OD

This rheumatoid arthritis patient presented with scleritis.

in this particular disease segment. Consequently, patient counseling and follow-up care are crucial to the success of the treatment strategy.

Adult Rheumatoid and Juvenile Idiopathic Arthritis

The pathogenesis of adult rheumatoid arthritis (RA) is not completely understood. Currently, there are several theories proposing intrinsic triggers based on the immunology of the rheumatoid factor (RF) and genetic epitopes of human leukocyte antigen clusters (HLA-DR4/DR1) present in up to 90% of patients with RA.[28-32] The chief consequence is symmetrical pattern destruction of the synovial joint tissue inclusive of the extremities, with collateral organ system damage.[29,30] Extra-articular involvement of organs such as the skin, heart, lungs and eyes is significant and present in 10% to 20% of patients, but more frequent in seropositive patients.[29]

The ocular sequelae of RA include chronic iridocyclitis, scleritis and secondary Sjögren's syndrome; however, the initial manifestation typically is aqueous deficient dry eye.[33-35] The incidence of DED associated with rheumatoid arthritis in the literature ranges from 11.6% to 50%.

One study indicated that it is imperative to understand that lymphocytic infiltration of the lacrimal and salivary glands alters the ocular surface environment, disrupting the natural flora, which permits a marked increase in antibiotic resistant organisms.[36] Consequently, severe ocular dryness can lead to significant corneal scarring, visual compromise or even blindness.

Juvenile idiopathic arthritis (JIA) differs somewhat in presentation from adult RA, but is the most common rheumatological disease in the pediatric population under age 16, with more 294,000 children affected in the United States alone.[37,38] Although it is a heterogeneous group of diseases with a poorly understood immunoinflammatory pathogenesis, multiple genetic traits, including HLA-B27, RF and antinuclear antibodies (ANA), are implicated in its systemic manifestation. It is characterized by chronic inflammation of the synovium secondary to lymphocytic infiltration of the musculoskeletal system; the first symptom of JIA typically is limping.[39]

Due to the robust inflammatory response observed in JIA, dry eye disease is a smaller concern compared to the chronic uveitis found in this patient population. Persistent anterior nongranulomatous uveitis occurs in 30% of patients with different variants of JIA.[40] The condition is most common in girls with ANA-positive JIA. Patients are usually asymptomatic, but may exhibit conjunctivitis, anisocoria and chronic, intermittent eye pain.

Sjögren's Syndrome

This is a complex, chronic inflammatory disease that occurs secondary to lymphocytic infiltration of exocrine organs. Similar to RA, Sjögren's syndrome demonstrates an association with HLA and possible infectious triggers. Most individuals exhibit xerostomia and parotid gland enlargement in addition to aqueous deficient dry eye; however, establishing the diagnosis can take years to decades. In an extensive literature review, its etiology has been linked to other rheumatic disorders, such as SLE and RA, which further complicate the diagnostic process. The classic ocular sign in this condition is chronic infiltration of the lacrimal gland, resulting in diminished aqueous tear production.[41]

According to various comparative studies, Sjögren's syndrome appears to have no ethnic predilection. It is distributed homogenously worldwide affecting 1 to 2 million Americans, with a female-to-male ratio of 9:1. Sjögren's affects patients of any age, but is most common in elderly individuals. More specifically, onset typically occurs in the fourth to fifth decades of life.[42]

With more than 3 million undiagnosed dry eye patients, a new tool may soon become more widely accepted than the conventional gold standard of invasive salivary gland biopsy.[43,44] Recently, a new in-office test called Sjö (Nicox) that uses three unique biomarkers has demonstrated high specificity in the early detection of Sjögren's syndrome in dry eye patients.[45] The traditional biomarkers were antibodies RF and ANA, which are specific but present a low sensitivity. Subsequently, this particular metric uses additional indicators, such as salivary protein-1, carbonic anhydrase-6 and parotid secretory protein, which is related to the symptomatology of dry mouth and parotid involvement.[45]

Dry Eye Management in RA, JIA and Sjögren's Patients

Our top priority concerning autoimmune diseases is prevention and early diagnosis.[46] The eye care community is uniquely positioned to contribute, considering we see a significant number of patients who present with "gateway" signs and symptoms.

Consequently, it is crucial to closely comanage with rheumatology, endocrinology and dentistry to control underlying systemic inflammation and ensure better quality of life. A rapid diagnosis can potentially thwart lacrimal and salivary gland destruction, which can lead to severe dry eye disease, depression, mobility issues in RA and JIA, and risk of non-Hodgkin's lymphoma—specifically found in 5% of Sjögren's patients.[45-49]

With regard to each of these inflammatory disease states, chronic treatment is required to stabilize both ocular symptoms and signs. Mainstay therapies include immunomodulators, oral and topical corticosteroids, aggressive tear supplementation and punctal plugs to manage the ocular sequelae.

In addition, certain disease-modifying antirheumatic drugs (e.g., methotrexate and hydroxychloroquine) can be beneficial in managing inflammation that's not limited to uveitis; however, care should be taken with constant monitoring and communication with a rheumatologist due to the increased potential for ocular side effects, such as macular toxicity.[49]

With a thorough history and examination, an appropriately targeted treatment strategy can be instituted to prevent further damage from concurrent systemic disease and dry eye. Such management likely will prove to be more successful than a generalized "kitchen sink" approach to dry eye treatment. However, to most effectively prevent severe ocular surface compromise in these individuals, a rudimentary knowledge of dry-eye inducing disease states—and the ocular side effects of the medications commonly used to manage them—is essential. ■

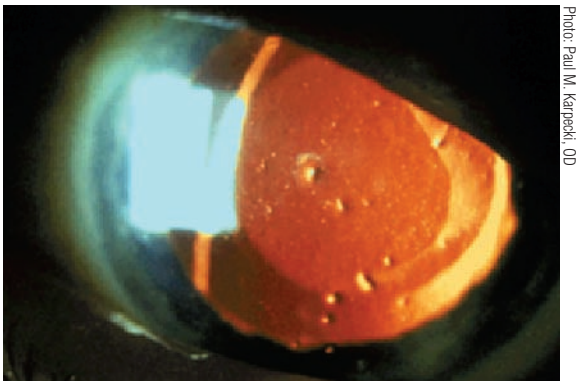


Photo: Paul M. Karpecki, OD

This patient with Sjögren's syndrome exhibited filamentary keratitis and dry eye disease.

Drs. Kabiri and Singh practice at Malik Eye Care, a multi-location MD/OD group setting in Queens, New York.

Dr. Cooper practices at an MD/OD group setting in Willimantic, Conn.

1. The definition and classification of dry eye disease: report of the Definition and Classification Subcommittee of the International Dry Eye Workshop (2007). Ocul Surf. Apr 2007;5(2):75-92.
2. Cheung A, Scott IU. Ocular changes during pregnancy. Available at http://aao.org/publications/eyenet/201205/pearls.cfm. Accessed May 20, 2014.
3. Karpecki P, Shechtman D. Systemic disease and dry eye. Available at www.revoptom.com/content/c/22531. Accessed May 20, 2014.
4. McCulley J. Systemic and autoimmune diseases that lead to dry eye. Available at: www.ophthalmologymanagement.com/articleviewer.aspx?articleID=105864. Accessed May 20, 2014.
5. Schaumberg DA, Buring JE, Sullivan DA, et al. Hormone replacement therapy and dry eye syndrome. JAMA. 2001 Nov 7;286(17):2114-9.
6. Tiffany JM. Tears in health and disease. Eye (Lond). 2003 Nov;17(8):923-6.
7. Seal DV, McGill JI, Mackie IA, et al. Bacteriology and tear protein profiles of the dry eye. Br J Ophthalmol. 1986 Feb;70(2):122-5.
8. Manaviat MR, Rashidi M, Ardekani MA. Prevalence of dry eye syndrome and diabetic retinopathy. BMC Ophthalmol. 2008 Jun 2;8:10.
9. Shimazaki J, Sakata M, Tsuboto K. Ocular surface changes and discomfort in patients with meibomian gland dysfunction. Arch Ophthalmol. 1995 Oct;113(10):1266-70.
10. Lemp MA, Crews LA, Bron AJ, et al. Distribution of aqueous deficient and evaporative dry eye in a clinic-based patient population. Cornea. 2012 May;31(5):472-8.
11. Nichols KK, Foulks GN, Bron AJ, et al. The international workshop on meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2011 Mar 30;52(4):1922-9.
12. Clifford CW, Fulk GW. Association of diabetes, lash loss, and *Staphylococcus aureus* with infestation of eyelids by *Demodex folliculorum* (Acari: Demodicidae). J Med Entomol. 1990 Jul;27(4):467-70.
13. Yamashita LS1, Cariello AJ, Geha NM, et al. *Demodex folliculorum* on the eyelash follicle of diabetic patients. Arq Bras Oftalmol. 2011 Nov-Dec;74(6):422-4.
14. Khanal S, Tomlinson A, Diaper CJ. Tear physiology of aqueous deficiency and evaporative dry eye. Optom Vis Sci. 2009 Nov;86(11):1235-40.
15. Bron AJ, Yokoi N, Gafney E, Tiffany JM. Predicted phenotypes of dry eye: Proposed consequences of its natural history. Ocul Surf. 2009 Apr;7(2):78-92.
16. Cooper MS. The Phoenician connection: translating the emerging ocular surface disease diagnostics. Advanced Ocular Care. Pending publication May/June 2014.
17. American Optometric Association. Essential Fatty Acids. Available at: www.aoa.org/patients-and-public/caring-for-your-vision/essential-fatty-acids?sso=y. Accessed May 20, 2014.
18. Kymionis GD, Panagiotoglou T, Tsilimbaris MK. The effect of intense, short-term topical dexamethasone disodium phosphate eyedrops on blood glucose level in diabetic patients. Ophthalmologica. 2007;221(6):426-9.
19. CDC. Vital signs: awareness and treatment of uncontrolled hypertension among adults—United States, 2003-2010. MMWR. 2012;61(35):703-9.
20. Drugs for Hypertension. Treatment guidelines from the medical letter. Treat Guidel Med Lett. 2012 Jan;10(113):1-10.
21. Bartlett JD. Ophthalmic toxicity by systemic drugs. GC Chiou (ed.). Ophthalmic Toxicology, 2nd ed. Michigan: Taylor and Francis; 1999:225-83.
22. Jaanus SD, Bartlett JD, Hiett, JA. Ocular effects of systemic drugs. Bartlett JD & Jaanus SD (eds.). Clinical Ocular Pharmacology, 3rd ed. Boston: Butterworth-Heinemann; 1995:957-1006.
23. Muchnick BG. Identify the ocular side effects of systemic medications. Rev Optom. 2008 Jan;145(1):60-73.
24. Sivaraj RR, Durrani OM, Denniston AK, et al. Ocular manifestations of systemic lupus erythematosus. Rheumatology (Oxford). 2007 Dec;46(12):1757-62.
25. Chen E, Brown DM, Benz MS, et al. Spectral domain optical coherence tomography as an effective screening test for hydroxychloroquine retinopathy (the "flying saucer" sign). Clin Ophthalmol. 2010 Oct 5;4:1073-9.
26. Tehrani R, Ostrowski RA, Hariman R, et al. Ocular toxicity of hydroxychloroquine. Semin Ophthalmol. 2008,23(3):201-9.
27. Medscape. Ophthalmologic Manifestations of Sjogren Syndrome. Available at http://emedicine.medscape.com/article/1192919-overview. Accessed May 20, 2014.
28. Sahatçiu-Meka V, Rexhepi S, Manxhuka-Kërliu S, et al. Extraarticular manifestation of seronegative and seropositive rheumatoid arthritis. Bosn J Basic Med Sci. 2010 Feb;10(1):26-31.
29. Lilleby V, Gran JT. Systemic rheumatoid arthritis: Tidsskr Nor Laegeforen. 1997 Nov 30;117(29):4223-5
30. Sobrin L, Kim EC, Christen W, et al. Infliximab therapy for the treatment of refractory ocular inflammatory disease. Arch Ophthalmol. 2007 Jul; 125(7):895-900.
31. Sainz de la Maza M, Foster CS, Jabbur NS. Scleritis associated with rheumatoid arthritis and with other systemic immune-mediated diseases. Ophthalmol. 1994 Jul;101(7):1281-86.
32. Zlatanovi G, Veselinovi D, Ceki S, et al. Ocular manifestation of rheumatoid arthritis-different forms and frequency. Bosn J Basic Med Sci. 2010 Nov;10(4):323-7.
33. Medscape. Rheumatoid Arthritis. Available at http://emedicine.medscape.com/article/331715-overview. Accessed May 20, 2014.
34. Cimmino MA, Salvarani C, Macchioni P, et al. Extra-articular manifestations in YZ Italian patients with rheumatoid arthritis. Rheumatol Int. 2000;19(6):213-7.
35. Kanski JJ. The Eye in Systemic Disease. London: Butterworths; 1986.

36. Hori Y. Bacteriologic profile of the conjunctiva in the patients with dry eye. Am J Ophthalmol. 2008 May;146(5):729-34.
37. US National Library of Medicine. Juvenile rheumatoid arthritis: MedlinePlus Medical Encyclopedia. US National Library of Medicine. Available at: www.nlm.nih.gov/medlineplus/ency/article/000451.htm. Accessed May 20, 2014.
38. Helmick CG, Felson DT, Lawrence RC, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the united states. Arthritis Rheum. 2008 Jan;58(1):15-25.
39. Cassidy JT, Petty RE. Chronic arthritis in childhood. Cassidy JT, Petty RE, Laxer RM, Lindsley CB (eds.). Textbook of Pediatric Rheumatology, 5th ed. Philadelphia: Elsevier Saunders; 2005:206-60.
40. Weiss JE, Ilowite NT. Juvenile idiopathic arthritis. Rheum Dis Clin North Am. 2007 Aug;33(3):441-70, vi.
41. Medscape. Sjogren's Syndrome. Available at http://emedicine.medscape.com/article/332125-overview. Accessed May 20, 2014.
42. Miller A. Medscape. Sjogrens Syndrome. Available at: http://emedicine.medscape.com/article/332125-overview. Accessed August 22, 2014.
43. Sjögren's Syndrome Foundation. 2001. Available at: www.sjogrens.org. Accessed May 25, 2014.
44. Kassan SS, Moutsopoulos HM. Clinical manifestations and early diagnosis of sjögren's syndrome. Arch Intern Med. 2004 Jun 28;164(12):1275-84.
45. Shen L, Suresh L, Lindemann M, et al. Novel autoantibodies in sjögren's syndrome. Clin Immunol. 2012 Dec;145(3):251-5.
46. 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 Dec;96(12):1498-503.
47. Fox RI. Sjögren's syndrome. Lancet. 2005 Jul 23-29;366(9482):321-31.
48. Shen L. Different stages of primary Sjögren's syndrome involving lymphotoxin and type 1 IFN. J Immunol. 2013;191:608-13.
49. Payne JF, Hubbard GB 3rd, Aaberg TM Sr, et al. Clinical characteristics of hydroxychloroquinine retinopathy. Br J Ophthalmol. 2011 Feb;95(2):245-50.

OSC QUIZ

You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form *(page 81)*, and return it with the $35 fee to: Optometric CE, P.O. Box 488, Canal Street Station, New York, NY 10013. To be eligible, please return the card within one year of publication.

You can also access the test form and submit your answers and payment via credit card at *Review of Optometry* online, www.revoptom.com.

You must achieve a score of 70 or higher to receive credit. Allow eight to 10 weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of transcript-quality credit from Pennsylvania College of Optometry and double credit toward the AOA Optometric Recognition Award—Category 1.

Please check with your state licensing board to see if this approval counts toward your CE requirement for relicensure.

1. What is a risk factor for dry eye disease (DED)?
a. Age.
b. Female gender.
c. Systemic disease.
d. All of the above.

2. With regard to patient history and DED risk, which statement is true?
a. Symptoms are the only aspect to consider.
b. Taking a history may uncover systemic etiologies of dry eye.
c. Dry eye that does not respond to conventional therapy doesn't warrant a systemic work up.
d. Visual complaints are never related to dry eye.

3. Lacrimal secretion is influenced by:
a. Hormonal factors.
b. The autonomic nervous system.
c. Tear evaporation.
d. All of the above.

4. Conjunctival goblet cell function is believed to be regulated by:
a. Parasympathetic and sympathetic stimulation.
b. Hormonal stimulation, as in other tissues of the body.
c. Both a and b.
d. None of the above.

5. As a patient begins to age, which tear film constituent increases in concentration, suggesting a rise in oxidative stress playing a role in ocular surface disease?
a. Lysozyme.
b. Lactoferrin.
c. Ceruloplasmin.
d. None of the above.

6. Lysozyme's function is to:
a. Work in conjunction with immunoglobulin G and A to target microbes.
b. Help microbes use iron in the tear film to their benefit.
c. Increase levels with age.
d. Function as a copper binding serum protein.

7. Diabetes patients:
a. Are not at risk for dry eye.
b. May experience neuropathy by disrupting corneal innervation and the tear feedback loop.
c. Suffer macrovascular damage due to hypoglycemia.
d. Have increased output from the lacrimal gland due to neuropathy.

8. Corneal complications experienced by diabetes patients with dry eye include:
a. Guttata.
b. Endothelial pigment.
c. Superficial punctate keratitis, trophic ulceration and persistent epithelial defects.
d. Pinguecula(e).

9. With regard to diabetes, which statement is FALSE?
a. Neuropathy associated with diabetes is proposed to play a role in reduced lacrimal gland output.
b. There is no correlation between severity or duration of diabetes and DED.
c. Good glycemic control is the primary goal in managing dry eye related to diabetes.
d. Nutritional guidance for the diabetes patient is complementary to topical therapy of dry eye.

10. Which statement is true regarding evaporative dry eye?
a. It is commonly associated with autoimmune conditions.
b. It accounts for 65% to 86% of dry eye cases in the United States.
c. It describes dry eye from reduced lacrimal gland secretion.
d. It accounts for a minority of dry eye cases in the United States.

11. Which statement is true with regard to encountering aqueous-deficient dry eye?
a. Consider the presence of an elevated inflammatory response and an underlying neuropathic autoimmune condition.
b. It is the most prevalent form of dry eye.
c. It has no inflammatory component.
d. Systemic disease is not likely to be involved.

12. Which statement regarding hypertension is true?
a. If left untreated, hypertension causes dry eye.
b. An improvement is seen in dry eye once antihypertensive agents (AHAs) are started.
c. Diuretics and beta blockers are common AHAs that contribute to dry eye.
d. Blood pressure measurement is a risk factor for dry eye.

13. Which statement regarding hypertension is FALSE?
a. Uncontrolled blood pressure is not directly associated with dry eye severity.
b. Beta blockers do not cause dry eye by elevating lysozyme and IgA levels in the tear film.
c. Diuretics and beta blockers are among the classes of antihypertensive medications associated with dry eye.
d. Unlike diabetes, hypertension is associated with nephropathy.

14. Management of dry eye in a patient with systemic hypertension may include:
a. Topical lubricants.
b. Topical anti-inflammatory therapy.
c. Punctal plugs.
d. All of the above.

OSC QUIZ

15. An elderly male states that he takes medication, but can neither recall the name of the drug nor the condition for which he is currently undergoing treatment. An UNLIKELY disease state in this scenario is:
a. Lupus.
b. Diabetes.
c. Hypertension.
d. All of the above are equally likely given his age and gender.

16. Fever, joint pain and rash are a triad of symptoms associated with:
a. Lupus.
b. Sjögren's syndrome.
c. Adult rheumatoid arthritis.
d. Thyroid dysfunction.

17. What is the initial ophthalmological manifestation of adult rheumatoid arthritis?
a. Scleritis.
b. Aqueous-deficient dry eye.
c. Evaporative dry eye.
d. Chronic iridocyclitis.

18. Which statement regarding juvenile rheumatoid arthritis (JIA) is FALSE?
a. It is the most common rheumatological disease in patients less than 16 years of age.
b. It has a similar pathophysiology to that of adult rheumatoid arthritis.
c. Multiple genetic traits—including HLA-B27, RF and antinuclear antibodies—are associated with JIA.
d. It is characterized by infiltration of synovial fluid.

19. Sjögren's syndrome is:
a. A clear, straightforward diagnosis.
b. A complex inflammatory disease state that can take years to diagnose.
c. Not linked to lupus or rheumatoid arthritis.
d. Both b and c.

20. Which test is appropriate to diagnose Sjögren's syndrome?
a. Invasive salivary biopsy.
b. Schirmer's test.
c. Sjö (Nicox).
d. Both a and c.

TAKE THE TEST ONLINE TODAY!
www.revoptom.com/continuing_education/

Examination Answer Sheet

Valid for credit through September 1, 2017

This exam can be taken online at www.revoptom.com/continuing_education. Upon passing the exam, you can view your results immediately and download a real-time CE certificate. You can also view your test history at any time from the website.

Beneath the Surface: Dry Eye's Link to Systemic Disease

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

Mail to: Jobson - Optometric CE, PO Box 488, Canal Street Station, New York, NY 10013

Payment: Remit $35 with this exam. Make check payable to Jobson Medical Information LLC.

COPE approval for 2 hours of CE credit is pending for this course.

This course is joint-sponsored by the Pennsylvania College of Optometry

There is an eight-to-ten week processing time for this exam.

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

1 = Excellent 2 = Very Good 3 = Good 4 = Fair 5 = Poor

Rate the effectiveness of how well the activity:

21. Met the goal statement: ① ② ③ ④ ⑤
22. Related to your practice needs: ① ② ③ ④ ⑤
23. Will help you improve patient care: ① ② ③ ④ ⑤
24. Avoided commercial bias/influence: ① ② ③ ④ ⑤
25. How would you rate the overall quality of the material presented? ① ② ③ ④ ⑤
26. Your knowledge of the subject was increased:
◯ Greatly ◯ Somewhat ◯ Little
27. The difficulty of the course was:
◯ Complex ◯ Appropriate ◯ Basic

How long did it take to complete this course?

Comments on this course:

Suggested topics for future CE articles:

Please retain a copy for your records. Please print clearly.

You must choose and complete one of the following three identifier types:

① SS # ___ - ___ - ____

Last 4 digits of your SS # and date of birth

② ____ · ____

State Code and License #: (Example: NY12345678)

③ ____

First Name

Last Name

E-Mail

The following is your: ☐ Home Address ☐ Business Address

Business Name

Address

City State

ZIP

Telephone # ___ - ___ - ____

Fax # ___ - ___ - ____

By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by any fraudulent or improper means.

Signature _____ Date _____

Lesson 110599

RO-OSC-0914

INTERNATIONAL VISION EXPO & CONFERENCE LAS VEGAS

VEW: Where CE is the Odds-On Favorite

The Las Vegas meeting's clinical offerings cover the full scope of eye care, from cornea to retina and everything in between. **By Jane Cole, Contributing Editor**

Today's changing health care climate requires optometrists to be on top of their game. They must be able to diagnose and treat myriad eye diseases, while keeping up with new research, technology and therapeutics, not to mention knowing about emerging federal regulations.

To that end, "Vision Expo West includes nearly 400 total hours of continuing education credits and 19 specialty tracks spanning the full scope of eye care—from clinical management of eye diseases and conditions, to practice management and profitability, to optical trends and technologies," says optometrist Mark Dunbar, co-chairman of VEW's conference advisory board.

Vision Expo West, held September 17 to 20 in Las Vegas, has a comprehensive lineup of 190 hours of clinically focused CE courses to help eye care practitioners stay ahead of the curve and bring their newfound knowledge back to their practices, Dr. Dunbar says.

"You ask it, we'll talk about it," says Mark Dunbar, OD, of the new, interactive, crowd-sourced learning courses at this year's Vision Expo West.

Have Your Say

The 2014 meeting features something new: crowd-sourced learning courses in which the audience directs the discussion. Attendees will connect to the speakers directly—via audience response system, text messaging and mobile polling—to share ideas and ask questions.

"You ask it, we'll talk about it," Dr. Dunbar says.

Eighteen hours of these interactive courses are offered, including "Discussions in Posterior Segment and Retinal Disease," presented by Dr. Dunbar and Steven Ferrucci, OD. This course focuses on posterior segment-related diseases such as macular degeneration, diabetes, peripheral retinal disease and pigmented lesions.

Another highlight in the crowd-sourced track is "Anterior Segment and Contact Lenses," presented by Louise Sclafani, OD, and Marc Bloomenstein, OD. In this course, the presenters share anterior

segment images and case histories with the audience. Attendees and presenters will jointly determine the diagnosis and craft the appropriate treatment plans, including alternative options.

Global Contact Lens Forum

Also new this year at VEW: the Global Contact Lens Forum, which kicks off the meeting with four hours of free education that combines the latest scientific content with critical business strategies related to contact lenses, Dr. Dunbar says.

Courses in this track include:

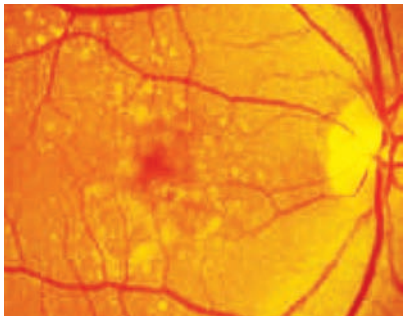
• "State of the Contact Lens Industry in 2014," which discusses GP contact lenses and if they are here to stay or passé, and whether the niche of specialty soft lenses and hybrids can continue to stand up to disposables.

• "Specialty Contact Lens Practice—How to Make it Happen and How to Succeed" guides attendees on how to set up a specialty lens practice, the latest specialty contact lens designs and technologies, and myopia progression management with contact lenses.

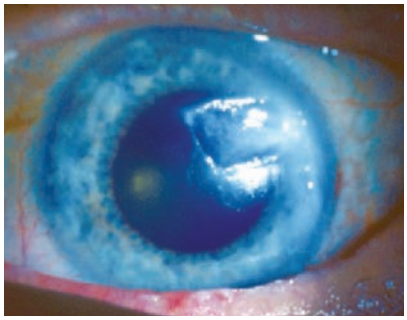
The 'Greatest' Courses

The "Greatest" series is also a highlight in this year's clinical lineup. Within this tremendous track, courses include:

• "The Greatest Anterior Segment Disease and Medical Management of Contact Lenses Course—Ever!" In this presentation, Jack Schaeffer, OD, Charlie Ficco, OD, and Dr. Bloomenstein discuss how new medical vision correction options also bring new complications. The trio offers management strategies for potential complications so attendees will feel comfortable diagnosing and treating even the most challenging cases.



Unique retinal cases and a wide range of topics will be covered in "The Greatest Posterior Segment Disease Course—Ever!"



Attendees will learn how to treat corneal conditions, like this persistent epithelial defect, in the "Amniotic Membrane Workshop," a hands-on wet lab course.

• "The Greatest Posterior Segment Disease Course—Ever!" by Drs. Dunbar, Ferrucci and Jerome Sherman, OD, will review unique cases and cover a wide array of retinal diseases.

More in Store

Another clinical course not to miss is the "Amniotic Membrane Workshop," which is being offered for the first time, Dr. Dunbar says. In this course, Doug Devries, OD, offers a two-hour session/wet lab designed to give attendees a hands-on experience in placing amniotic membranes. Indications for clinical use, proper coding and billing will also be discussed.

VEW rounds out its clinical education with more than 25 hours on glaucoma-related topics, 10 hours on pharmacology, macular degeneration and nutritional supplements, 11 hours devoted to systemic disease and neuro, and more than 60 hours on business solutions. Additionally, 38 hours will be focused on technologies, from the latest advances in fundus imaging and OCT to breakthroughs in cataract surgery and corneal crosslinking.

"There is truly something for every member of your practice at Vision Expo West," Dr. Dunbar says.

For more information or to register, go to www.visionexpowest.com. ■

CE: How to Provide Four-Star Service

For the first time at Vision Expo West, the Ritz-Carlton Leadership Center will share its service delivery expertise in a two-hour continuing education course, "Radar On—Antenna Up: The Ritz-Carlton Method of Fulfilling Unexpressed Wishes and Needs."

Dr. Dunbar explains: "We wanted to offer educational experiences that went beyond eye care but are valuable in our day-to-day lives. Who better to do that than Ritz-Carlton? Widely recognized as the gold standard in customer service, the Ritz-Carlton knows how to create an unforgettable experience."

How to Handle a Hyper Kid

When we encounter an inattentive child, we tend to think that a vision problem is affecting his attention. Here's what to do when it's not. **Edited by Paul C. Ajamian, OD**

Q A 6-year-old boy presented at the request of his teacher. He's been "acting up" in class and his teacher and his mother are wondering if he's distracted because he can't see the board. I performed a full exam and found the child sees 20/20 and has no amblyopia, convergence insufficiency, binocular vision or accommodation problems. Now what?

A "This is a very common scenario in the first few months of the school year—particularly when a child is moving from preschool to kindergarten or first-grade, when more is expected of children," says Andrea Gregory, MD, a pediatrician at Self Regional Healthcare in Greenwood, SC.

"Sensory deficits—both visual and auditory—are high on the differential for children with behavior concerns, and should be the first to be ruled out if concerns arise," Dr. Gregory says.

However, she adds, other possible causes include specific learning disabilities, language disorder, attention-deficit/hyperactivity disorder (ADHD), substance abuse (in older children/adolescents), family stressors, lack of sleep, thyroid disorder, adverse effects of medication, autism spectrum disorders and psychiatric disorders.

"It's important to know whether the concerning behaviors are occurring in more than one setting," Dr. Gregory says. "If those behaviors occur only at school, problems such as sensory deficit or learning disorders are higher on the differential. If they are only at home, family stressors and lack of adequate boundaries may be more likely."

However, she adds, "if the child is bouncing off the walls of your office and it's difficult to get through the exam—and this is the norm according to the parents—then ADHD should be strongly considered."

Photo: Juawana Hall, OD

Is this child hyperactive, vision impaired or just a "busy bee"? Take a closer look.

Epidemiologic studies of ADHD indicate that 3% to 8% of children in the US are affected, mostly boys.[1] There is no known cause of ADHD, but neuroimaging studies have shown differences in brain structure and function between people with ADHD and those without. Specifically, areas of the brain associated with executive function (problem solving and goal orientation) seem to be less active in patients with ADHD.[2]

ADHD is not a simple diagnosis to make, Dr. Gregory says. Several criteria are required:

- The child must exhibit six of nine symptoms of inattention (things like "often fails to give close attention to details or makes careless mistakes in schoolwork, work or other activities," "often does not seem to listen when spoken to directly" and "often has difficulty organizing tasks and activities").
- Or, the child must show six of nine symptoms of hyperactivity or impulsivity ("often fidgets with hands or feet or squirms in seat," "often interrupts or intrudes on others" and "often talks excessively").

A few simple questions can help determine if the child could benefit from further evaluation by his pediatrician (whether or not the child is found to have a visual deficit):

- How is the behavior at home or out in public?
- Does he seem easily distracted when trying to complete a task?
- Does he act as if he is "driven by a motor"?

"An emphatic referral to the child's primary physician, along with a note with your findings and concerns, can be invaluable," Dr. Gregory says.

She adds, "Many children fall behind academically if they are left undiagnosed and untreated, so it's important to start the evaluation and possible treatment as early as possible. This is especially true for older children and adolescents who may go years without seeing their physician—but may see you annually to get their eyes examined." ■

1. Willcutt EG. The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. Neurotherapeutics. 2012 Jul;9(3):490-9.
2. Sowell ER, Thompson PM, Welcome SE, et al. Cortical abnormalities in children and adolescents with attention-deficit hyperactivity disorder. Lancet. 2003 Nov 22;362(9397):1699-707.

A Giant Leap
for Optometry

Unveiling a
Revolutionary Product

9.18.2014

At an **Unprecedented** New Price

optovue

Vision Expo West
Booth MS5035

300-49763 Rev.A

Can a Donor Cornea be Diseased?

Put patients at ease. It's extremely rare for a corneal transplant to transmit disease.

Edited by Joseph P. Shovlin, OD

Q I have a patient who must undergo a full-thickness corneal transplant for advanced keratoconus. He asked about any risk or concern for transmitted disease, including prion disease. What precautions do eye banks take to assure there's no transmission with prion disease or other diseases that could be transmitted through transplantation (HIV, hepatitis B, etc.)?

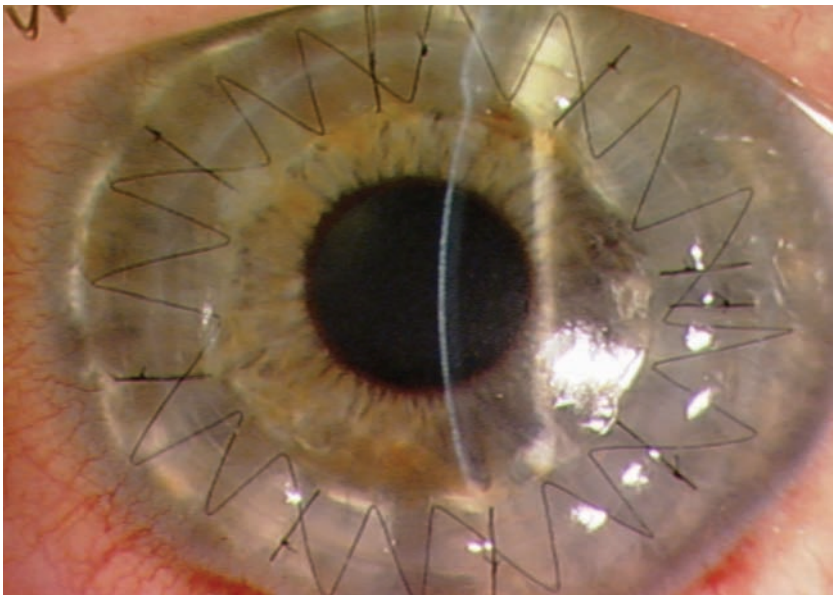
A "Eye banks have strict standards for evaluating the safety of donor corneas, so the risk for transmitted disease is extremely low," says Penny Asbell, MD, professor of ophthalmology and director of the Cornea Service at the Icahn School of Medicine at Mount Sinai in New York.

In over 50 years of eye banking—and more than 1 million successful transplantations—only three diseases have been reported as transmitted: Creutzfeldt-Jakob disease (three cases), hepatitis B (two cases) and rabies (one case).[1,2]

"Of the three cases reported of Creutzfeldt-Jakob disease (CJD)—a prion disease—transmitted by corneal transplantation, only one was confirmed by histopathologic examination of both subject and donor," says An Vo, MD, who also practices at Mount Sinai.

The medical standards of the Eye Bank Association of America (EBAA) exclude all donors with known CJD, as well as anyone with a family history of a blood relative with CJD.

"Since the imposition of medical standards in 1980, there have been no cases of CJD detected in the United States donor pool," Dr. Asbell says.[2]



Can a donor cornea transmit prion disease, such as Creutzfeldt-Jakob, to the recipient? Before eye bank standards were revised, it was rare. Now, the risk is even lower.

EBAA's medical standards also exclude those who have had human pituitary-derived growth hormone, cases of death of unknown cause, and cases of death with neurologic disease where the diagnosis is not established.

Additional safeguards were instituted by the Food and Drug Administration in 1993, with the establishment of the Interim Rule for Human Tissue Intended for Transplantation. This regulation requires screening of corneal donors and serologic testing for HIV and hepatitis B and C. This rule duplicated existing EBAA medical standards, which had already been in effect for several years, to screen and test for these three agents.

"Although no cases of syphilis transmitted through corneal transplantation have ever been reported, many eye banks still test for syphilis as part of donor evaluation, because syphilis screening is required for organ and other tissue donors" Dr. Vo says.

Dr. Asbell noted, "Corneal transplant surgery has transformed the lives of thousands of individuals. The transplanted tissue does carry a small risk of transmission of infection, but the risk is very small and the benefit immeasurable for most recipients." ■

1. Chu W. The past 25 years in eye banking. Cornea. 2000 Sep;19(5):754-65.
2. Hogan RN, Heck E, Cavanagh HD. Risk of prion disease transmission from ocular donor tissue transplantation. Cornea. 1999 Jan;18(1):2-11.

Hungry for success?

At Jobson, we have more effective ways for you to reach the optical market than anyone. So our approach to serving clients is unique. First, we develop a thorough understanding of your specific goals. This understanding, plus our extensive offering of products and services, enables us to then suggest solutions that will help achieve those goals. This often includes innovative ideas and premium positions. For advertising information contact Michele Barrett (610-492-1014, mbarrett@jobson.com) or Jim Henne (610-492-1017, jhenne@jobson.com). Let us satisfy your hunger for success.

REVIEW® OF OPTOMETRY | Review of Cornea & Contact Lenses | REVIEW® OF OPTOMETRY www.revoptom.com | REVIEW® of Ophthalmology www.revophth.com

Jobson Optical Group

The vision to help you succeed

Essentials of Hypertension

High blood pressure remains a major health issue worldwide, with profound—and often silent—multisystemic effects. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

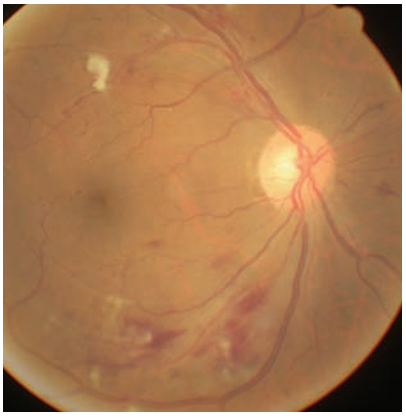
Systemic hypertension (HTN) is a common health problem—affecting more than 800 million people worldwide—that often remains asymptomatic until late in the disease course.[1] It is a major risk factor for both coronary artery disease and cerebrovascular accident.

Although HTN has both genetic and environmental factors, the exact mechanism in the majority of affected individuals is largely unknown. Cardiac hypertrophy, heart failure, aortic dissection and renal failure are all systemic sequelae of the disease process.

In the eye, hypertensive ocular changes can be the initial finding in a patient with undiagnosed HTN, and may have sight-threatening consequences. In this article, we'll review the ocular implications of hypertension.

Autoregulation in Retinal Vessels

Blood flow in the retina—as well as in the kidneys, brain, heart and skeletal muscle—is controlled by autoregulation, which is the intrinsic ability to maintain a constant blood flow despite changes in perfusion pressure. Thanks to autoregulation, retinal and other blood vessels constrict or dilate depending on hyper- or hypoperfusion status.[4,5]



A patient with significant hypertensive retinopathy shows cotton-wool spots, striated retinal hemorrhages and narrowed retinal arterioles.

Autoregulation operates within a certain range of perfusion pressure, and can be disrupted by alterations in blood pressure due to a variety of local and systemic causes. An increase or decrease in perfusion pressure beyond a critical autoregulatory range causes a breakdown of autoregulation. That is, autoregulation does not protect retinal vessels all of the time. The perfusion pressure may go above (malignant hypertension) or fall below (arterial hypotension) the critical range and thus subject the retinal tissue to ischemic damage.[4,5]

Blood Pressure Norms[4]

Normal	Systolic: less than 120mm Hg Diastolic: less than 80mm Hg
At risk (prehypertension)	Systolic: 120-139mm Hg Diastolic: 80-89mm Hg
High	Systolic: 140mm Hg or higher Diastolic: 90mm Hg or higher

Types of Hypertension

Blood pressure varies throughout the population based on age, gender, body mass index and diet.[4] (*See "Blood Pressure Norms," below.*)

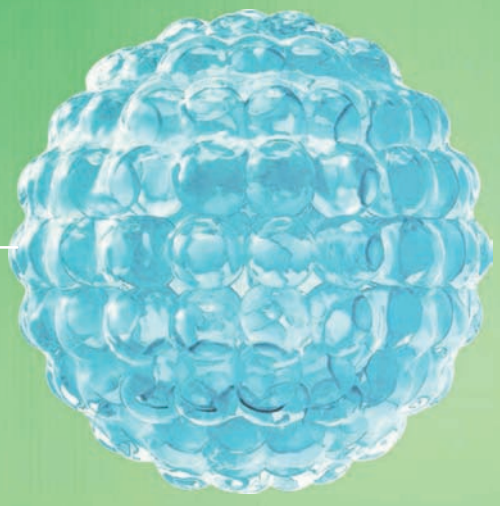
- ***Essential hypertension.*** About 95% of HTN occurs as this form. Essential hypertension does not cause short-term problems, and is often called benign hypertension.
- ***Secondary hypertension.*** A small amount of HTN patients (5%) have this form. It's caused by underlying conditions, such as renal or adrenal disease.[4,5]
- ***Malignant hypertension.*** Some patients have a rapid rise in blood pressure that, if untreated, may lead to death within a year or two. Malignant hypertension, also called accelerated hypertension, is characterized by severely elevated blood pressure (systolic greater than 200mg Hg, diastolic greater than 120mm Hg), renal failure, retinal hemorrhages and exudates with or without optic nerve head swelling. Malignant hypertension may occur in previously normotensive patients, but is usually superimposed on benign or secondary hypertension.[4]

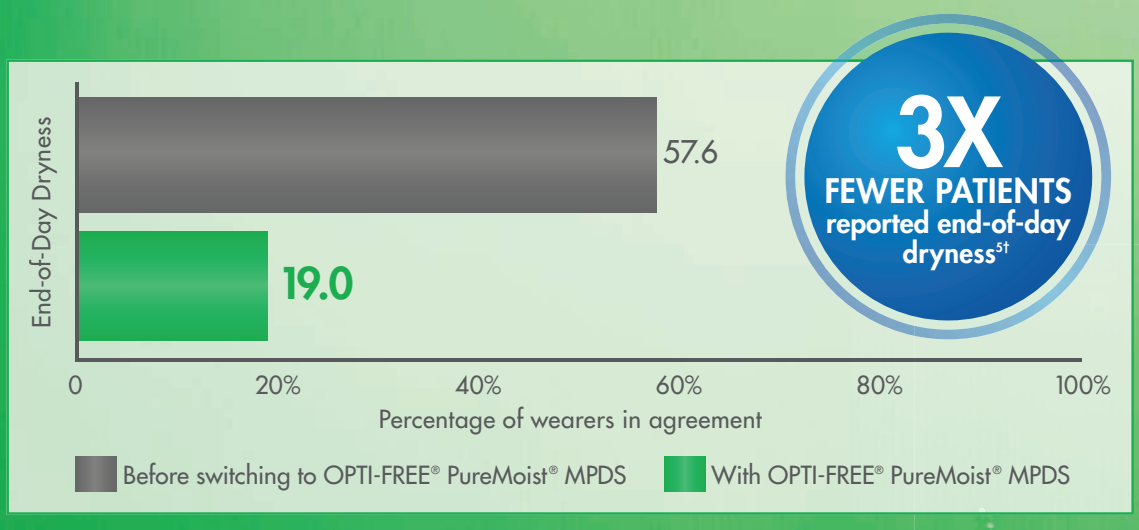
Posterior Segment in HTN

During the early, acute phase of malignant hypertension, terminal arterioles may show two types of

THIS IS WHY OPTI-FREE® PureMoist® Solution allows patients to wear contact lenses MORE COMFORTABLY[1] AND LONGER each day.[2]

The HydraGlyde® Moisture Matrix attaches and forms a hydrophilic environment across the surface of the lens—providing moisture from morning 'til night.[2-4]





3X FEWER PATIENTS reported end-of-day dryness[5†]

†Based on a global survey of 10,611 contact lens wearers who tried OPTI-FREE® PureMoist® for two weeks.

Talk to your patients about the benefit of OPTI-FREE® brand products. Learn more at MYALCON.COM

PERFORMANCE DRIVEN BY SCIENCE™

References: 1. Campbell R, Kame G, Leach N, et al. Clinical benefits of a new multipurpose disinfecting solution in silicone hydrogel and soft contact lens users. *Eye & Contact Lens.* 2012;38(2):93-101. **2.** Alcon data on file, 2011. **3.** Lally J, Ketelson H, Borazjani R, et al. A new lens care solution provide moisture and comfort with today's contact lenses. *Optician.* 2011;241:42-46. **4.** Davis J, Ketelson HA, Shows A, Meadows DL, A lens care solution designed for wetting silicone hydrogel materials. ARVO Meeting Abstracts. 2012;38(2):93-101. **5.** Lemp J, Kern JR. Results from global survey of contact lens-wearer satisfaction with OPTI-FREE® PureMoist® MPDS. *Clinical Optometry.* 2013:5 39-46.

Alcon
a Novartis company

Public Health Implications of Hypertension

Systemic hypertension puts patients at risk for heart disease and stroke, which are leading causes of death in the United States. We need to know about this because:

- **78 million American adults** (33%) have HTN—that's one in every three adults.[1]
- Only **about half** (53%) of people with HTN have their condition under control.[2]
- Hypertension is the **single most important risk factor for stroke**, and its control is essential to reducing death from stroke.[3]
- **Forty-four percent of African-Americans** have high-blood pressure, among the highest rates of any population in the world.[1]
- HTN costs the nation **$46.4 billion** each year. This total includes the cost of health care services, medications to treat high blood pressure and missed days of work.[1]

change. Dilation of the arterioles may occur, along with arteriolar occlusion, which eventually results in the formation of inner retinal ischemic "cotton-wool" spots and focal retinal capillary obliteration.

Focal intraretinal periarteriolar transudates are caused by a severe rise of arterial blood pressure. The retinal vasculature cannot properly autoregulate, causing focal dilation of precapillary retinal arterioles. Breakdown of the blood-retinal barrier occurs, causing increased permeability of the dilated arterioles to plasmatic deposits that flow into the retinal tissue.

The pathogenesis of optic neuropathy in malignant HTN has been a highly controversial subject. The various explanations can be divided into four categories:

• Disc edema due to raised intracranial pressure.

• Disc edema similar to hypertensive encephalopathy.

• Disc edema as a part of hypertensive retinopathy.

• Disc edema that is ischemic in nature.

Studies have shown that hypertensive optic neuropathy is primarily ischemic in nature. Similarly, clinical patterns of optic disc edema and optic disc changes in malignant hypertension are similar to those associated with anterior ischemic optic neuropathy (AION).[5,6]

Treatment and Management

The primary treatment of hypertensive retinopathy is systemic control of the arterial blood pressure with blood pressure medication and lifestyle changes.

The visual prognosis is excellent in mild to moderate cases. However, permanent visual loss may occur when there is persistent foveal edema and/or lipid deposition.

Be aware that a rapid reduction of blood pressure in a patient with hypertensive optic neuropathy may pose a risk of worsening ischemic damage to the optic nerve. A precipitous reduction in blood pressure will reduce perfusion to the optic nerve and central nervous system as a consequence of their autoregulatory changes, resulting in infarction of the optic nerve head and, potentially, acute ischemic neurologic lesions of the central nervous system.

A thorough systemic evaluation should focus on the etiology of the hypertension. Secondary causes should be ruled out in selected patients.

The eye is unique in that it allows direct observation of hypertensive consequences of the microvasculature in vivo. These ocular changes are of value in the management of systemic complications due to hypertension, including diabetes, cardiovascular, cerebrovascular and other systemic vascular diseases. ■

1. Go AS, Mozaffarian D, Roger VL, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2014 Jan 21;129(3):e28-e292.
2. Egan BM, Zhao Y, Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988-2008. JAMA. 2010 May 26;303(20):2043-50.
3. Centers for Disease Control and Prevention. Vital signs: awareness and treatment of uncontrolled hypertension among adults—United States, 2003-2010. MMWR. 2012;61(35);703-9.
4. Kumar V, Abbas A, Fausto N. Robbins and Cotran's Pathologic Basis of Disease, 7th ed. Philadelphia: Elsevier Saunders; 2005.
5. Spencer WH. Systemic diseases with retinal involvement: vascular diseases. In: Ophthalmic Pathology: An Atlas and Textbook (CD-ROM), 4th ed. WB Saunders Co.; 1995.
6. Quillen DA, Blodi BA, Bennett TJ. Clinical Features of Retinal Disease. In: Quillen DA, Blodi BA (eds.). Clinical Retina. Chicago: AMA Press; 2002:136-7.

Retinal and Choroidal Lesions In Hypertension[5,6]

Retinal vascular lesions

- Retinal arteriolar changes (narrowing, nicking)
- Focal intraretinal periarteriolar transudates (FIPTs)
- Inner retinal ischemic spots (cotton-wool spots)
- Retinal capillary changes
- Retinal venous changes
- Increased permeability of the retinal vascular bed

Extravascular retinal lesions

- Retinal hemorrhages (usually situated in the nerve fiber layer and commonly in the distribution of the radial peripapillary retinal capillaries, although they could be located anywhere in the fundus)
- Retinal and macular edema
- Retinal lipid deposits (hard exudates)
- Retinal nerve fiber loss

RPE/Choroidal lesions

- Choroidal vascular bed abnormalities
- Retinal pigment epithelial (RPE) lesions
- Acute focal RPE lesions
- RPE degenerative lesions
- Serous retinal detachment

WHAT'S THE SOLUTION?

By Gregory W. DeNaeyer, OD

Topping Off Happens: Disinfection Efficacy Matters

The real-world disinfection efficacy of a multi-purpose contact lens solution can be affected by how it is used – and even by the contact lenses it is used with.

Microbial keratitis is a rare but serious complication of soft contact lens wear. After devastating outbreaks of multi-purpose contact lens solution-related *Fusarium* and *Acanthamoeba keratitis* in 2006 and 2007, the FDA launched in-depth investigations to identify the causes and associated risk factors.

Certain multi-purpose solutions were linked to the outbreaks, but were not themselves contaminated; rather, in some circumstances, they failed to disinfect against pathogens introduced from the environment.[1,2] In addition, patients' failure to follow recommended disinfection practices was found to be a contributing factor in both outbreaks.[2,3]

TOPPING OFF

A critical noncompliant patient behavior identified in these investigations was "topping off," in which, rather than emptying, rinsing, and drying their contact lens cases after each use, patients simply remove the lenses, adding just enough fresh multi-purpose solution to "top off" the case.[2,3]

In a recent survey of 100 soft contact lens wearers, over a quarter of participants reported occasional or frequent topping off.[4] Many subjects were unaware of a solution-related infection risk and thought lens care was only for removing deposits – not microorganisms.[4]

Topping off may contribute to infection risk by multiple interrelated mechanisms. Failing to empty the case and refresh the solution gives contaminating microbes a chance to proliferate.[2] Recent FDA-sponsored studies looked at the impact that uptake and re-use have on multi-purpose disinfection solution efficacy.[1]

KEY POINTS

- Patient noncompliance – especially "topping off" – is associated with reduced multi-purpose solution efficacy.[3]
- In in-vitro testing of a PHMB solution, uptake of disinfectant into the lens reduced the residual concentration of PHMB.[5]
- In similar testing, the residual concentration of POLYQUAD® and ALDOX® was not significantly diminished in the residual solution.[6]

LENS-SOLUTION INTERACTIONS

The ISO/FDA require that a multi-purpose disinfecting solution demonstrate antimicrobial efficacy. Since stand-alone antimicrobial efficacy tests are performed with fresh multi-purpose solution – without exposure to contact lens or a lens case – results may not reflect the impact of lens storage in a lens case.[1] Recent FDA-sponsored research, however, has looked specifically at interactions between contact lenses and multi-purpose solutions, finding that some soft lenses will absorb some preservatives over time, thus diminishing the disinfectant concentration in the solution and reducing its efficacy against some microorganisms.[1,5,6]

DISINFECTION DIFFERENCES

Clavet and coworkers studied the effects of soaking six silicone hydrogel and two hydrogel lens types in a multi-purpose solution contacting the disinfectant polyhexamethylene biguanide (PHMB, 0.0001%, 6-hour soak). Lens cases filled with the multi-purpose solution, but no lenses, served as controls. At intervals of 6, 12, 72, and 168 hours, multi-purpose solution was analyzed for PHMB concentration and biocidal activity against *Fusarium solani*.[1] Certain lens materials (balafilcon A, etafilcon A, and polymacon) absorbed the PHMB, significantly reducing its concentration and lowering the residual solutions' efficacy against *Fusarium*.[1]

A separate, similarly designed experiment also showed depletion of PHMB in the presence of certain lens materials (galyfilcon A, comfilcon A, balafilcon A, polymacon, and etafilcon A), and demonstrated a significant reduction in the disinfecting efficacy against *Staphylococcus aureus*.[5] Reusing and topping off a solution may reduce its antimicrobial efficacy in the presence of a lens.

In similar testing, however, soaking silicone hydrogel and hydrogel lenses in a solution containing the dual biocidal agents polyquaternium-1 (0.001%) and myristamidopropyl dimethylamine (0.0005%) did not significantly reduce residual preservative levels or antimicrobial efficacy against *S. aureus*.[6]

CLINICAL VALUE

Helping patients to be successful in contact lenses requires clear, repeated education about choosing the right lens care solution and using it properly. I always ask returning patients about their contact lens care; I make sure they are aware of the dangers of topping off, and of differences in multi-purpose solutions.

Recommending a multi-purpose solution and talking about proper lens care and multi-purpose solution use are important first steps. Reinforcing this discussion, as our practice does, with written instructions, gives patients a road map to successful and comfortable contact lens wear. ■

Gregory W. DeNaeyer, OD, practices at Arena Eye Surgeons, in Columbus, Ohio.

1.Clavet CR, Chaput MP, Silverman MD, et al. Impact of contact lens materials on multipurpose contact lens solution disinfection activity against Fusarium solani. Eye Contact Lens. 2012;38:379-84.
2.Verani JR, Lorick SA, Yoder JS, et al. National outbreak of Acanthamoeba keratitis associated with use of contact lens solution, United States. Emerg Infect Dis. 2009;15:1236-42.
3.Chang DC, Grant GB, O'Donnell K, et al. Multistate outbreak of Fusarium keratitis associated with use of contact lens solution. JAMA. 2006;296:953-63.
4.Dumbleton KA, Spafford MM, Sivak A, et al. Exploring compliance: a mixed methods study of contact lens wearer perspective. Optom Vis Sci. 2013;90:898-908.
5.Shoff ME, Lucas AD, Brown JN, et al. The effects of contact lens materials on a multipurpose contact lens solution disinfection activity against Staphylococcus aureus. Eye Contact Lens. 2012;38:368-73.
6.Shoff ME, Lucas AD, Phillips KS, et al. The effect of contact lens materials on disinfection activity of polyquaternium-1 and myristamidopropyl dimethylene multipurpose solution against Staphylococcus aureus. Eye Contact Lens. 2012;38:374-8.

Left your *Review of Optometry* magazine at the office? No problem!

Read *Review* on the go from any mobile device!

Just simply go to www.revoptom.com and click on the digimag link to get your current issue.

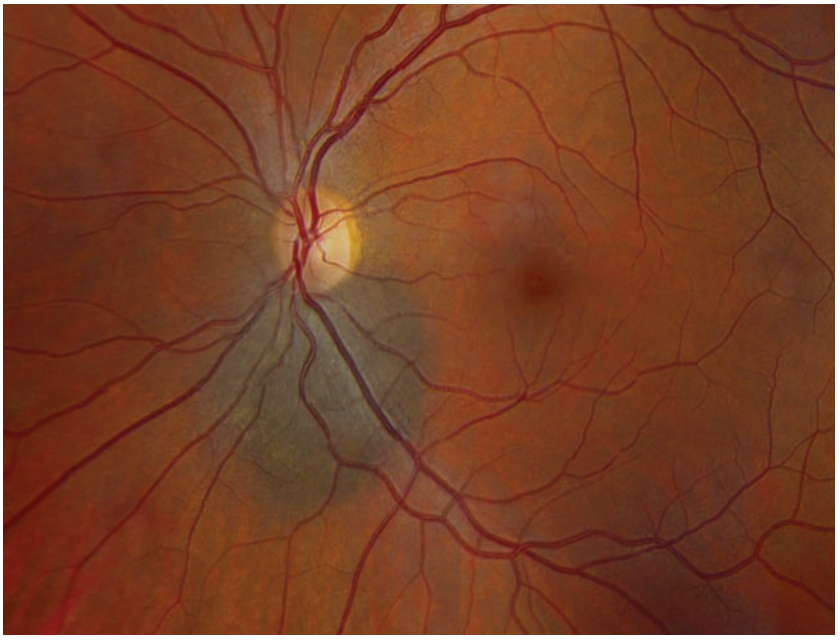
Significant Growth Potential?

This patient presented with an unidentified lesion in her left eye. Is it likely to turn malignant? **By Mark T. Dunbar, OD**

A 43-year-old Hispanic female presented with a chief complaint of reduced near acuity that had persisted for six months. Her last eye exam was several years ago and, at that time, she didn't require any optical correction. Her medical history was unremarkable. The patient reported a 20-year history of smoking, but added that she quit five years ago.

On examination, entering distance acuities measured 20/20 OU. She read J2 OU at near, and improved to J1+ with +1.25D of correction OU. Extraocular motility testing was normal. Confrontation visual fields were full to careful finger counting OU. The pupils were equally round and strongly reactive, with no evidence of afferent defect in either eye. The anterior segment examination was unremarkable.

Dilated fundus exam of the right eye was completely normal. The left eye, however, exhibited a clinically significant change (*figure 1*). So, we obtained a spectral-domain optical coherence tomography (SD-OCT) scan of her left eye (*figure 2*). The maculae and peripheral retinae were normal in both eyes.



1. A fundus photograph of our patient's left eye. Note the lesion located adjacent to the optic nerve.

Take the Retina Quiz

1. What is the likely diagnosis?
a. Choroidal nevus.
b. Choroidal melanoma.
c. Melanocytoma.
d. Metastatic carcinoma.

2. What additional testing would be most helpful for this patient?
a. Fluorescein angiogram.
b. Ultrasound.
c. Liver function panel.
d. Chest X-ray.

3. How should this patient be managed?
a. Observation.
b. Enucleation.
c. Plaque radiotherapy.
d. Chemotherapy.

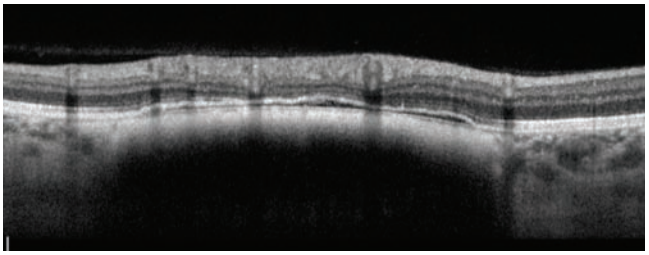
4. What is the estimated likelihood that the lesion will grow over the next five years?
a. Very unlikely.
b. Unlikely—approximately a 25% chance.
c. Likely—approximately a 50% chance.
d. Very likely.

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

For answers, turn to page 118.

Discussion

The slate gray-colored lesion located adjacent to the optic nerve likely is a choroidal nevus—a benign developmental tumor usually observed as an incidental finding during a routine eye examination.



2. Enhanced-depth imaging scan of the lesion on Spectralis OCT. What do you see?

Ninety-five percent of choroidal nevi will be flat, slate gray in appearance and less than three disc diameters in size.[1] They are comprised of benign spindle cells and branched melanocytes. By contrast, most melanomas are usually more than 3.0mm in thickness, can be variably pigmented, and may have overlying orange pigment, which represents lipofuscin—a worrisome finding suggestive of an increased potential for malignancy.

The diagnosis is usually straightforward, with little or no concern that the lesion is a choroidal melanoma. However, that's not always the case. Occasionally, choroidal nevi can be difficult to distinguish from small malignant melanomas—especially if the lesions aren't flat, as was the case in our patient. Even though stereoscopic clinical exam didn't reveal any elevation, we could definitely see that the lesion exhibited some degree of thickness. Indeed, we estimated that the lesion measured 6.0mm x 5.0mm x 1.1mm using both ultrasound and SD-OCT.

Other SD-OCT findings also were quite interesting. We noted retinal pigment epithelium (RPE) irregularity above the lesion, as well as mild anterior bowing secondary to the lesion's elevation. Further, the scan revealed shallow subretinal fluid located underneath the inferior RPE, which is a risk factor for growth and/or transformation.

Using the enhanced-depth imaging module on Spectralis OCT (Heidelberg Engineering), we also noted acoustic shadowing within the choroid, which was seen as a darker, optically empty space. This is due to the inability of low coherent light to pass through the lesion secondary to increased melanin density within the tumor.

Additional clues that may be helpful in differentiating a suspicious nevus from a small melanoma include the presence of drusen, RPE hyperplasia, bone spicule pigment, RPE atrophy and choroidal neovascularization. Any of those findings are indicative of chronicity and suggest that the lesion has been present for some time. An actively growing melanoma, on the other hand, does not have time to develop these more chronic changes.

Ultimately, the best way to determine if the lesion is a nevus or a melanoma is to photodocument the presentation over time. Choroidal nevi will rarely exhibit any significant growth.

An interesting question to consider: Do melanomas develop from a malignant transformation of preexisting nevi, or are they completely separate entities? Most experts agree that the vast majority of melanomas manifest as separate entities.[2-4] Malignant transformation from preexisting nevi is exceedingly rare, with an incidence of one in 4,800 to one in 8,800 individuals.[2]

In 1995, ophthalmologists Carol and Jerry Shields and associates at the Wills Eye Institute in Philadelphia identified five common risk factors associated with small choroidal melanocytic lesion growth in 1,329 patients:[3]

- **Tumor thickness greater than 2.0mm.**
- **Presence of subretinal fluid.**
- **Visual symptoms.**
- **Presence of orange pigment.**
- **Posterior tumor margin touching the disc.**

The researchers observed lesion growth in just 4% of patients who had no risk factors. However, 36% of patients who had one risk factor and 50% of those who had three or more risk factors experienced lesion growth.[3]

In a subsequent study, the Shields research team noted that patients were at an increased risk for metastasis if they exhibited posterior tumor margin touching the disc, documented growth and/or increased tumor thickness (greater than or equal to 1.1mm).[4]

Even though we believe our patient's lesion is a choroidal nevus, she has a few risk factors for growth and/or malignant transformation. These include the close proximity to the optic nerve, its thickness and the presence of subretinal fluid. Because of the relatively small lesion size and uncertainty for growth potential, we elected to follow her every four to six months. We also prescribed a pair of reading glasses to address her near vision difficulties.

After three years of follow-up evaluations, our patient's lesion has yet to show any signs of growth. ■

1. Gass JD. Problems in the differential diagnosis of choroidal nevi and malignant melanomas. Am J Ophthalmol. 1977 Mar;83(3):299-323.
2. Singh AD, Kalyani P, Topham A. Estimating the risk of malignant transformation of a choroidal nevus. Ophthalmology. 2005 Oct;112(10):1784-9.
3. Shields CL, Shields JA, Kiratli H, et al. Risk factors for growth and metastasis of small choroidal melanocytic lesions. Ophthalmology. 1995 Sep;102(9):1351-61.
4. Shields, CL, Cater JC, Shields JA, et al. Combination of clinical factors predictive of growth of small choroidal menalocytic lesions tumors. Arch Ophthalmol. 2000 Mar;118(3):360-4.

12 CE Credits
(COPE approval pending)

Mark your calendars for the

6th Annual

West Coast Optometric Glaucoma Symposium

December 12-13, 2014

HILTON WATERFRONT

Huntington Beach, California

Discounted Room Rate: $190/night+tax.
Room rate is limited and based on hotel availability.

Call: 800-822-7873 and identify yourself as a participant of the West Coast Optometric Glaucoma Symposium. (WCOGS)

12 COPE Credits* for only $185!

MEETING CO-CHAIRS:

MURRAY FINGERET, OD

ROBERT N. WEINREB, MD

SPEAKERS

Howard Barnebey, MD • John Flanagan, OD, PhD
Jeffrey Goldberg, MD, PhD • Richard Madonna, OD

3 WAYS TO REGISTER

ONLINE: www.revoptom.com/WCOGS2014

EMAIL: ReviewMeetings@Jobson.com

CALL: Lois DiDomenico 877-451-6514

Brought to you by

REVIEW® OF OPTOMETRY

cope®

** Approval pending*

Partially supported by an educational grant from

Alcon


Treatment or Disease?

In some instances, properly medicating a patient may lead to other problems.

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

The sacred motto in medicine is, "Above all else, do no harm." This seems reasonable enough, but sometimes there are untoward effects of therapy. For example, we all know that topical steroids can cause glaucoma and cataracts. But several other frequently used agents can yield adverse effects, too. In this month's column, we'll review of few of these.

Avastin

In recent years, intravitreal Avastin (bevacizumab, Genentech) has been used regularly to treat neovascular disease and macular edema resulting from diabetic retinopathy, retinal vein occlusion, and several other ocular conditions that cause retinal capillary closure and hypoxia. These conditions all feature up-regulation of vascular endothelial growth factor (VEGF), which stimulates angiogenesis and promotes endothelial cell migration and proliferation.[1] Also, VEGF is associated with blood-retinal barrier breakdown, increased vascular permeability and fluid leakage. These mechanisms precipitate neovascularization and retinal edema.[2]

Avastin, a VEGF inhibitor, is a humanized monoclonal antibody that was initially developed as treatment for metastatic colorectal cancer.[3] Such inhibitors block the up-regulation of VEGF, eliminate the stimulus for neovascularization and reduce the vascular permeability that causes retinal edema.

One of the potential complications of Avastin use is endophthalmitis associated with intraocular injection.[4] While uncommon (occurring in approximately one in 3,000 injections), the threat of potential vision loss is tremendous. Use of an eyelid speculum and povidone iodine, as well as avoidance of needle contact with the eyelid margin, greatly reduce the patient's risk of post-injection endophthalmitis.

Despite these risk reduction strategies, vision loss from endophthalmitis cannot be absolutely eliminated. Intravitreal Avastin typically is prepared by a compounding pharmacy, which naturally introduces a risk of contamination. As national media sources reported throughout the country, 12 patients in South Florida developed endophthalmitis in July 2011 after receiving intravitreal Avastin injections.[5,6] Seven of the 12 patients ultimately were enucleated, and only one patient achieved a final visual acuity better than finger counting.

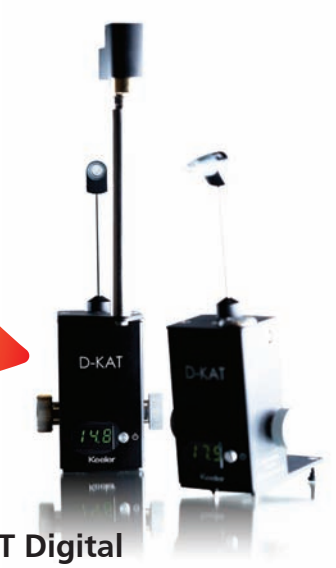
Because this situation included an unusually high number of endophthalmitis cases within a short time period in one geographic area, some researchers speculated that the cause originated from one source. Ultimately, this proved to be the case, as *Streptococcus mitis/oralis* was cultured from the majority of patients and from all unused syringes that came from a single compounding pharmacy.[7] While much of the country reacted in fear, this was a localized issue that was caused by a lack of infection protocol at one facility. Overall, however, intravitreal treatment with Avastin is a safe, effective option for patients.

Jetrea

Vitreomacular adhesion and vitreomacular traction syndrome may reduce vision and can progress to macular hole development, which can cause even greater visual morbidity. Vitreoretinal adhesion and traction exist due to fibrocellular proliferation at the macula. Previously, these findings only could be relieved through vitrectomy. Now, the proteolytic enzyme ocriplasmin (Jetrea, ThromboGenics) can resolve vitreomacular adhesions via intravitreal injection rather than invasive surgery. Ocriplasmin cleaves fibronectin and laminin at the vitreoretinal interface. Success has been reported in up to 25% of patients with symptomatic vitreomacular traction syndrome following a single intravitreal injection of Jetrea.[8,9]

Recently, however, there have been reports of possible retinal toxicity and vision loss after Jetrea use.[10] One report detailed a 71-year-old woman with symptomatic vitreomacular traction who received intravitreal Jetrea and experienced vision darkening in dim illumination that persisted for four months, despite traction release and a general improvement

Tonometry Done Right



D-KAT Digital
Keeler quality.

Pulsair Desktop
Smallest footprint and simple to use!

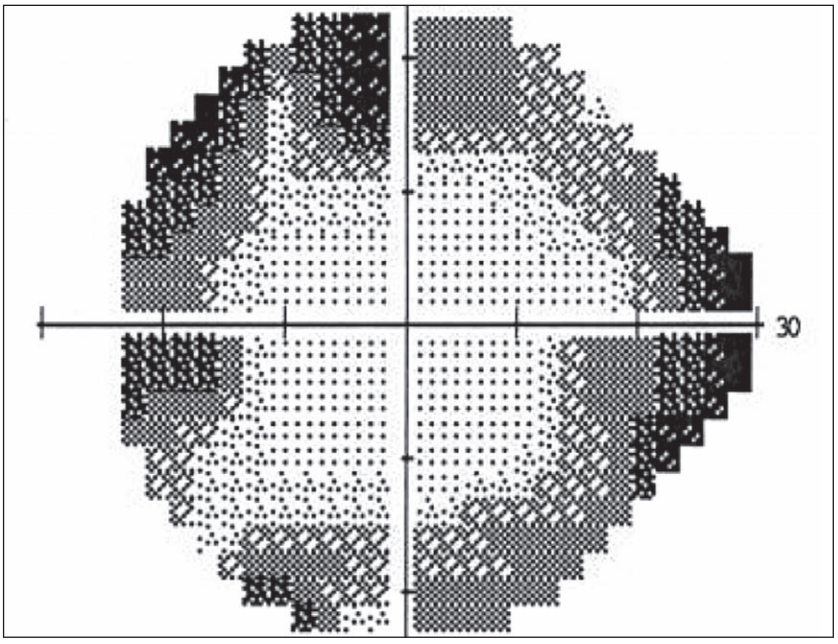
Purchase a Pulsair Desktop by October 31, 2014 and get a $1,300 Instant Rebate!

Intellipuff
The standard for hand held mobility.

Buy Online!
keelerusa.com

Keeler
OPTICS

Keeler Instruments, Inc. • 456 Parkway • Broomall, PA 19008 • Tel: (800) 523-5620 • Fax: (610) 353-7814 • email: keeler@keelerusa.com



Patients who use the antiepileptic agent Sabril may experience peripheral visual field constriction, as seen here.

in visual acuity. Spectral-domain optical coherence tomography (SD-OCT) showed a disruption of her photoreceptor inner segment/outer segment layer. Additionally, a reduction of electroretinogram (ERG) amplitudes accounted for her complaint of darkened vision. The authors speculated that Jetrea may have a diffuse enzymatic effect on the photoreceptors or the retinal pigment epithelium located outside of immediate areas of vitreomacular adhesion.[10]

Another report detailed a 63-year-old woman who experienced acute panretinal dysfunction after intravitreous Jetrea. The patient demonstrated acute visual acuity loss, visual field constriction, attenuated retinal arteries, loss of retinal outer segment retinal signals on SD-OCT and severely reduced ERG responses. The authors concluded that retinal dysfunction associated with intravitreous Jetrea injection is not limited to the macular region and may involve the entire retina. Further, they suggested that enzymatic cleavage of intraretinal laminin is a biologically plausible mechanism for acute ocriplasmin retinal toxic effects.[11]

While these reports may cause concern and perhaps hesitation to recommend Jetrea as an alternative to vitreoretinal surgery, be aware that the medication otherwise showed a positive safety profile prior to these reports.

Jetrea is supplied in a single-use glass vial, which contains 0.5mg ocriplasmin for intravitreal injection after dilution. Thus, Jetrea has to be reconstituted in the office. While the toxic effects recently reported could be due to the formulation itself, some have speculated that such complications may have arisen due to improper dilution or other untoward effects from the reconstitution process.

Sabril

Sabril (vigabatrin, Lundbeck) is an effective antiepileptic drug used to treat refractory complex partial seizures and infantile spasms. While Sabril has been used in other countries since 1989, it didn't receive FDA approval until 2009. This 20-year delay likely was influenced by the drug's potential to cause profound visual field constriction. Specifically, Sabril has been noted to cause reduction of cone d-wave amplitude on ERG testing.[12]

Binasal defects are detected initially, progressing to bilateral concentric field constriction that preserves central acuity.[13] In one prospective analysis of 734 patients treated with Sabril, 71% exhibited visual field defects.[14] A retrospective analysis documented visual field defects in 66% of men and 54% of women who used Sabril.[15]

Further, researchers noted that the prevalence of visual field loss seems to increase with dose size, patient age and duration of therapy.[16,17] Beyond visual field loss, another study indicated that the parapapillary retinal nerve fiber layer is notably thin on SD-OCT scans of patients who used Sabril.[18]

Because Sabril is not a commonly prescribed drug, we wanted to alert eye care practitioners of the possibility of potentially devastating visual field loss and the need for ongoing screening. It appears that visual field loss does not progress after Sabril cessation, but functional visual field improvement won't occur either.

For this reason, order threshold perimetry at least every six months for patients on Sabril treatment. Also, consider SD-OCT as an adjunctive monitoring tool, particularly in children who cannot perform perimetry. In these instances, ERG may also be a helpful. Should any structural or functional dysfunction develop, the medication most likely will need to be

NEW

Lombart CS-5 Package

Quality, Style & Value

Package includes:

- The Lombart CS-5 Chair & Stand
- Topcon VT-10 Refractor
- Topcon SL-2G Slit Lamp
- Reichert LongLife Chart Projector with mount, slide & screen
- Upgrade to the Lombart CVS-PC for only $1500 or to the CVSi21 for only $2000 more
- Additional upgrades & configurations available.

$13,595

Or lease for $265/mo. for 60 months*

*Lease rate subject to credit approval, 1st payment is due at signing with 59 remaining rental payments of $265 and a $1.00 purchase option. Taxes, freight and installation additional. Hand Instruments optional. Subject to change without notice.

1-800-566-2278

Call 1-800-Lombart

Or Your Local Lombart Representative

Corporate Office - 5358 Robin Hood Road, Norfolk, VA 23513-2430
757-853-8888 | FAX 757-855-1232 | 800-566-2278
www.lombartinstrument.com
lombart@lombartinstrument.com

Sales and Service Centers Coast to Coast

ATLANTA • BALTIMORE/WASHINGTON D.C. • BOSTON • BOYNTON BEACH/MIAMI • BRADENTON • CHARLOTTE • CHICAGO • CINCINNATI • DALLAS • DENVER • DETROIT • GREENSBORO • HOUSTON
JACKSON • KANSAS CITY • KNOXVILLE • LOS ANGELES • MILWAUKEE • MINNEAPOLIS • NEW JERSEY/NEW YORK/PENNSYLVANIA • NORFOLK • PORTLAND • SAN ANTONIO • SAN DIEGO • SAN FRANCISCO

THE REVOLUTION BEGINS NOW

> "We are changing the perception of optometry one market and one organization at a time."
>
> – **Jim Greenwood**
> President and CEO
> Vision Source®
> April 10, 2014

At the 2014 Exchange®, the annual meeting for Vision Source®, Jim Greenwood addressed the success to date in aligning Vision Source® members with health care organizations. Already in 2014, CoxHealth, Baptist Health Quality Network, Arizona Care Network, Memorial Hermann Health, and WellMed have selected Vision Source® to be their preferred eye health care provider. Together, these organizations represent more than 7 million lives.

This success is not the result of chance. Vision Source® has a plan that enables members to connect with ACOs, Medicare Advantage plans, and other emerging health care organizations in their communities. At the same time, Vision Source® is leading the way in positioning optometry as a vital partner in patient-centered care to the health care community as a whole.

There's a revolution taking place in health care. If you would like to be at the forefront of changing minds and behavior when it comes to optometry, Vision Source® would be honored to have you join us.

VisionSourcePlan.com

Vision Source® SIGNATURE EYE CARE

discontinued immediately upon consult with patients' neurologists.

Any medical treatment always carries some risk of adverse effects—some anticipated, some not. Occasionally, there may be quality issues associated with otherwise safe treatments. Additionally, there may be systemic medications with profound ocular implications that are unfamiliar to many practitioners. ■

1. Priglinger SG, Wolf AH, Kreutzer TC, et al. Intravitreal bevacizumab injections for treatment of central retinal vein occlusion: six-month results of a prospective trial. Retina. 2007 Oct;27(8):1004-12.
2. Pe'er J, Folberg R, Itin A, et al. Vascular endothelial growth factor upregulation in human central retinal vein occlusion. Ophthalmology. 1998 Mar;105(3):412-6.
3. Marshall J. The role of bevacizumab as first-line therapy for colon cancer. Semin Oncol. 2005 Dec;32(6 Suppl 9):S43-7.
4. Schwartz SG, Flynn HW Jr. Endophthalmitis associated with intravitreal anti-vascular endothelial growth factor injections. Curr Ophthalmol Rep. 2014;2(1):1-5.
5. Goldberg RA. *Streptococcus* endophthalmitis outbreak after intravitreal injection of bevacizumab: one-year outcomes and investigative results. Ophthalmology. 2013;120(7):1448-53.
6. Goldberg RA, Flynn HW Jr, Isom RF, et al. An outbreak of *Streptococcus* endophthalmitis after intravitreal injection of bevacizumab. Am J Ophthalmol. 2012 Feb;153(2):204-8.
7. Matthews JL, Dubovy SR, Goldberg RA, Flynn HW Jr. Histopathology of *Streptococcus mitis/oralis* endophthalmitis after intravitreal injection with bevacizumab: a report of 7 patients. Ophthalmology. 2014;121(3):702-8.
8. Kuppermann BD. Ocriplasmin for pharmacologic vitreolysis. Retina. 2012;32 Suppl 2:S225-8.
9. Stalmans P, Benz MS, Gandorfer A, et al. Enzymatic vitreolysis with ocriplasmin for vitreomacular traction and macular holes. N Engl J Med. 2012;367(7):606-15.
10. Tibbetts MD. Vision loss after intravitreal ocriplasmin: correlation of spectral-domain optical coherence tomography and electroretinography. JAMA Ophthalmol. 2014;132(4):487-90.
11. Fahim AT, Khan NW, Johnson MW. Acute panretinal structural and functional abnormalities after intravitreous ocriplasmin injection. JAMA Ophthalmol. 2014 Apr 1;132(4):484-6.
12. Dragas R, Westall C, Wright T. Changes in the ERG d-wave with vigabatrin treatment in a pediatric cohort. Doc Ophthalmol. 2014 Jul 10. [Epub ahead of print].
13. Willmore LJ, Abelson MB, Ben-Menachem E, et al. Vigabatrin: 2008 update. Epilepsia. 2009;50(2):163-73.
14. Wild JM, Chiron C, Ahn H, et al. Visual field loss in patients with refractory partial epilepsy treated with vigabatrin: final results from an open-label, observational, multicentre study. CNS Drugs. 2009;23(11):965-82.
15. Wild JM, Fone DL, Aljarudi S, et al. Modelling the risk of visual field loss arising from long-term exposure to the antiepileptic drug vigabatrin: a cross-sectional approach. CNS Drugs. 2013;27(10):841-9.
16. Maguire MJ, Hemming K, Wild JM, et al. Prevalence of visual field loss following exposure to vigabatrin therapy: a systematic review. Epilepsia. 2010;51(12):2423-31.
17. Pellock JM. Balancing clinical benefits of vigabatrin with its associated risk of vision loss. Acta Neurol Scand Suppl. 2011;(192):83-91.
18. Clayton LM, Devile M, Punte T, et al. Patterns of peripapillary retinal nerve fiber layer thinning in vigabatrin-exposed individuals. Ophthalmology. 2012;119(10):2152-60.

11th Annual OCRT Refractive Symposium

The Meeting of the Year for ODs involved in advanced ocular disease management, refractive surgery, cataract surgery and innovative technologies

November 11, 2014

Colorado Convention Center
700 14th Street
Denver, CO 80202

Up to 8 CE Credits*

SCHEDULE

7:30am - 8:00am	Breakfast
8:00am - 12:00pm	**CE Program**
12:00pm - 1:30pm	Lunch
1:30pm - 5:30pm	**CE Program**

The OCRT will sponsor its 11th Annual Refractive Technology Symposium, bringing together some of the brightest minds in the refractive arena to discuss complication management, evolving technologies, management of dry eye and a new feature – an open forum to discuss cases submitted by you and other meeting attendees. The meeting, being held the day before the American Academy of Optometry meeting in Denver, Co., presents a unique forum to share your ideas, thoughts, and experiences with your colleagues. Eight hours of COPE-approved CE will be awarded to attendees. The registration fee includes a continental breakfast and lunch.

TO REGISTER ONLINE:
www.ocrt.org/Symposium
$200 for 8 hours of CE; $150 for OCRT Members

For more information, contact Josh Johnston at drj@gaeyepartners.com or Bill Tullo at bill.tullo@tlcvision.com or visit www.ocrt.org

Administered by

REVIEW OF OPTOMETRY

cope® *Approval Pending

ocrt

SPONSORS

ALLERGAN

TearLab

GLAUKOS Changing Perspectives

OCULUS We focus on progress

STAAR SURGICAL

Supported by Unrestricted Educational Grants from:

Abbott Medical Optics Alcon Bausch & Lomb


Is Damage Still Occurring?

Can Plaquenil macular toxicity continue to progress long after the patient stops taking the drug? **By Diana L. Shechtman, OD, and Paul M. Karpecki, OD**

A 55-year-old black female returned to the clinic for a six-month follow-up evaluation. Her ocular history was remarkable for Plaquenil (hydroxychloroquine, Sanofi-Aventis) macular toxicity.

Her systemic history was significant for lupus, which was initially diagnosed 15 years earlier. For the last 14 years, she took 400mg Plaquenil QD. At her last visit, we instructed the patient to discontinue medication use.

At this visit, her dilated fundus exam showed some mottling within both maculae. Spectral-domain optical coherence tomography (SD-OCT) macular change analysis revealed increased parafoveal thinning since her last visit.

Does the increased thinning documented on the SD-OCT scan represent Plaquenil macular toxicity progression—even though she discontinued the medication six months earlier?

Toxicity Risk and Screening

Plaquenil is an antimalarial agent that's commonly used to manage several autoimmune disorders, including rheumatoid arthritis and systemic lupus erythematosus. Although the drug has a relatively safe systemic profile, its use is associated with an increased incidence of macular toxicity—particularly if an individual has been on Plaquenil therapy for many years.[1,2] Specifically, a 2010 study indicated that the overall risk of Plaquenil macular toxicity increases by about 2% for every 10 to 15 years of continuous drug use.[3] Various risk factors, including dosing size and duration, also contribute to the development of macular toxicity.[4]

Late-stage Plaquenil macular toxicity characteristically appears as a "bull's eye" maculopathy with associated vision loss. To prevent significant visual compromise, the American Academy of Ophthalmology (AAO) established preliminary screening guidelines in 2002.[5] Recommended testing includes a comprehensive eye exam consisting of a posterior segment assessment and a careful evaluation of associated macular changes or signs of retinal disease. Baseline fundus photography is considered optional.[5]

In 2011, the AAO published updated ocular examination guidelines for screening patients on Plaquenil therapy.[4] Recommended testing includes a comprehensive eye exam with an assessment of posterior segment via dilated funduscopy.

When evaluating patients for suspected Plaquenil toxicity, baseline fundus photos can be used to initially document disease severity or to evaluate subsequent changes. Neither dilated funduscopy nor fundus photography is sensitive enough to identify early findings associated with macular toxicity. Thus, the AAO's guidelines recommend routine screening and monitoring of patients using spectral-domain optical coherence tomography (SD-OCT), multifocal electroretinogram and/or fundus autofluorescence (FAF).[4] Also, consider performing a 10-2 visual field test (using white light stimulus) to assess any functional change.

Early structural changes documented on the SD-OCT scan may reflect localized areas of macular thinning, as well as disruption at the photoreceptor integrity line (the junction where the inner segment meets the outer segment) or the ellipsoid zone (EZ). These findings will be particularly evident in the parafoveal area.[6] SD-OCT parafoveal defects associated with Plaquenil toxicity have been described as a "flying saucer sign" and/or "sinkhole displacement."[7]

Take note that objective documentation of functional and structural change, via SD-OCT for example, not only assesses early retinal changes, but also can track progressive damage.[8,9]

Continued Deterioration?

In 2014, Michael F. Marmor, MD, and associates evaluated the effects of current disease stage on the progression of Plaquenil macular toxicity following dosing cessation.[10] Eleven subjects with variable degrees of maculopathy secondary to Plaquenil use were evaluated for one to three years following drug discontinuation. The researchers performed SD-OCT, FAF and 10-2 visual field tests on all patients. EZ line length (as measured from the foveal center to the area of EZ line loss) and foveal thickness measurements served as the standard metrics on SD-OCT testing.

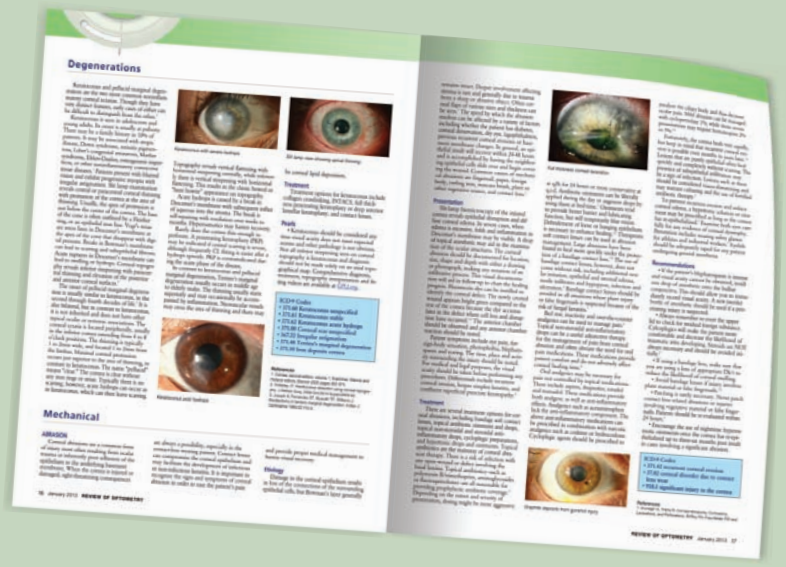
Supplement to the October issue of REVIEW OF OPTOMETRY

COMING IN OCTOBER!

The Corneal Atlas

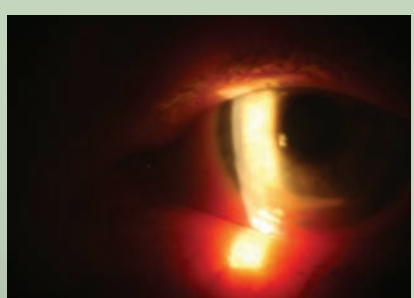
A desk reference for practicing optometrists.

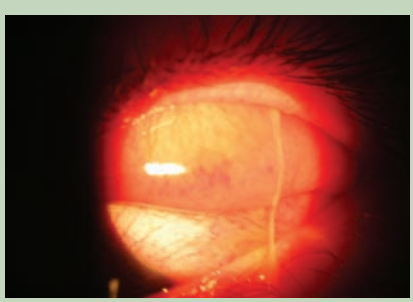
When patients present with a corneal anomaly that can compromise vision, busy clinicians need quick access to authoritative information on how to make the diagnosis and restore ocular health.

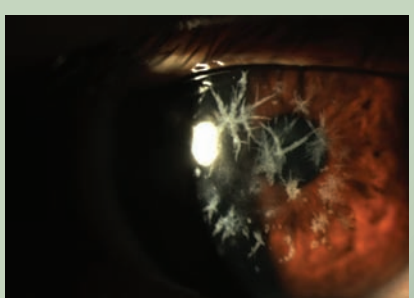


In this high-profile annual supplement, *Review of Optometry* gives its readers a valuable desk reference that summarizes the latest thinking on the etiology and clinical presentations of corneal diseases, plus expert advice on how they should be managed. The dozens of high-quality images in this "atlas" provides readers with visual guidance that augments the practical commentary from our panel of corneal experts.

The common—and not-so-common—corneal anomalies discussed will include: microbial keratitis, adenoviral keratoconjunctivitis, herpes simplex keratitis, herpes zoster ophthalmicus, Acanthamoeba keratitis, fungal keratitis, epithelial basement membrane dystrophy, corneal abrasions, Reis-Bucklers dystrophy, lattice corneal dystrophy, Fuchs endothelial dystrophy, toxicity reactions, dry eye, uveitis and more!







Patients exhibited a wide range of disease severity. Staging was quantified as "early" in subjects with structural or functional parafoveal damage; "moderate" in those with 50% to 100% parafoveal damage, with marked thinning of the parafoveal area and no associated retinal pigment epithelium (RPE) damage on SD-OCT; and "severe" in patients who exhibited the classic bull's eye maculopathy and secondary RPE damage.

The authors reported that the visual fields and FAF results were of limited use, given that the data did not show any consistent change over time.[10] However, patients with severe macular toxicity did show a pronounced decrease in autofluorescence on FAF testing, which was associated with progressive RPE damage.

SD-OCT, on the other hand, showed characteristic progressive findings that correlated to the stage of macular toxicity.[10] The researchers documented no associated EZ line damage in either the mild or moderate stages of disease. Patients with severe macular toxicity, however, exhibited progressive EZ line loss that averaged 100µm per year.

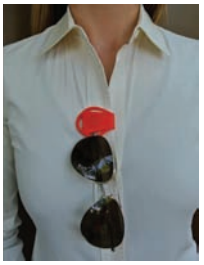
So, was our patient's increased parafoveal thinning a result of continued disease progression? It is a definite possibility. All we can be certain of, however, is that earlier detection of macular toxicity using the latest Plaquenil screening guidelines may help limit or prevent further damage following drug cessation. Longer follow-up periods and larger study populations are still required to better assess long-term progression and stabilization rates. ■

1. Levy GD, Munz SJ, Paschal J, et al. Incidence of hydrochloroquinine retinopathy in a large multicentered outpatient practice. Arthritis Rheum. 1997 Aug;40(8):1482-6.
2. Mavrikakis I, Sfikakis PP, Mavrikakis E, et al. The incidence of irreversible retinal toxicity in patients treated with hydroxychloroquine: a reappraisal. Ophthalmology. 2003 Jul;110(7):1321-6.
3. Wolfe F, Marmor MF. Rates and predictors of hydroxychloroquine retinal toxicity in patients with rheumatoid arthritis and systemic lupus erythematosus. Arthritis Care Res (Hoboken). 2010 Jun;62(6):775-84.
4. Marmor MF, Kellner U, Lai T, et al. Revised recommendations on screening for chloroquinine and hydroxychloroquinine retinopathy. Ophthalmology. 2011 Feb;118(2):415-22.
5. Marmor MF, Carr RE, Easterbrook M, et al. Recommendations on screening for chloroquine and hydroxychloroquine retinopathy: a report by the American Academy of Ophthalmology. Ophthalmology. 2002 Jul;109(7):1377-82.
6. Payne JF, Hubbard GB 3rd, Aaberg TM Sr, Yan J. Clinical characteristics of hydroxychloroquinine retinopathy. Br J Ophthalmol. 2011 Feb;95(2):245-50.
7. Chen E, Brown DM, Benz M, et al. Spectral domain optical coherence tomography as an effective screening test for hydroxychloroquine retinopathy (the "flying saucer" sign). Clin Ophthalmol. 2010 Oct 21;4:1151-8.
8. Kellner S. Spectral domain optical coherence tomography detects early stages of chloroquine retinopathy similar to multifocal electroretinography, fundus autofluorescence and near-infrared autofluorescence. Br J Ophthalmol. 2009 Nov;93(11):1444-7.
9. Lai TY, Ngai JW, Chan WM, et al. Visual field and multifocal electroretinography and their correlations in patients on hydroxychloroquine therapy. Doc Ophthalmol. 2006 May;112(3):177-87
10. Marmor MF, Hu J. Effect of disease stage on progression of hydroxychloroquine retinopathy. JAMA Ophthalmol. 2014 Jun 12. [Epub ahead of print]

Product Review

Accessories

Handy Eyeglass Clip



The Lens Friend is a versatile, one-piece, magnetic clip that can hold reading glasses, sunglasses and more on any article of clothing or bag. Made of soft silicone, the stylish yet powerful clip ensures glasses won't fall out, nor will the device damage clothing or scratch expensive eyewear, the company says.

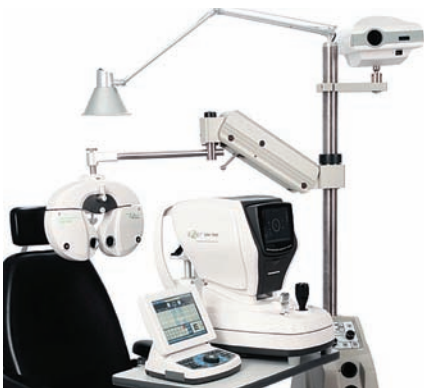
The Lens Friend is available for purchase online ($12.99) in five different colors: black, ivory, light blue, royal blue and red.

Visit www.thelensfriend.com.

Ophthalmic Equipment

All-in-One Refraction

The Ezer Digital Practice 7800 is an all-inclusive refraction system from US Ophthalmic. Making its debut at Vision Expo West, the Ezer Digital Practice 7800 is made up of three devices that are networked to communicate as one unit to improve clinical precision and elevate your level of care, the company says:



• ***ERK-7800 Autorefractor/Keratometer.*** Perform refractometry and keratometry alone or simultaneously, with features such as a measurement range of -25.00D to +22.00D, retro-illumination, an IOL mode and more.

• ***EDR-7800 Digital Refractor.*** Achieve speed without sacrificing precision or the personal touch, the company says. This recently re-engineered device features a full range of built-in, customizable tests, including color blindness and near vision. It features an 8" LCD touch panel and offers 180° of tilt.

• ***ECP-5400 Chart Projector.*** This unit's highly versatile programmable remote control lets you randomly select any of the 31 test charts, or you can use the program feature to create your own user-defined chart sequences. The masking controls allow you to isolate horizontal and vertical lines or individual characters, and the red/green filter can be applied to any chart.

Visit www.usophthalmic.com.

Ophthalmic Lenses

Anti-Glare Lenses for Kids

Crizal Prevencia Kids No-Glare lenses, from Essilor, are now available through managed vision care plans offered by VSP.

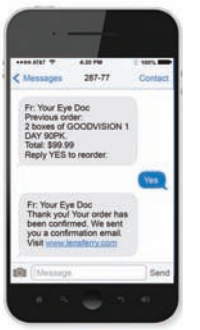
Some light is, of course, necessary for children's vision. Blue-turquoise light helps kids see more clearly, regulates their sleep cycles and supports healthy memory, mood and brain functions, Essilor says. In light of that, Crizal Prevencia Kids is the first no-glare lens specially designed for children to selectively block harmful blue light and UV while allowing beneficial light to reach their eyes.

Visit www.crizal.com.

Contact Lenses

E-commerce Lens Ordering

LensFerry is a new electronic service that allows contact lens wearers to quickly and conveniently order replacement lenses from any manufacturer via their mobile device, tablet or computer for fast shipment to their homes, says CooperVision, which developed LensFerry through its subsidiary WebSystem3.



Your practice sets the price and you receive the sales revenue as if the lenses had been ordered in office. LensFerry further enhances your relationship with the patient, because all communications are customized with your practice's name and/or logo.

If a wearer is not immediately interested in placing a lens order after an in-office appointment, you can send customized follow-up messages through LensFerry to encourage enrollment and online ordering, build wearer loyalty and maintain relationships, CooperVision says.

Now in its US beta release, the service will be part of WebSystem3's EyeCare Prime suite later this year.

Visit www.LensFerry.com. ■

CAN'T WAIT UNTIL NEXT MONTH?

Optometric Physician delivers UP-TO-DATE news and research to your inbox every Monday morning, allowing you to view all of the latest clinical information on a convenient and consistent basis.

Subscribing to Optometric Physician is an efficient and easy way to stay current with all of the information and events going on in the field. To order your free subscription, e-mail: optometricphysician @jobson.com today.

Optometric Physician™

September 2014

■ **17-20.** *Envision Conference 2014.* Hyatt Regency Minneapolis, Minneapolis, Minn. Hosted by: Envision University. CE hours: 23. Keynote: Rebecca Kammer, OD. Email michael.epp@envisionus.com or call (316) 440-1515. Visit www.envisionconference.org.

■ **17-20.** *Vision Expo West 2014.* Sands Expo & Convention Center, Las Vegas, Nev. CE hours: 350+. Hosted by: International Vision Expo and Conference. Visit www.visionexpowest.com.

■ **18.** *IOA Annual Conference.* Crowne Plaza Hotel, Springfield, Ill. Hosted by: Illinois Optometric Association. CE hours: 15. Email Charlene Marsh at ioabb@ioaweb.org or call (217) 525-8012. Visit www.ioaweb.org.

■ **19-20.** *New Mexico Optometric Association Mid-Year Convention.* Inn of the Mountain Gods, Mescalero, NM. Hosted by: New Mexico Optometric Association. CE hours: 8. Email Richard Montoya at newmexicooptometry@gmail.com or call (575) 751-7542. Visit www.newmexicooptometry.org.

■ **19-21.** *KOA 2014 Fall Congress.* Marriott River Center Hotel, Covington, Ky. Hosted by: Kentucky Optometric Association. CE hours: 20. Email Sarah Unger at sarah@kyeyes.org or call (502) 875-3516. Visit www.kyeyes.org.

■ **21.** *CPOS Annual CE Forum.* Hotel Hershey, Hershey, Pa. Hosted by: Central Pennsylvania Optometric Society. CE hours: 6. Email Mary Good, OD, at cposrsvp@gmail.org.

■ **21-23.** *CE in Italy.* Rome, Italy. Hosted by: James Fanelli, OD. CE hours: 12. Key faculty: Leonard Messner, OD, Lorraine Lombardi, PhD, James Fanelli, OD. Email James Fanelli, OD, at jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

■ **25-27.** *CE in Italy.* Florence, Italy. Hosted by: James Fanelli, OD. CE hours: 12. Key faculty: Leonard Messner, OD, Lorraine Lombardi, PhD, James Fanelli, OD, Carlo Pelino, OD. Email James Fanelli, OD, at jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

■ **25-27.** *Idaho Optometric Physicians Annual Congress.* Boise Centre, Boise, ID. Hosted by: Idaho Optometric Physicians CE hours: 19. Email Randy Andregg at randregg7@frontier.com or call (208) 461-0001. Visit idaho.aoa.org.

■ **26-28.** *NOA Fall Convention.* Younes Conference Center, Kearney, Neb. Hosted by: Nebraska Optometric Association. CE hours: 10. Contact Alissa Johnson at noa@assocoffice.net. Call (402) 474-7716. Visit nebraska.aoa.org/fallconvention.

■ **28-30.** *CE in Italy.* Tuscany, Italy. Hosted by: James Fanelli, OD. CE hours: 12. Key faculty: Leonard Messner, OD, James Fanelli, OD, Carlo Pelino, OD. Email James Fanelli, OD, at jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

October 2014

■ **2-4.** *OAOP Fall Conference.* Renaissance Tulsa Hotel & Convention Center, Tulsa, Okla. Hosted by: Oklahoma Association of Optometric Physicians. CE hours: 18. Email Heatherlyn Burton at heatherlyn@oaop.org or call (405) 524-1075. Visit www.oaop.org.

■ **2-5.** *2014 Missouri Optometric Association Annual Conference.* University Plaza Hotel, Springfield, Mo. Hosted by: Missouri Optometric Association. CE hours: 14. Key faculty: Ben Gaddie, OD, Kia Eldred, OD, Sally Bodenhamer, OD. Email Sue Brown at sue@moeyecare.org or call (573) 635-6151. Visit www.moeyecare.org.

■ **9-12.** *GWCO Congress 2014.* Oregon Convention Center, Portland, Ore. Hosted by: Great Western Council of Optometry. CE hours: 70. Key faculty: Charles Brownlow, OD, Ben Gaddie, OD, Jimmy Jackson, OD, David Kading, OD, Robert Prouty, OD, Eric Schmidt, OD, Diana Shechtman, OD, Nancy Torgerson, OD. Email Tracy Oman at gwco@gwco.org or call (503) 654-1062. Visit www.gwco.org.

■ **18-19.** *Orlando Super Weekend.* Nova Southeastern University, Orlando, Fla. Hosted by: Nova Southeastern University. CE hours: 12. Key faculty: Michael Chaglasian, OD, Joseph Sowka, OD. Email Vanessa McDonald at oceaa@nova.edu or call (954) 262-4224. Visit optometry.nova.edu/ce/index.html.

■ **21-25.** *COVD 44th Annual Meeting.* Sheraton San Diego Hotel and Marina, San Diego. Hosted by: College of

Optometrists in Vision Development. Email Jackie Cencer at jcencer@covd.org or call (330) 995-0718. Visit www.covd.org.

November 2014

■ **1.** *Fall Conference.* Hilton Hotel & Towers, Lafayette, LA. Hosted by: Optometry Association of Louisiana. Email Dr. Jim Sanderfur at optla@bellsouth.net or call (318) 335-0675. Visit www.optla.org.

■ **6-9.** *Monterey Symposium 2014.* Monterey Marriott Hotel, Monterey, Calif. Hosted by: California Optometric Association. CE hours: 40+. Key faculty: Melissa Barnett, OD, Jay Binkowitz, Michael Chaglasian, OD, Dickon Chan, A. Paul Chous, MA, OD, George Comer, OD, MBA. Email Rachael Van Cleave at contact@coavision.org or call (916) 441-3990. Visit www.coavision.org.

■ **10.** *AFOS/Academy 2014.* Denver Marriott City Center & Colorado Convention Center, Denver, Colo. Hosted by: Armed Forces Optometric Society & American Academy of Optometry. CE hours: 45. Email Gina Borgognoni at execdir@afos2020.org or call (214) 533-0227. Visit www.afos2020.org.

■ **11.** *Fall 2014 Educational Symposium.* Colorado Convention Center, Denver, Colo. Hosted by: Ocular Nutrition Society. CE hours: 6. Email Jeffrey Anshel at ocularnutritionsociety@gmail.com or call (800) 383-1202. Visit www.ocularnutritionsociety.org.

■ **12-15.** *Academy 2014 Denver.* Colorado Convention Center, Denver. Hosted by: American Academy of Optometry. Email Helen Viksnins at HelenV@aaoptom.org or call (321) 710-3937. Visit www.aaopt.org.

■ **13-15.** *NCSOS Fall Congress.* The Grove Park Inn, Asheville, NC. Hosted by: North Carolina State Optometric Society. CE hours: 18. Email Adrianne Drollette at adrianne@nceyes.org or call (252)237-6197. Visit www.nceyes.org.

■ **16.** *VOSH International Meeting: Embracing Traditions, Expanding Horizons.* Embassy Suites Downtown, Denver, Colo. Hosted by: Volunteer Optometric Services to Humanity (VOSH). To register, visit www.vosh.org.

December 2014

■ **6-7.** *31st Annual Cornea, Contact Lens & Contemporary Vision Care Symposium.* Hosted by: University of Houston College of Optometry. Email Amanda Johnson at ajohnson@optometry.uh.edu or call (713)743-1900.

To list your meeting, please send the details to:
Renee Buddle, Senior Associate Editor
Email: rbuddle@jobson.com
Phone: (610) 492-1005

Advertisers Index

This advertiser index is published as a convenience and not as part of the advertising contract. Every care will be taken to index correctly. No allowance will be made for errors due to spelling, incorrect page number, or failure to insert.

Save the Date

6th annual EAST COAST OPTOMETRIC GLAUCOMA SYMPOSIUM

12 CE Credits
(COPE approval pending)

Loews Hotel
PHILADELPHIA, PA
October 24-25, 2014

Discounted Room Rates: $219 single/ $229 double. Call 888-575-6397 and identify yourself as a participant of the East Coast Optometric Glaucoma Symposium (ECOGS)

12 COPE Credits* for only $225!

MEETING CO-CHAIRS:

MURRAY FINGERET, OD

ROBERT N. WEINREB, MD

Speakers:

Michael Chaglasian, OD • Ben Gaddie, OD
Nils Loewen, MD, PhD • Jonathan Myers, MD

3 Ways to Register

Online: www.revoptom.com/ECOGS2014
Email: Reviewmeetings@jobson.com
Call Lois DiDomenico: 877-451-6514

Brought to you by
REVIEW OF OPTOMETRY

cope® *Approval pending

Partially supported by an educational grant from
Alcon

Merchandise Offered

Equipment and Supplies

Merchandise Offered

Continuing Education

Equipment and Supplies

Storage and Design

Practice For Sale

Continuing Education

Software

Practice For Sale

Professional Opportunities

Professional Opportunities

Products and Services

Why All the Tears?

Here's how to diagnose, and even clear, most lacrimal duct obstructions.

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

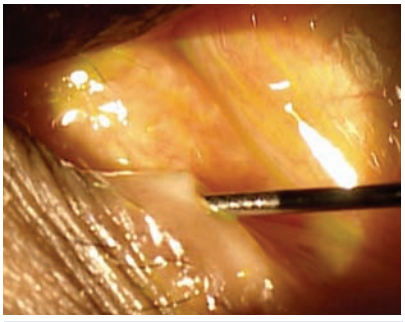
Epiphora is a common sign and symptom that's often a nuisance for our patients. It is usually caused by a blockage located somewhere along the lacrimal excretory system, and leads to an abnormal flow of tears down the cheek. Affected individuals generally complain of constant tearing, while others report intermittent symptoms. Also, epiphora patients typically experience unilateral and/or asymmetric symptoms.

There are several differentials for epiphora, including dry eye disease, conjunctivochalasis, lid abnormalities and/or nasolacrimal obstructions. Patient history, lid evaluation, lacrimal sac palpitation, slit lamp examination, schirmer testing, dye disappearance and Jones I test, lacrimal dilation and irrigation, lower lid taping, nasal speculum exam and radiography will help you most effectively determine the underlying cause.

Try Rolling the Dye

The dye disappearance test followed by lacrimal dilation and irrigation is essential in determining the correct diagnosis. This test will help you evaluate tear lake malposition, tear pump function and punctal stenosis or blockage of the canaliculus, lacrimal sac or the nasolacrimal duct.

To perform the dye disappearance test, instill 1gtt Fluress (fluorescein sodium 0.25% and benoxinate 0.4%, Akorn) into the lower lid cul-de-sac. The dye should drain properly through the lacrimal excretory system after approximately five minutes, indicating a negative (i.e., normal) result. A positive result, however, is confirmed by an increased tear meniscus or frank epiphora, which suggest a blockage somewhere within the drainage system.



Go to www.revoptom.com or scan the QR code at left to see a narrated video of dilation and irrigation.

Test the Flow

Lacrimal dilation and irrigation should follow any positive dye disappearance result. Indications for the procedure include canalicular obstruction, intracanalicular plug removal and canaliculitis. Instruments frequently used for dilation and irrigation include a sterile disposable syringe, lacrimal cannula and saline.

Prior to the procedure, topical anesthetic is instilled into the eye. Some ODs may apply additional anesthesia by holding a soaked cotton-tipped applicator against the puncta for several minutes.

Next, use the index finger of your non-dominant had to gently pull the lid and expose the punctum. The thin-tapered dilator is inserted perpendicularly into the inferior punctum (2mm deep) while the dilator is rolled to expand the puncta. If needed, the punctum can be further stretched by switching to the medium-tapered dilator or by inserting the dilator further into the horizontal canaliculus.

Once the punctum is adequately dilated, the lacrimal cannula is inserted perpendicular to the lid both vertically (2mm deep) and then horizontally (3mm to 4mm deep). Next, apply gentle pressure to the plunger to eject saline into the lacrimal secretory system, which will help remove any obstructions.

A normal result is confirmed when the saline flows freely through the system and the patient feels/tastes the solution in their nose or throat.

If the saline flows out of the inferior puncta and discharges from the superior punctum, a blockage is located at either the common canaliculus or the lacrimal sac. In these cases, further evaluation includes repeating the procedure while occluding the superior punctum with a dilating instrument.

If you observe saline egress from the same punctum you are testing, there is likely a blockage located along the inferior canaliculus. In such instances, a surgical consult may be indicated. ■



The Rick Bay Foundation

for Excellence in Eyecare Education

www.rickbayfoundation.org

Support the Education of Future Healthcare & Eyecare Professionals



About Rick

Rick Bay served as the publisher of The *Review* Group since 1991.

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

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

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

Interested in being a partner with us?

Visit www.rickbayfoundation.org

(Contributions are tax-deductible in accordance with section 170 of the Internal Revenue Code.)

THE RICK BAY FOUNDATION
for Excellence in Eyecare Education

(The Rick Bay Foundation for Excellence in Eyecare Education is a nonprofit, tax-exempt organization under section 501(c)(3) of the Internal Revenue Code.)

Diagnostic **Quiz**

You Ain't Got Time to Bleed

By Andrew S. Gurwood, OD

History

A 54-year-old white female presented with a chief complaint of gradual vision loss in her left eye that had persisted since her most recent examination two years earlier. At that exam, her eye care provider suggested that she had "bleeding in her eye" and needed to see a retinal specialist.

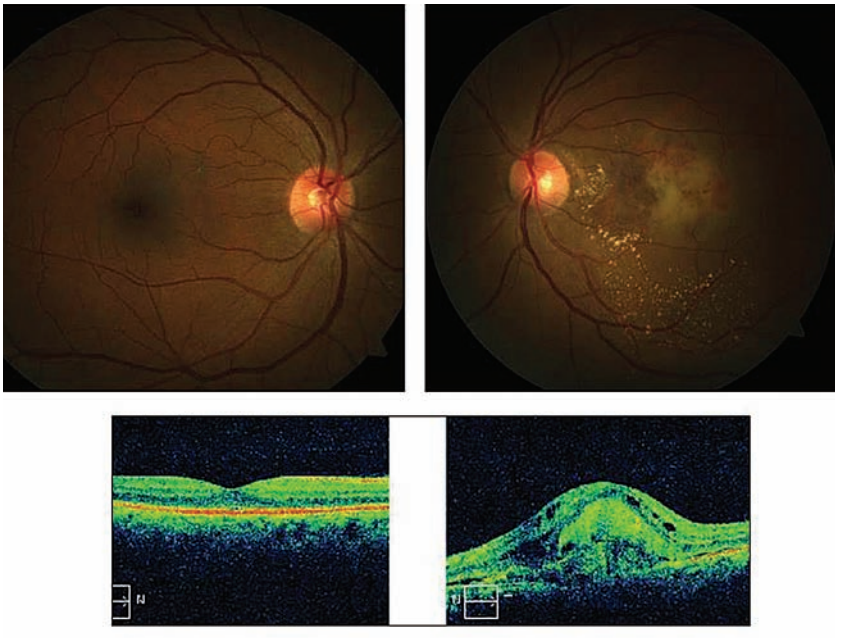
She explained that, after a lengthy delay, she made an appointment with us following a recommendation from her general physician.

Her systemic history was significant for hypertension, myocardial infarction, hypercholesterolemia, gastroesophageal reflux disorder, arthritis and anemia.

Current medications included 5mg amlodipine besylate, 3.125mg carvedilol, 75mg clopidogrel, 100mg losartan, 40mg pantoprazole, 20mg simvastatin and low-dose (81mg) aspirin. She reported no known allergies of any kind.

Diagnostic Data

Her best-corrected visual acuity measured 20/20 OD and 20/300 OS, with no improvement upon pinhole testing. Pupils were equal, round and reactive to light, with no evidence of afferent defect OU.



Fundus and optical coherence tomography images of our 54-year-old patient who reported vision loss in her left eye (OD top/bottom left, OS top/bottom right).

Extraocular muscle movements were full and smooth. Confrontation fields were full to finger counting in each eye, with a blurry and distorted facial Amsler result in the left eye.

Slit lamp examination was remarkable for bilateral corneal arcus. Intraocular pressure measured 14mm Hg OU, and her blood pressure was 107mm Hg/89mm Hg.

Your Diagnosis

How would you approach this case? Does the patient require any additional tests? What is your diagnosis? How would you manage this patient? 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. ■

Retina Quiz Answers *(from page 93):* 1) a; 2) b; 3) a; 4) b; 5) c.

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 81, CONGERS, NY 10920-0081. 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); OUTSIDE USA, CALL (845) 267-3065. OR EMAIL US AT REVOPTOMETRY@CAMBEYWEST.COM. PUBLICATIONS MAIL AGREEMENT NO: 40612608. CANADA RETURNS TO BE SENT TO BLEUCHIP INTERNATIONAL, P.O. BOX 25542, LONDON, ON N6C 6B2.

Why your patients are happy with **Biofinity**

Aquaform® Technology used in Biofinity lenses delivers unmatched balance against these key comfort measures.

Breathability.

High Oxygen Transmissibility:
Biofinity lenses provide superb breathability for daily or extended wear (6 nights/7 days), helping maintain healthier corneal physiology.

Wettability.

Natural and Uniform Wettability:
Biofinity lenses stay moist without additives or surface treatments, helping maximize comfort.

Flexibility.

Optimum Modulus:
Less silicone results in an optimum modulus, creating a softer and more flexible lens.

Biofinity & Biofinity XR | **Biofinity toric** | **Biofinity multifocal**

Impossibly comfortable.

Learn more about how Biofinity makes your patients happy at **coopervision.com/biofinity**

CooperVision®
Live Brightly.®

Because today's world doesn't give your patients' eyes a break...

THIS IS **WHY I CARE**

Choose Alcon Vision Care products
to help your patients remain in contact lenses

PERFORMANCE DRIVEN BY SCIENCE™

Visit myalcon.com for more information on
Alcon Vision Care products.

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

Important information for AIR OPTIX® NIGHT & DAY® AQUA (lotrafilcon A) contact lenses: Indicated for vision correction for daily wear (worn only while awake) or extended wear (worn while awake and asleep) for up to 30 nights. **Relevant Warnings:** A corneal ulcer may develop rapidly and cause eye pain, redness or blurry vision as it progresses. If left untreated, a scar, and in rare cases loss of vision, may result. The risk of serious problems is greater for extended wear vs daily wear and smoking increases this risk. A one-year postmarket study found 0.18% (18 out of 10,000) of wearers developed a severe corneal infection, with 0.04% (4 out of 10,000) of wearers experiencing a permanent reduction in vision by two or more rows of letters on an eye chart. **Relevant Precautions:** Not everyone can wear for 30 nights. Approximately 80% of wearers can wear the lenses for extended wear. About two-thirds of wearers achieve the full 30 nights continuous wear. **Side Effects:** In clinical trials, approximately 3-5% of wearers experience at least one episode of infiltrative keratitis, a localized inflammation of the cornea which may be accompanied by mild to severe pain and may require the use of antibiotic eye drops for up to one week. Other less serious side effects were conjunctivitis, lid irritation or lens discomfort including dryness, mild burning or stinging. **Contraindications:** Contact lenses should not be worn if you have: eye infection or inflammation (redness and/or swelling); eye disease, injury or dryness that interferes with contact lens wear; systemic disease that may be affected by or impact lens wear; certain allergic conditions or using certain medications (ex. some eye medications). **Additional Information:** Lenses should be replaced every month. If removed before then, lenses should be cleaned and disinfected before wearing again. Always follow the eye care professional's recommended lens wear, care, and replacement schedule. Consult package insert for complete information, available without charge from **Alcon** at **(800) 241-5999 or myalcon.com.**

See product instructions for complete wear, care and safety information.

Rx only contact lenses

 1/14 WIC14001JAD

Alcon®
a Novartis company