A Photo Atlas of Conjunctival and Scleral Anomalies, p. 84

Earn 2 CE Credits: Identifying Systemic Sources of Uveitis, **p. 96**

REVIEW® OF OPTOMETRY

November 15, 2016

www.reviewofoptometry.com

FIND YOUR NICHE

With OD ranks on the rise, you can stand out from the crowd—and better serve the public—by developing a specialty within your practice.

In this special series, six pros help you explore opportunities for growth.

REVIEW OF OPTOMETRY
125 YEARS
SERVING THE OPTOMETRIC PROFESSION
ESTABLISHED 1891

Because I know their eyes are prone to discomfort, I prescribe the 1-DAY ACUVUE® MOIST Family.

§ 88% of all BLINK STABILIZED® Design contact lenses were fitted in the first attempt, and 99.5% within 2 trial fittings.

** Based on *in vitro* data. Clinical studies have not been done directly linking differences in lysozyme profile with specific clinical benefits.

* UV-blocking percentages are based on an average across the wavelength spectrum.

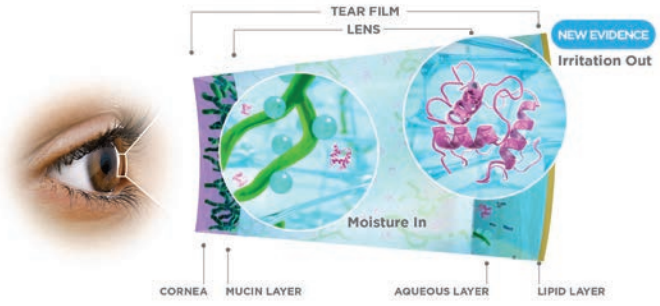
† Helps protect against transmission of harmful UV radiation to the cornea and into the eye.

‡ **WARNING:** UV-absorbing contact lenses are NOT substitutes for protective UV-absorbing eyewear such as UV-absorbing goggles or sunglasses because they do not completely cover the eye and surrounding area. You should continue to use UV-absorbing eyewear as directed. NOTE: Long-term exposure to UV radiation is one of the risk factors associated with cataracts. Exposure is based on a number of factors such as environmental conditions (altitude, geography, cloud cover) and personal factors (extent and nature of outdoor activities). UV-blocking contact lenses help provide protection against harmful UV radiation. However, clinical studies have not been done to demonstrate that wearing UV-blocking contact lenses reduces the risk of developing cataracts or other eye disorders. Consult your eye care practitioner for more information.

EYE-INSPIRED™ Design

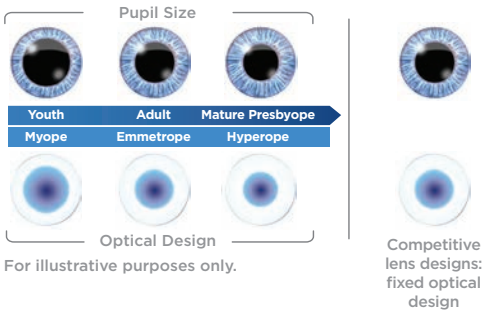
Dual Action Technology helps keep moisture in and irritation out

1-DAY ACUVUE® MOIST Brand Contact Lens Family



- **LACREON® Technology** uses a locked-in wetting agent to create a long-lasting cushion of moisture.
- **New evidence**—Etafilcon A uniquely attracts and retains more lysozyme in its natural state[1],which, along with an INFINITY EDGE™ Design and a low modulus, helps to keep irritation out. **

1-DAY ACUVUE® MOIST MULTIFOCAL



For illustrative purposes only.

Competitive lens designs: fixed optical design

- **INTUISIGHT™ Technology** is the first and only multifocal lens technology that uniquely optimizes the optical design according to age and refractive power
- **A superior vision experience**—for presbyopic patients.

1-DAY ACUVUE® MOIST for ASTIGMATISM

- **BLINK STABILIZED™ Design** harnesses the natural power of a blinking eye, delivering exceptional stability and clear vision for astigmatic patients
- **First fit success**—nearly 90% of the time[§]

for more information visit acuvueprofessional.com

The #1 prescribed daily disposable around the world now satisfies a broader range of patients—available in Sphere, Toric and Multifocal

1-DAY ACUVUE® MOIST BRAND CONTACT LENSES

82% UVA 97% UVB UV BLOCKING*†‡

ACUVUE® Brand Contact Lenses are indicated for vision correction. As with any contact lens, eye problems, including corneal ulcers, can develop. Some wearers may experience mild irritation, itching or discomfort. Lenses should not be prescribed if patients have any eye infection, or experience eye discomfort, excessive tearing, vision changes, redness or other eye problems. Consult the package insert for complete information. Complete information is also available by visiting acuvueprofessional.com, or by calling Johnson & Johnson Vision Care, Inc. at 1-800-843-2020.

Reference: 1. Suwala M, Glasier MA, Subbaraman LN, et al. Quantity and conformation of lysozyme deposited on conventional and silicone hydrogel contact lens materials using an in vitro model. *Eye Contact Lens.* 2007;33(3):138-143.

VOL. 153 NO. 11 ■ NOVEMBER 15, 2016

IN THE NEWS

Researchers have uncovered a unifying pathway involved in the damage to rods and cones, possibly leading to **treatments for blinding diseases such as retinitis pigmentosa and dry AMD.** The study, published in *Cell Reports*, found that defects in a key molecule pathway, NAD, are involved in several diseases of the retina. When they treated damaged photoreceptor cells in mice with a precursor molecule that boosts levels of NAD, **degeneration of the cells ceased and vision was restored**.

Lin JB, Kubota S, Ban N, et al. NAMPT-mediated NAD biosynthesis is essential for vision in mice. Cell Reports. 2016;17(1):69-85.

A new encapsulated cell therapy, known as NT-501 ECT, might be a new **glaucoma treatment** in the pipeline. Currently in phase II trials, NT-501 ECT is a capsule filled with genetically modified human cells that **secrete ciliary neurotrophic factor (CNF)**. The capsule is implanted into the eye, providing a slow release of CNF to help the **retinal ganglion cells resist damage and protect the optic nerve**. The trial will follow participants for two years to track any vision changes.

Goldberg JL. Study of NT-501 encapsulated cell therapy for glaucoma neuroprotection and vision restoration. Phase II trial. Available at https://clinicaltrials.gov/ct2/show/NCT02862938. Accessed October 17, 2016.

The **visual cortex** plays a key role in promoting the **plasticity of innate, spontaneous eye movements**, according to a new study. Researchers studied eye movements in mice and found silencing the visual cortex significantly reduced optokinetic reflex activity, suggesting it is involved in mediating the plasticity between the optokinetic and the vestibulo-ocular reflexes.

Liu B, Huberman AD, Scanziani M. Cortico-fugal output from visual cortex promotes plasticity of innate motor behavior. Nature. October 12, 2016. [Epub].

Retina Changes May Foretell Brain Diseases

Eye exams can offer a window into the central nervous system, according to a new study.

By Bill Kekevian, Senior Editor

New research suggests retinal evidence of diseases of the central nervous system may present prior to changes to the brain, according to a report in *Human Molecular Genetics*. The study, which employed mouse models, posits that eye examinations could serve as a noninvasive screening tool for some brain diseases.

"This particular study found a correlation between retinal and neurologic disease using electroretinography and visual evoked potentials. Other studies have used OCT or fundus autofluorescence as a retinal screening tool for neurologic conditions," says Denise Goodwin, OD, who teaches on neuro-ophthalmic disease at Pacific University. "Ultimately, the retina is an extension of neurologic tissue in the brain; therefore, the use of the retina to screen for these conditions makes sense. The same processes that cause damage in the cortex may contribute to death of the retinal nerve fiber layer."

Using functional testing methods, such as electroretinography and visual evoked potentials, investigators from the University of Eastern Finland evaluated several disease types in mouse models, including Huntington's disease, Alzheimer's and neuronal ceroid lipofuscinosis (NCL). All three disease types showed functional impairments prior to anatomical changes. For instance, in the Huntington's disease group, day and color vision changes were recorded while the mouse was presymptomatic, the report says. The Alzheimer's group showed changes to their night vision, and the NCL group demonstrated impaired retinal ganglion cell function, similar to that seen in age-related macular degeneration.

According to Dr. Goodwin, a number of studies are showing a correlation between the retina and degenerative brain conditions.

Relying on these noninvasive measures may provide an opportunity for less expensive and quicker diagnostic methods, she adds.

"Because neuronal damage can occur well before symptoms of cognitive decline, early detection can have a critical impact in the lives of these patients, Dr. Goodwin says. "It is exciting to think that something as basic as a vision examination could allow for earlier detection and diagnosis of these disabling neurologic diseases."

Leinonen H, Rossi M, Salo A, et al. Lack of P4H-TM in mice results in age-related retinal and renal alterations. Human Molecular Genetics. July 27, 2016. [Epub ahead of print]. Available at http://hmg.oxfordjournals.org/content/early/2016/08/17/hmg.ddw228. Accessed October 14, 2016.

Introducing the Keeler Z Series Slit Lamp & Digital Tonometer

Keeler Optics...says it all.

The Z Series Slit Lamp is the latest line from Keeler featuring legendary Keeler optics housed in a stylish, contemporary design.

Your choice of 3 or 5 step magnification option in a standard, digital ready, or comprehensive digital capture system.

Check out some of these great features:

- Converging or parallel binoculars
- 14mm Slit length / maximum aperture
- X 6 to x 40 (5 step) or X 10 to X 25 (3 step) magnifications
- Blue, red-free, clear, neutral density and diffuser filters
- 360 degree continuous slit rotation
- Integrated yellow barrier filter
- 1m Square aperture for Uveitis assessment
- LED illumination
- Illumination control mounted next to joystick
- 3 year warranty

Learn more at www.keelerusa.com

Order now and receive a limited edition bronze D-KAT tonometer.

Contact your preferred Keeler distributor for details.

Keeler

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

Is a Corneal Scar Treatment on the Horizon?

With severe corneal trauma, scar tissue and vision loss are often the unfortunate byproducts of the healing process. Current treatments, including topical steroids and surgical modalities such as keratoplasty and limbal stem cell and amnion transplantation, are subject to limitations such as increased risks for infection and transplant rejection.

In recent years, research suggests mesenchymal stem cells (MSC) have the ability to clear the corneal opacity that often forms following injury. Until recently, how the cells offer these benefits remained elusive.

A new study, published in *Stem Cell Reports*, sheds light on the mechanism of action that allows MSCs to clear cornea opacity following trauma. Researchers from Schepens Eye Research Institute of Massachusetts Eye and Ear found that hepatocyte growth factor (HGF), secreted by MSCs, is the key to their ability to encourage wound healing by interfering with the inflammatory response. Although the findings suggest the efficacy of HGF-based treatments to restore vision, according to a press release, the results were found use an animal model of corneal trauma, and more research is necessary to learn more about the process in the human eye.

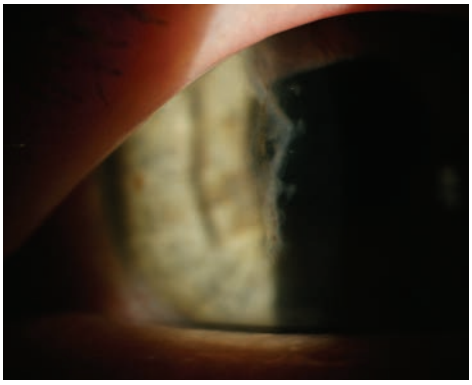


Photo: Elyse L. Chaglasian, OD, and Gregg Eric Russell, OD

Corneal scarring, as seen here, was nearly eliminated in MSC-treated animal corneas following injury.

HGF was applied topically to an injured animal model eye twice daily for up to seven days post-injury, and investigators used slit-lamp biomicroscopy to monitor corneal opacity. Corneas of HGF-treated mice showed reduced opacity at day five and, according to the researchers, near complete restoration of transparency on day seven, compared with the control group.

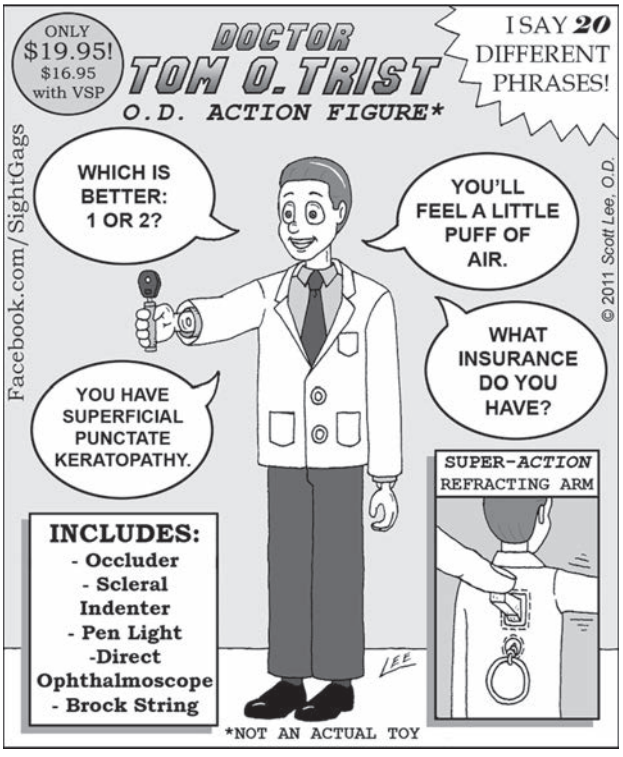
"Our results show that mesenchymal stem cells, in an inflamed environment, secrete high levels of HGF, which inhibit scar formation and restore corneal transparency," senior author of the study, Sunil L. Chauhan, PhD, said in a press release. According to the researchers, MSC cells, when injected into the mice after being stimulated by immune system proteins to secrete HGF, migrated to the source of corneal injury via the bloodstream and induced corneal healing.

But when the researchers suppress HGF gene expression using small interfering RNAs, Dr. Chauhan said in the press release, "the stem cells lose their capacity to inhibit scar formation."

The researchers are hopeful for the implications the results have on future treatment options for severe corneal trauma. "That HGF alone can restore corneal transparency is highly significant and has tremendous translational implications for developing new treatment modalities," Dr. Chauhan said.

Mittal SK, Omoto M, Amouzegar A, et al. Restoration of corneal transparency by mesenchymal stem cells. Stem Cell Reports. 2016. [Epub ahead of print].

Sight Gags By Scott Lee, OD



The most comprehensive GP lens care system available.

Introducing LacriPure

Menicon's new rinsing and insertion saline solution.

Indicated for use with soft, hybrid and rigid gas permeable lenses, LacriPure is a sterile, non-preserved saline which provides an alternative to tap water rinsing.

Packaged in a 5ml unit-dose vial, LacriPure has been cleared as a scleral lens insertion solution and provides the patient with exceptional sterility.

Menicon LacriPure joins **Menicon Unique pH®** multi-purpose solution and **Menicon PROGENT** protein remover to complete the industry's most comprehensive GP lens care system.

Menicon

800-636-4266 | meniconamerica.com | information@menicon.com

Are Politics Seeping Into Your Exam Lane?

Are you confusing the exam lane with the voting booth? According to a recent study Yale researchers published in *Proceedings of the National Academy of Sciences* (*PNAS*), primary care doctors who fall on different sides of the aisle treat some of the more politicized health issues differently depending on their party affiliation. For instance, researchers say, Republicans rated "likely" the prospect of discussing the mental health consequences of abortion whereas Democrats expressed concern about the dangers of keeping a firearm in a home with a small child. Doctors on the left, according to the study, are also less concerned about marijuana use in otherwise healthy patients.

"We linked the records of over 20,000 primary care physicians in 29 states to a voter registration database, obtaining the physicians' political party affiliations. We then surveyed a sample of Democratic and Republican primary care physicians," the study reads.

Respondents were asked to consider nine patient vignettes and rate the seriousness of each issue as well as their likelihood of engaging with patients about them. The results show remarkable difference only on the three politicized issues (abortion, marijuana use and firearms). Doctors were relatively in agreement over the nonpoliticized issues presented, such as tobacco and alcohol use and obesity.

Hersh ED, Goldenberg MN. Democratic and Republican physicians provide different care on politicized health issues. Proceedings of the National Academy of Sciences. October 3, 2016. [Epub ahead of print]. Available at www.pnas.org/content/early/2016/09/28/1606609113. Accessed October 14, 2016.

Cat-Scratch Fever on the Rise

Optometrists practicing in the Southern United States, or those who see patients between the ages of five and nine, might be more likely to encounter the ocular side effects of cat-scratch disease (CSD), a new study suggests. Investigators from the Centers for Disease Control and Prevention and Emory University reviewed insurance claim databases from 2005 to 2013, and discovered approximately 12,000 outpatients were given a CSD diagnosis, and 500 inpatients were hospitalized for the disease.

The study, recently published in *Emerging Infectious Diseases*, found the incidences of CSD were highest in southern states and among children between ages five

(*continued on pg. 10*)

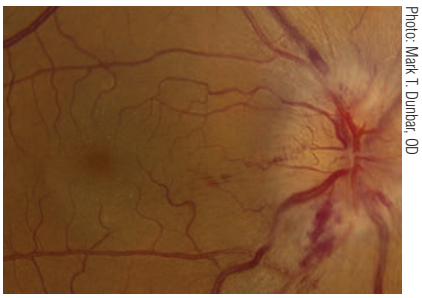


Photo: Mark T. Dunbar, OD

If you look closely at the macula in this fundus photo, you'll see exudate in a "macular star" pattern, a classic sign of cat-scratch disease.

REVIEW® OF OPTOMETRY

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

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

REGIONAL SALES MANAGER
Michael Hoster
(610) 492-1028 • mhoster@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: (845) 267-3065

CIRCULATION
PO Box 81
Congers, NY 10920
Tel: (Toll Free): (877) 529-1746
Outside US: (845) 267-3065

jmi

CEO, INFORMATION SERVICES GROUP
Marc Ferrara

SENIOR VICE PRESIDENT, OPERATIONS
Jeff Levitz

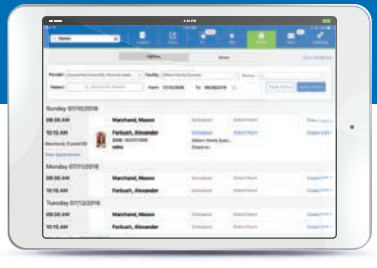
VICE PRESIDENT, HUMAN RESOURCES
Tammy Garcia

VICE PRESIDENT, CREATIVE SERVICES & PRODUCTION
Monica Tettamanzi

CORPORATE PRODUCTION DIRECTOR
John Anthony Caggiano

VICE PRESIDENT, CIRCULATION
Emelda Barea

That's the sound of you hitting your forehead when you realize…



…you should've made the switch to Eyefinity® EHR years ago. With the fewest clicks of any EHR in the industry, Eyefinity EHR helps you fly through exams and enhance your workflow. Discover what a difference it makes to click less and practice more.

For more information visit www.eyefinity.com/smack.

eyefinity®

Cat-Scratch

(*continued from pg. 8*)
and nine. Children younger than 14 accounted for 32.5% of all diagnoses and, although women and girls accounted for 62.0% of outpatient and 55.6% of inpatient diagnoses, inpatients were more likely than outpatients to be male and between the ages of 50 and 64.[1]

CSD is a zoonosis caused by *Bartonella henselae*, which is spread among cats by fleas; transmission to humans occurs via cat scratches, and possibly bites. While the predominant clinical feature of CSD is lymphadenopathy proximal to the site of the scratch or bite, some patients experience ocular effects such as neuroretinitis or parinaud oculoglandular syndrome. Other serious manifestations include osteomyelitis, encephalitis or endocarditis.[2] While patients with typical signs of and history for CSD can be given a presumptive clinical diagnosis, diagnostic tests such as serology, PCR and culture can be useful for confirming the diagnosis when atypical.

Although the number of American households with cats has increased in recent decades, suggesting practitioners might see more of this diagnosis, the study found the annual incidence of outpatient CSD diagnoses steadily declined from 2005 to 2013. Still, optometrists should be aware of the ocular side effects of CSD and be prepared to treat these patients, and children in particular.[3] ■

1. Nelson CA, Saha S, Mead PS. Cat scratch disease in the United States, 2005–2013. Emerg Infect Dis. 2016 Oct;22(10). [Epub ahead of print].
2. Florin TA, Zaoutis TE, Zaoutis LB. Beyond cat scratch disease: widening spectrum of Bartonella henselae infection. Pediatrics. 2008;121:e1413–25.
3. American Pet Product Manufacturers Association. New survey reveals pet ownership at all-time high. http://media.americanpetproducts.org/press.php?include=144262. Accessed October 19, 2016.

Down, Boy.

Help Tame Postoperative Ocular Inflammation and Pain With LOTEMAX® GEL

Indication

LOTEMAX® GEL (loteprednol etabonate ophthalmic gel) 0.5% is indicated for the treatment of post-operative inflammation and pain following ocular surgery.

Important Safety Information about LOTEMAX® GEL

- LOTEMAX® GEL is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.
- Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. If this product is used for 10 days or longer, IOP should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.
- Use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation and occurrence of perforations in those with diseases causing corneal and scleral thinning. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, and where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infection. In acute purulent conditions, steroids may mask infection or enhance existing infection.
- Use of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.
- Patients should not wear contact lenses when using LOTEMAX® GEL.
- The most common ocular adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%) and foreign body sensation (2%).

Please see brief summary of Prescribing Information on adjacent page.

LOTEMAX® GEL
loteprednol etabonate
ophthalmic gel 0.5%

BAUSCH + LOMB

BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to prescribe Lotemax Gel safely and effectively. See full prescribing information for Lotemax Gel.

Lotemax (loteprednol etabonate ophthalmic gel) 0.5%

Rx only
Initial Rx Approval: 1998

INDICATIONS AND USAGE

LOTEMAX is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

DOSAGE AND ADMINISTRATION

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

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

CONTRAINDICATIONS

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

WARNINGS AND PRECAUTIONS

Intraocular Pressure (IOP) Increase

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

Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing

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

Bacterial Infections

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

Viral Infections

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

Fungal Infections

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

Contact Lens Wear

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

ADVERSE REACTIONS

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects: Pregnancy Category C.

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

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

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

Nursing Mothers

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

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment Of Fertility

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

PATIENT COUNSELING INFORMATION

Administration

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

Risk of Contamination

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

Contact Lens Wear

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

Risk of Secondary Infection

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

Bausch & Lomb Incorporated
Tampa, Florida 33637 USA

US Patent No. 5,800,807

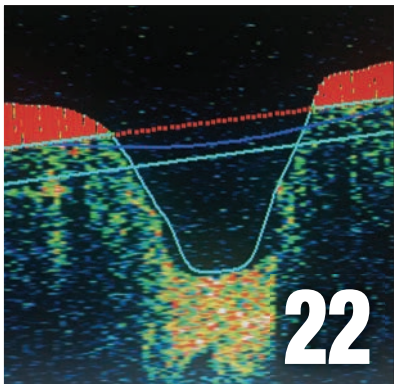
US/LGX/15/0042
Based on 9269100-9269200 Revised: 9/2012

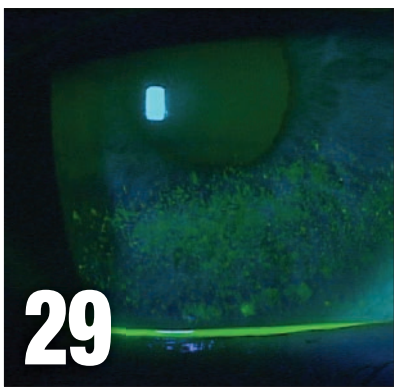
Contents

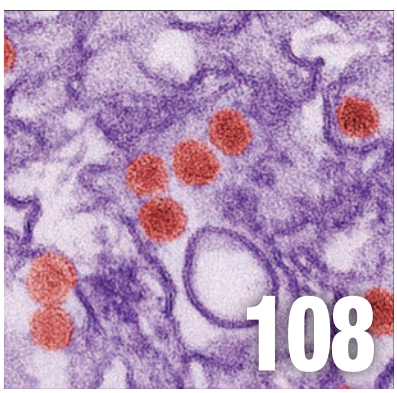
Review of Optometry
November 2016

Departments

Review of Optometry November 2016







On The Web »
and more

Check out our multimedia and continuing education online at: ***www.reviewofoptometry.com***

Digital Edition

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

Go to www.reviewofoptometry.com and click on the digimag link 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

Look for augmented content and special offers from *Review* and our advertisers. Specified pages work in conjunction with your smartphone or other mobile device to enhance the experience. With Layar, interactive content leaps off the page!

Step1: Download the free Layar app for iPhone or Android.

Step 2: Look for pages with the Layar Logo. layar INTERACTIVE PRINT

Step 3: Open the Layar app, hold the phone above the page and tap to scan it. Hold the phone above the page to view the interactive content.

The first 150 app downloads and completed forms will be entered into a drawing for a complimentary registration to one of *Review*'s 14-hour CE meetings, valued at $495.

xiidra™
(lifitegrast
ophthalmic solution)5%

Well this CHANGES

Xiidra is a lymphocyte function-associated antigen-1 (LFA-1) antagonist, the first medication in a new class of drugs.[1]

Check it out at Xiidra-ECP.com

Reference: 1. FDA approves new medication for dry eye disease. FDA News Release. July 2016. *http://www.fda.gov/newsevents/newsroom/pressannouncements/ucm510720.htm*. Accessed July 12, 2016.

THïINGS

The first prescription eye drop FDA-approved to treat both the signs and symptoms of Dry Eye Disease

Indication

Xiidra™ (lifitegrast ophthalmic solution) 5% is indicated for the treatment of signs and symptoms of dry eye disease (DED).

Important Safety Information

In clinical trials, the most common adverse reactions reported in 5-25% of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.

To avoid the potential for eye injury or contamination of the solution, patients should not touch the tip of the single-use container to their eye or to any surface.

Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.

Safety and efficacy in pediatric patients below the age of 17 years have not been established.

For additional safety information, see accompanying Brief Summary of Safety Information on the following page and Full Prescribing Information on Xiidra-ECP.com.

Shire

xiidra™
(lifitegrast
ophthalmic solution)5%

Rx Only

BRIEF SUMMARY:
Consult the Full Prescribing Information for complete product information.

INDICATIONS AND USAGE
Xiidra™ (lifitegrast ophthalmic solution) 5% is indicated for the treatment of the signs and symptoms of dry eye disease (DED).

DOSAGE AND ADMINISTRATION
Instill one drop of Xiidra twice daily (approximately 12 hours apart) into each eye using a single use container. Discard the single use container immediately after using in each eye. Contact lenses should be removed prior to the administration of Xiidra and may be reinserted 15 minutes following administration.

ADVERSE REACTIONS
Clinical Trials Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in clinical studies 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 five clinical studies of dry eye disease conducted with lifitegrast ophthalmic solution, 1401 patients received at least 1 dose of lifitegrast (1287 of which received lifitegrast 5%). The majority of patients (84%) had ≤3 months of treatment exposure. 170 patients were exposed to lifitegrast for approximately 12 months. The majority of the treated patients were female (77%). The most common adverse reactions reported in 5-25 % of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.

USE IN SPECIFIC POPULATIONS
Pregnancy
There are no available data on Xiidra use in pregnant women to inform any drug associated risks. Intravenous (IV) administration of lifitegrast to pregnant rats, from pre-mating through gestation day 17, did not produce teratogenicity at clinically relevant systemic exposures. Intravenous administration of lifitegrast to pregnant rabbits during organogenesis produced an increased incidence of omphalocele at the lowest dose tested, 3 mg/kg/day (400-fold the human plasma exposure at the recommended human ophthalmic dose [RHOD], based on the area under the curve [AUC] level). Since human systemic exposure to lifitegrast following ocular administration of Xiidra at the RHOD is low, the applicability of animal findings to the risk of Xiidra use in humans during pregnancy is unclear.

Animal Data
Lifitegrast administered daily by intravenous (IV) injection to rats, from pre-mating through gestation day 17, caused an increase in mean preimplantation loss and an increased incidence of several minor skeletal anomalies at 30 mg /kg /day, representing 5,400-fold the human plasma exposure at the RHOD of Xiidra, based on AUC. No teratogenicity was observed in the rat at 10 mg /kg /day (460-fold the human plasma exposure at the RHOD, based on AUC). In the rabbit, an increased incidence of omphalocele was observed at the lowest dose tested, 3 mg /kg /day (400-fold the human plasma exposure at the RHOD, based on AUC), when administered by IV injection daily from gestation days 7 through 19. A fetal No Observed Adverse Effect Level (NOAEL) was not identified in the rabbit.

Lactation
There are no data on the presence of lifitegrast in human milk, the effects on the breastfed infant, or the effects on milk production. However, systemic exposure to lifitegrast from ocular administration is low. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for Xiidra and any potential adverse effects on the breastfed child from Xiidra.

Pediatric Use
Safety and efficacy in pediatric patients below the age of 17 years have not been established.

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Carcinogenesis: Animal studies have not been conducted to determine the carcinogenic potential of lifitegrast. *Mutagenesis:* Lifitegrast was not mutagenic in the *in vitro* Ames assay. Lifitegrast was not clastogenic in the *in vivo* mouse micronucleus assay. In an *in vitro* chromosomal aberration assay using mammalian cells (Chinese hamster ovary cells), lifitegrast was positive at the highest concentration tested, without metabolic activation. *Impairment of fertility:* Lifitegrast administered at intravenous (IV) doses of up to 30 mg/kg/day (5400-fold the human plasma exposure at the recommended human ophthalmic dose (RHOD) of lifitegrast ophthalmic solution, 5%) had no effect on fertility and reproductive performance in male and female treated rats.

Shire

Manufactured for: Shire US Inc., 300 Shire Way, Lexington, MA 02421.

For more information, go to www.Xiidra.com or call 1-800-828-2088.

Last Modified: 07/2016 S13681

REVIEW OF OPTOMETRY

PRINTED IN USA

FOUNDING EDITOR, FREDERICK BOGER
1891-1913

EDITORIAL OFFICES
11 CAMPUS BLVD., SUITE 100
NEWTOWN SQUARE, PA 19073

WEBSITE • WWW.REVIEWOFOPTOMETRY.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

MANAGING EDITOR • REBECCA HEPP
(610) 492-1005 • RHEPP@JOBSON.COM

SENIOR EDITOR • BILL KEKEVIAN
(610) 492-1003 • BKEKEVIAN@JOBSON.COM

ASSOCIATE EDITOR • MICHAEL RIVIELLO
(610) 492-1021 • MRIVIELLO@JOBSON.COM

ASSOCIATE EDITOR • ADRIENNE TARON
(610) 492-1005 • ATARON@JOBSON.COM

SPECIAL PROJECTS EDITOR • JILL HOFFMAN
(610) 492-1037 • JHOFFMAN@JOBSON.COM

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

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

EDITORIAL BOARD

CHIEF CLINICAL EDITOR • PAUL M. KARPECKI, OD

ASSOCIATE CLINICAL EDITORS • JOSEPH P. SHOVLIN, OD; ALAN G. KABAT, OD; CHRISTINE W. SINDT, OD

DIRECTOR OPTOMETRIC PROGRAMS • ARTHUR EPSTEIN, OD

CLINICAL & EDUCATION CONFERENCE ADVISOR
PAUL M. KARPECKI, OD

CASE REPORTS COORDINATOR • ANDREW S. GURWOOD, OD

CLINICAL CODING EDITOR • JOHN RUMPAKIS, OD, MBA

CONSULTING EDITOR • FRANK FONTANA, OD

COLUMNISTS

CHAIRSIDE • MONTGOMERY VICKERS, OD

CLINICAL QUANDARIES • PAUL C. AJAMIAN, OD

CODING CONNECTION • JOHN RUMPAKIS, OD

CORNEA & CONTACT LENS Q+A • JOSEPH P. SHOVLIN, OD

DIAGNOSTIC QUIZ • ANDREW S. GURWOOD, OD

FOCUS ON REFRACTION • MARC TAUB, OD; PAUL HARRIS, OD

GLAUCOMA GRAND ROUNDS • JAMES L. FANELLI, OD

NEURO CLINIC • MICHAEL TROTTINI, OD; MICHAEL DELGIODICE, OD

OCULAR SURFACE REVIEW • PAUL M. KARPECKI, OD

RETINA QUIZ • MARK T. DUNBAR, OD

REVIEW OF SYSTEMS • CARLO J. PELINO, OD; JOSEPH J. PIZZIMENTI, OD

SURGICAL MINUTE • DEREK N. CUNNINGHAM, OD; WALTER O. WHITLEY, OD, MBA

THERAPEUTIC REVIEW • JOSEPH W. SOWKA, OD; ALAN G. KABAT, OD

URGENT CARE • RICHARD B. MANGAN, OD

JOBSON MEDICAL INFORMATION LLC

Outlook

By Jack Persico, Editor-in-Chief

Double Vision

Can patients see you as both a generalist *and* a specialist? With a little finesse, it can be done.

Optometry used to have one, and only one, specialty: vision correction. In the profession's earliest years, the precision and attention to detail practitioners brought to the task of refracting patients and fitting glasses was a mark of pride, and indeed a defining trait. It elevated the nascent field and created for its practitioners a distinct identity head and shoulders above the dubious reputation of the "spectacle peddlers" who came before them. Any huckster with a trunk full of wares could sell you a pair of glasses—only an optometrist had truly mastered the art.

But no sooner had optometry created a clearly defined public persona than it began diluting it. The push for the legal authority to use diagnostic agents, then therapeutics and, lately, minor surgical procedures confused the public, creating ambiguity about what defines an optometrist. (This magazine deserves a share of the credit or blame for that—we started publishing articles on disease diagnosis back in the 1930s.)

Optometry's transition from *vision specialists* to *primary eye care providers* may have been a messy one, but it was necessary. There's simply too many people in need of eye health services for ophthalmologists to shoulder it all. The current working relationship between optometry and ophthalmology allows ODs to provide the lion's share of primary eye care while feeding surgical patients to high-volume MD practices.

The optometry profession once again has a pretty clear conception of itself and its public face.

But is another schism coming? Where once optometry's scope of practice was limited by legislatures, now it may well be limited by its own practitioners, as a strategic choice.

Responding to both clinical need and business opportunity, ODs are increasingly looking to carve out a niche for their practices rather than aspiring to be the generalists of the eye. That's the model in ophthalmology, where retina specialists limit their practice to the posterior segment, glaucoma specialists confine their interests to that field, and so on. The Subspecialty Day events at the Academy of Ophthalmology annual meeting are among the most popular—and the most segregated. Will optometry splinter in the same manner? Should it?

Some ODs may well prefer to narrow their scope with such conviction that they turn away patients outside their purview. For most, however, the best course is to augment a general optometry practice with a special interest that adds to it without redefining it.

That balancing act is the focus of this issue's series on specialties in optometry. We invited six ODs who were able to create these dual identities to describe the clinical and practice management changes they had to make. Each presents a model of specialty practice for you to ponder. The choice is yours—including the choice to contentedly remain a generalist. ■

Oh, How I Wish

You can't always get what you want, but maybe one day you will meet a genie in a bottle. Be prepared with your list of demands. **By Montgomery Vickers, OD**

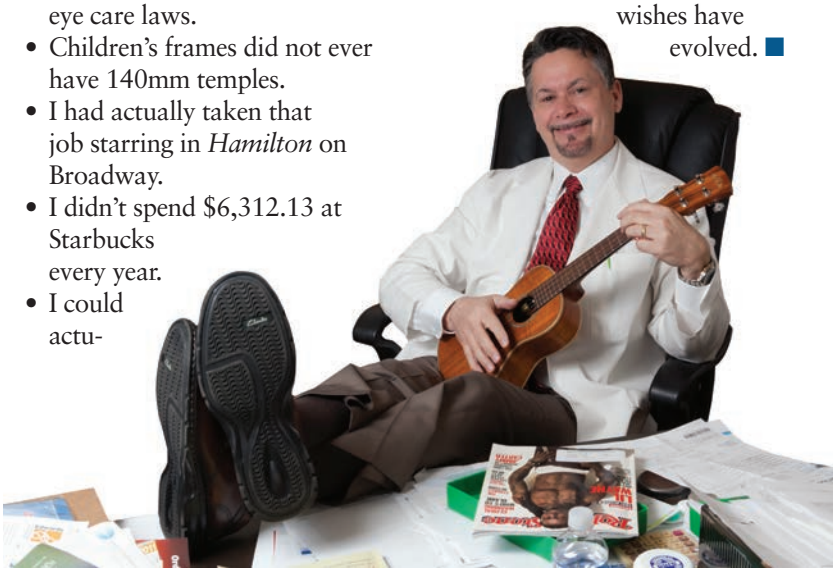
Throughout our careers, we all use the phrase "I wish…" more than 186 times a year, and that's just during office hours. You can only imagine how many times we say it after work, like when the lawnmower won't start or when our spouse wants to visit Mom.

Most of what we wish for is just spur of the moment hogwash, like I wish I hadn't upsized my burger meal. Sometimes, we wish for things that are good, like wishing that nice lady in the chair did not have macular drusen; sometimes we wish for things that are, well, not so good.

I like to collect wishes, write them down and study them later, which is always instructive. Here are some examples. I wish…

- There was a way to reverse LASIK on a 45-year-old computer programmer who was plano OD and -1.25 OS before he had the OS corrected.
- Multifocal lenses beeped when they were inside out.
- Children of staff members would quit getting sick every Sunday night.
- I would explain astigmatism and the patient would not look at me like, "huh?"
- I could be a retinal subspecialist for one day so I could retire.
- Our contact lens rep would not want to meet to introduce me to a revolutionary new contact lens design.
- Doctors who advertise free eye exams would spontaneously combust.
- Somebody would actually give a CE lecture instead of reading slides for two hours.
- During this CE, the lecturer would not say, "Stop me at any time if you have a question."
- I would invent a cure for pinguecula.
- My staff understood that my open door policy does not mean they can speak to me during the day.
- Vision plans wouldn't tell patients they get a free exam every other year, but instead told them they get 50% off every year.
- All optometric graduates would be required to own their own practices for the first five years of their careers.
- All state legislators would have to prove they can pronounce *glaucoma* before voting on any eye care laws.
- Children's frames did not ever have 140mm temples.
- I had actually taken that job starring in *Hamilton* on Broadway.
- I didn't spend $6,312.13 at Starbucks every year.
- I could actually read the fine print on my business card.
- Patients would understand that when they say "I can't see nothing," it actually means they are saying "I can see everything."
- Certain patients would understand that *daily disposable* does not, in fact, mean *monthly extended wear*.
- My wife would understand it's not my fault when she's late for work if she told me to set the alarm for 7am when she has to be at work at 6:30am (I only admit this because she never reads *Chairside*).

I have many, many more wishes I could share—and some that should probably never be shared. I can tell you this much: When I was young, the Dallas Cowboy cheerleaders were involved. Now, it's all about Liliane Bettencourt. I guess my wishes have evolved. ■



THAT'S WHAT MAKES IT ONE OF THE BEST.

Share your story and show your peers and patients why your eye care practice is one of the best. Receive the recognition you deserve and help elevate excellence in the profession.

Submit your practice for a chance to be named one of the 2016 Best Practices at **eyecarebestpractices.com**

BEST PRACTICES
RECOGNITION FOR EXCELLENCE IN EYE CARE
2016

AN INITIATIVE OF CooperVision®

More Than Meets the Eye

When a patient's third nerve palsy resolves, the findings on follow up reveal panuveitis secondary to systemic disease. **By Michael Trottini, OD, and Michael DelGiodice, OD**

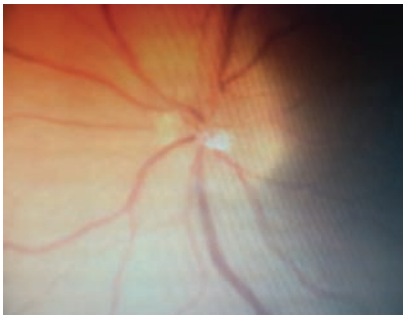
A 50-year-old white male presented emergently for torsional diplopia in left gaze, left upper eyelid drooping and severe stabbing pain around the left orbit for one week. His medical and ocular histories were unremarkable, and he was not taking any medications. He reported no drug allergies and his family and social histories were unremarkable.

His best-corrected visual acuities were 20/20 in each eye. On primary gaze, he showed mild ptosis and exotropia of the left eye. Ocular motilities were full in the right eye and minimally limited in supraduction and adduction in the left eye. The interpalpebral apertures were 6mm OD and 4mm OS. Pupils were equal, round and reactive to light with no afferent defect. We conducted a neurologic exam, and additional testing revealed no associated cranial nerve involvement.

IOPs measured 16mm Hg OU. The anterior segment exam was unremarkable. Fundus exam showed healthy lenticular, vitreous, vascular and retinal structures. The optic nerves showed a cup-to-disc ratio of 0.15 with good perfusion and healthy, distinct margins.

Initial Diagnosis

Based on symptoms and clinical findings, we diagnosed him with partial left third nerve palsy. The presence of a partial third nerve palsy in a patient younger than 60 without vascular risk factors prompted us to order emergent erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP) to rule out vasculitis, as well as contrast-enhanced MRI and angiography to discount an aneurysm of the posterior communicating artery. Both were unremarkable.



Follow-up revealed blurred margins of the left optic nerve.

In the absence of intracranial pathologies and vasculitis, we leaned towards a diagnosis of small vessel disease, with the periorbital pain secondary to ischemia. We scheduled him for evaluation with his general physician and comanaging neurologist. A physical exam and laboratory studies revealed undiagnosed primary hypertension and hyperlipidemia. He was started on the appropriate medication and aspirin.

The Plot Thickens

A follow-up exam one month later revealed complete resolution of the third nerve palsy. However, his anterior segment exam was remarkable for mild diffuse ciliary injection and grade 1 anterior chamber cells of the left eye. Dilated fundus exam revealed anterior vitreous cells, hemorrhagic edema of the optic nerve, choroidal folds within the macula, and multiple midperipheral detachments of both the neurosensory retina and retinal pigment epithelium (RPE) of the left eye—all consistent with a diagnosis of panuveitis.

We referred him for fluorescein angiography, which confirmed our findings. Visual fields revealed mild enlargement of the physiologic blind spot of the affected eye.

Given the recent clinical findings and previous partial third nerve palsy, we ordered the following tests: complete blood count with differentials, comprehensive metabolic panel, ESR, CRP, fluorescent treponemal antibody absorption, Lyme antibody, cytoplasmic antineutrophil cytoplasmic antibodies, perinuclear anti-neutrophil cytoplasmic antibodies, rheumatoid factor, quantiferon-TB gold, angiotensin-converting enzyme and lysozyme. Additionally, we ordered a CT of the chest to discount lung infiltrates, which can occur in tuberculosis, sarcoidosis and granulomatosis with polyangiitis. Results were unremarkable for all tests.

In the absence of laboratory-confirmed systemic disease, the most common cause of panuveitis with multiple serous neurosensory detachments is Vogt-Koyanagi-Harada (VKH) syndrome. The patient was referred back to the neurologist for lumbar puncture and cerebrospinal fluid (CSF) analysis; results confirmed leukocytosis (increased white blood cell count).

Not Just Eye Drops,
Dry Eye Therapy™

When used together, TheraTears® products are proven to improve the signs and symptoms of Dry Eye Disease.[1]

RESTORES
EYES NATURAL BALANCE®

+

CLEANSES
EYELIDS AND LASHES

+

NOURISHES
EYES TO PROMOTE HEALTHY TEARS*

TheraTears®
Dry Eye Therapy
Lubricant Eye Drops

TheraTears®
SteriLid®
Eyelid Cleanser

TheraTears®
Eye Nutrition
1200mg Omega-3 Supplement

thera tears®
THERAPY FOR YOUR EYES®

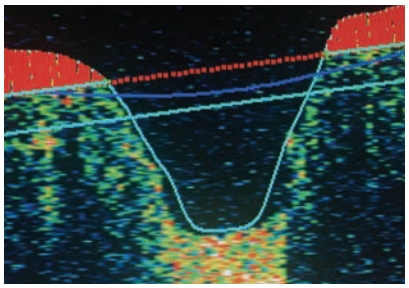
DRY EYE THERAPY

theratears.com

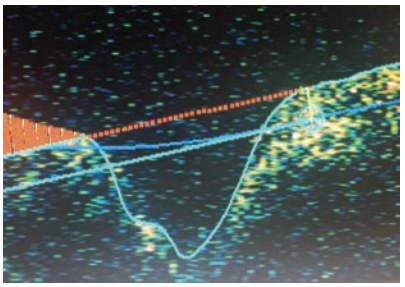
***These statements have not been evaluated by the FDA. This product is not intended to diagnose, treat, cure, or prevent any disease.**

Reference: 1. Srinivasan S, Ngo W, Jones L. The relief of dry eye signs and symptoms using a combination of lubricants, lid hygiene, and ocular nutraceuticals. Poster presented at: ARVO annual meeting; April 2015; Denver, CO.

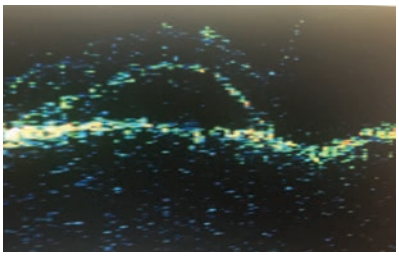
AKORN Consumer Health



Normal nerve fiber OD.



Nerve fiber layer edema OS.



OS midperipheral detachments of the neurosensory retina and RPE.

Based on the revised criteria, we made a diagnosis of probable VKH. The patient was started on 80mg of prednisone and 150mg of ranitidine.

Currently, the panuveitis has resolved and the prednisone was tapered. The patient is taking 40mg of prednisone daily and will start subcutaneous methotrexate injections to reduce the long-term side effects of systemic steroids.

The ABCs of VKH

VKH is a multisystemic disorder characterized by granulomatous panuveitis with serous retinal detachments often associated with neurologic and cutaneous manifestations.[1] Vogt, Koyanagi and Harada described a wide spectrum of clinical findings that incorporated a similar pathology involving an overactive immune response to melanocytes.[1] To clarify the diagnostic features of the disease, the International Committee on Nomenclature established revised criteria and a new three-category classification system: complete, incomplete and probable.[2]

Complete VKH is characterized by bilateral multifocal serous retinal detachments, evidence of previous early manifestations of the disease (including nummular chorioretinal scars and RPE clumping), as well as CSF leukocytosis, cranial nerve palsies, meningitis, tinnitus, alopecia, vitiligo and poliosis.[2]

Incomplete VKH is characterized by a combination of neurologic and auditory disease manifestations or integumentary signs, but not both.[2]

Probable VKH disease only manifests ocular findings.[2]

Although the etiology of VKH is uncertain, the presence of HLA-DR4 in affected individuals highlights the possibility that the disease has an autoimmune pathogenesis against an antigenic component shared by uveal, dermal and meningeal melanocytes.[3] Most cases are sporadic, but some suggest that certain ethnic groups share a common immunogenic predisposition for an autosomal recessive trait.[4] In one study of patients with VKH disease, 50% were white, 35% African American and 13% Hispanic—with a large percentage of affected individuals having remote Native American ancestry.[5] While the age of onset is variable, it most commonly occurs within the third decade, affecting females more than males (2:1).[5]

VKH diagnosis is primarily clinical. The differential diagnosis includes sympathetic ophthalmia, sarcoidosis, primary intraocular B-cell lymphoma, posterior scleritis and uveal effusion syndrome.[6] Long-term ocular complications can be both reversible and irreversible, the latter of which may include cataract, glaucoma, subretinal fibrosis, choroidal neovascularization and optic atrophy.

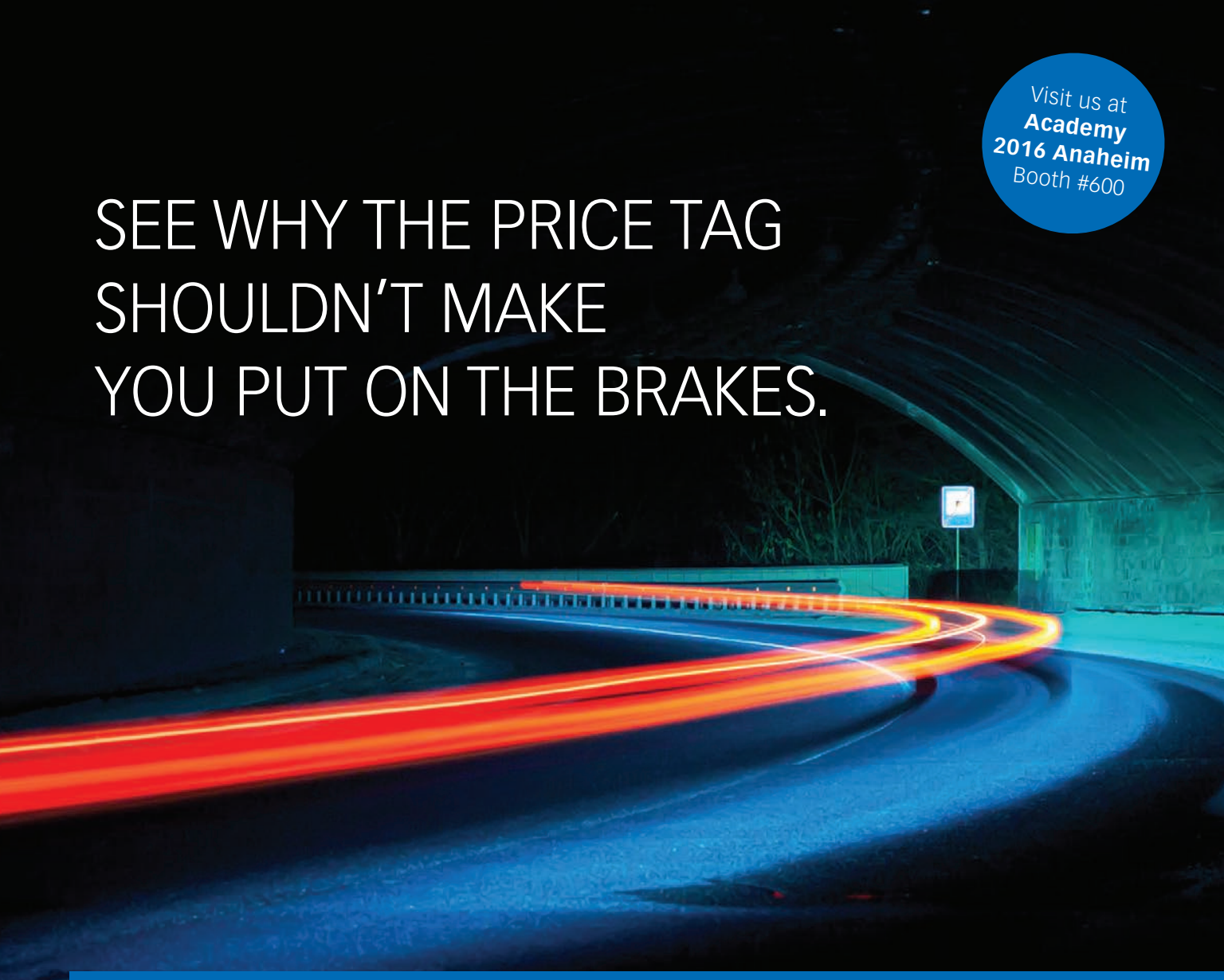
Treatment efficacy largely depends on early diagnosis and high-dose systemic corticosteroid therapy. Once initiated, steroids should not be discontinued until there is significant improvement in the ocular findings.[1] Neurologic and auditory manifestations also respond well to corticosteroids, with immunomodulatory agents being used as adjunctive treatment or primary treatment when corticosteroid side effects are not tolerated. Conversely, integumentary signs—including poliosis, vitiligo and alopecia—persist and progress despite treatment.

In this particular case, the initial presentation of partial third nerve palsy was a precursor to fundus findings consistent with VKH. Patients with neuro-ophthalmic disease require detailed anterior and posterior segment examination to look for active or previous findings of intraocular inflammation, since a low threshold for VKH disease is important to establish an early diagnosis and prevent long-term systemic and ocular morbidity. ■

1. Andreoli CM, Foster CS. Vogt-Koyanagi-Harada disease. Int Ophthalmol Clin. 2006;46:111–2.
2. Read RW, Holland GN, Rao NA, et al. Revised diagnostic criteria for Vogt-Koyanagi-Harada disease: report of an international committee on nomenclature. Am J Ophthalmol. 2001;131(5):647-52.
3. Davis JL, Mittal KK, Freidlin V, et al. HLA associations and ancestry in Vogt-Koyanagi-Harada disease and sympathetic ophthalmia. Ophthalmol. 1990;97(9):1137-42.
4. Levinson RD, Du Z, Luo L, et al. KIR and HLA gene combinations in Vogt-Koyanagi-Harada disease. Hum Immunol. 2008;69(6):349-53.
5. Nussenblatt RB. Clinical studies of Vogt-Koyanagi-Harada's disease at the National Eye Institute, NIH, USA. Jpn J Ophthalmol. 1988;32(3):330-3.
6. Pahk PJ, Todd DJ, Blaha GR, et al. Intravascular lymphoma masquerading as Vogt-Koyanagi-Harada syndrome. Ocul Immunol Inflamm. 2008;16(3):123-6.

Visit us at
Academy 2016 Anaheim
Booth #600

SEE WHY THE PRICE TAG SHOULDN'T MAKE YOU PUT ON THE BRAKES.



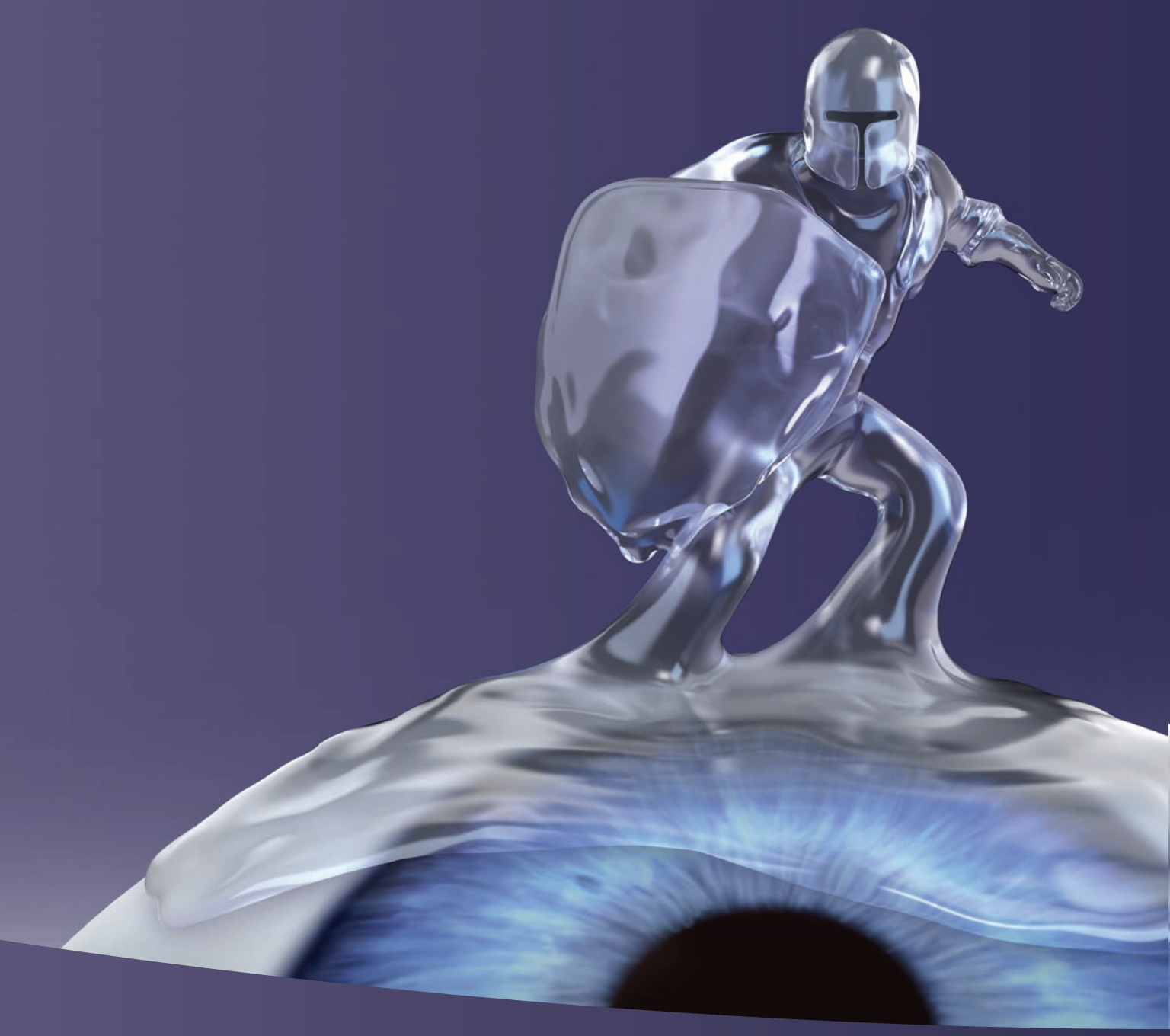
A Haag-Streit slit lamp can more than pay for itself.

Slow performance, repairs, downtime, and unreliable results are a drag on both patient care and profits. With industry-leading Haag-Streit slit lamps, you can expect unrivaled reliability, longevity, and accuracy for the level of excellence you envision and your patients deserve. Add that to minimal depreciation in value, and every dollar spent on a Haag-Streit slit lamp is a wise investment. So what's stopping you?

Set a course for perfection. Learn more at haag-streit-usa.com

800.787.5426
haag-streit-usa.com

HAAG-STREIT USA



Indications and Usage
BromSite™ (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

  It is recommended that BromSite be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- Use of topical NSAIDs may result in keratitis. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular

A DROP OF PREVENTION

FOR YOUR CATARACT SURGERY PATIENTS

Introducing the *FIRST* and *ONLY* NSAID indicated to prevent ocular pain in cataract surgery patients[1]

Defend against pain and combat postoperative inflammation with the penetrating power of BromSite™ formulated with DuraSite®[1]

- DuraSite increases retention time on the ocular surface and absorption of bromfenac[2-5]
  - Allows for increased aqueous humor concentrations
- Ensures complete coverage throughout the day with BID dosing[1]

Visit bromsite.com to find out more.

BromSite™
(bromfenac ophthalmic solution) 0.075%
Formulated with DURASITE® DELIVERY SYSTEM

surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

- BromSite should not be administered while wearing contact lenses. The preservative in BromSite, benzalkonium chloride, may be absorbed by soft contact lenses.
- The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

You are encouraged to report negative side effects of prescription drugs to the FDA. Visit www.fda.gov/medwatch or call 1-800-FDA-1088.

Please see brief summary of full Prescribing Information on the adjacent page.

NSAID=nonsteroidal anti-inflammatory drug.

References: 1. BromSite [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016. **2.** Hosseini K, Hutcheson J, Bowman L. Aqueous humor concentration of bromfenac 0.09% (Bromday™) compared with bromfenac in DuraSite® 0.075% (BromSite™) in cataract patients undergoing phacoemulsification after 3 days dosing. Poster presented at: ARVO Annual Meeting; May 5-9, 2013; Seattle, Washington. **3.** Bowman LM, Si E, Pang J, et al. Development of a topical polymeric mucoadhesive ocular delivery system for azithromycin. *J Ocul Pharmacol Ther.* 2009;25(2):133-139. **4.** ClinicalTrials.gov. Aqueous humor concentration of InSite Vision (ISV) 303 (bromfenac in DuraSite) to Bromday once daily (QD) prior to cataract surgery. https://clinicaltrials.gov/ct2/show/results/NCT01387464?sect=X70156&term=insite+vision&rank=1. Accessed July 18, 2016. **5.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66.

SUN OPHTHALMICS

BromSite™ (bromfenac ophthalmic solution) 0.075% Brief Summary

INDICATIONS AND USAGE

BromSite™ (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

DOSAGE AND ADMINISTRATION

Recommended Dosing

One drop of BromSite should be applied to the affected eye twice daily (morning and evening) 1 day prior to surgery, the day of surgery, and 14 days postsurgery.

Use with Other Topical Ophthalmic Medications

BromSite should be administered at least 5 minutes after instillation of other topical medications.

Dosage Forms and Strengths

Topical ophthalmic solution: bromfenac 0.075%.

CONTRAINDICATIONS

None

WARNINGS AND PRECAUTIONS

Slow or Delayed Healing

All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.

Potential for Cross-Sensitivity

There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.

Increased Bleeding Time of Ocular Tissue

With some NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

It is recommended that BromSite be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.

Keratitis and Corneal Reactions

Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. These events may be sight threatening. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health.

Post-marketing experience with topical NSAIDs suggests that patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients.

Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

Contact Lens Wear

BromSite should not be administered while wearing contact lenses. The preservative in BromSite, benzalkonium chloride, may be absorbed by soft contact lenses.

ADVERSE REACTIONS

Clinical Trial 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 clinical practice.

The most commonly reported adverse reactions in 1–8% of patients were: anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain and ocular hypertension.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

There are no adequate and well-controlled studies in pregnant women to inform any drug associated risks. Treatment of pregnant rats and rabbits with oral bromfenac did not produce teratogenic effects at clinically relevant doses.

Clinical Considerations

Because of the known effects of prostaglandin biosynthesis-inhibiting drugs on the fetal cardiovascular system (closure of ductus arteriosus), the use of BromSite during late pregnancy should be avoided.

Data

Animal Data

Treatment of rats with bromfenac at oral doses up to 0.9 mg/kg/day (195 times a unilateral daily human ophthalmic dose on a mg/m^2 basis, assuming 100% absorbed) and rabbits at oral doses up to 7.5 mg/kg/day (3243 times a unilateral daily dose on a mg/m^2 basis) produced no structural teratogenicity in reproduction studies. However, embryo-fetal lethality, neonatal mortality and reduced postnatal growth were produced in rats at 0.9 mg/kg/day, and embryo-fetal lethality was produced in rabbits at 7.5 mg/kg/day. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Lactation

There are no data on the presence of bromfenac in human milk, the effects on the breastfed infant, or the effects on milk production; however, systemic exposure to bromfenac from ocular administration is low. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for bromfenac and any potential adverse effects on the breast-fed child from bromfenac or from the underlying maternal condition.

Pediatric Use

Safety and efficacy in pediatric patients below the age of 18 years have not been established.

Geriatric Use

There is no evidence that the efficacy or safety profiles for BromSite differ in patients 65 years of age and older compared to younger adult patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis and Impairment of Fertility

Long-term carcinogenicity studies in rats and mice given oral doses of bromfenac up to 0.6 mg/kg/day (129 times a unilateral daily dose assuming 100% absorbed, on a mg/m^2 basis) and 5 mg/kg/day (540 times a unilateral daily dose on a mg/m^2 basis), respectively revealed no significant increases in tumor incidence.

Bromfenac did not show mutagenic potential in various mutagenicity studies, including the bacterial reverse mutation, chromosomal aberration, and micronucleus tests.

Bromfenac did not impair fertility when administered orally to male and female rats at doses up to 0.9 mg/kg/day and 0.3 mg/kg/day, respectively (195 and 65 times a unilateral daily dose, respectively, on a mg/m^2 basis).

PATIENT COUNSELING INFORMATION

Slow or Delayed Healing

Advise patients of the possibility that slow or delayed healing may occur while using NSAIDs.

Concomitant Topical Ocular Therapy

If more than one topical ophthalmic medication is being used, advise patients to administer BromSite at least 5 minutes after instillation of other topical medications.

Concomitant Use of Contact Lenses

Advise patients not to wear contact lenses during administration of BromSite. The preservative in this product, benzalkonium chloride, may be absorbed by soft contact lenses.

Sterility of Dropper Tip/Product Use

Advise patients to replace the bottle cap after use and do not touch the dropper tip to any surface as this may contaminate the contents.

Advise patients to thoroughly wash hands prior to using BromSite.

Rx Only

Distributed by: Sun Pharmaceutical Industries, Inc. Cranbury, NJ 08512

SUN-OPH-BRO-017 09/2016

SUN OPHTHALMICS

When the Pain Won't Go Away

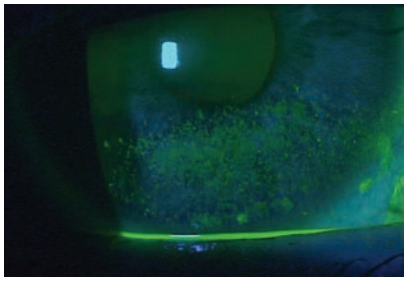
Early diagnosis is key to helping patients overcome ocular neuropathic pain associated with dry eye disease. **By Paul M. Karpecki, OD**

All eye care providers know dry eye signs and symptoms do not correlate.[1] Patients reporting dryness, burning, itching, fluctuating vision and even redness could be diagnosed with any number of conditions, including allergic conjunctivitis; blepharitis; eye misalignment issues such as convergence insufficiency and vertical imbalance; epithelial basement membrane dystrophy; giant papillary conjunctivitis; conjunctivochalasis; and conjunctival concretions, to name only a few. Fortunately, point-of-care tests such as osmolarity and inflammatory markers may help you make the differential diagnosis.[2]

However, there are also patients that truly have dry eye disease (DED) but lack significant symptoms (e.g., neurotrophic dry eye), while others may have a subtype of DED such as neuropathic ocular disease where symptoms are disproportionately advanced compared to signs.[3] Making things even more confusing is the fact that many patients have clear physical signs of DED, including meibomian gland dysfunction, but are still asymptomatic.[4] To help solve this confusion, let's look at neuropathic ocular pain to better understand these phenomena.

Understanding Chronic Pain

In general, there are two types of pain associated with most conditions such as DED. Nociceptive pain is the pain we generally think of, commonly stemming from trauma, insult or the potential of insult to a specific location. Neuropathic pain, however, occurs due to a lesion or disease of the somatosensory nervous system.[5] Neuropathic pain is a complex, chronic pain state that usually is accompanied by tissue damage; yet, sometimes it is simply dysfunction of the physiological nervous system.[6]



Patients with corneal staining are already in the late stage of DED. You must catch it earlier with new diagnostic tools to avoid neuropathic pain.

With neuropathic pain, the nerve fibers themselves are often damaged, altered or injured, and the tissue, in this case the ocular surface, itself is not affected.[7] These nerves may become damaged as a result of hypersensitization of the corneal or conjunctival somatosensory nerves and can send incorrect signals to other pain centers within the body, such as the peripheral and central trigeminal sensory network.[8]

Although pain management specialists are familiar with managing pain without obvious signs, most optometrists are not. We want to be able to see improvement in signs and symptoms. Chronic stress such as lid wiper, hyperosmolarity, surgical procedures or inflammation can result in hypersensitization and eventual neuropathic pain, possibly explaining the significant variances that exist in symptoms among patients with various levels of DED.[9]

The more DED we manage, the more cases we'll see of patients who, despite mild signs, are in such severe pain they cannot function (and patients with severe signs who have little to no symptoms at all). Some have referred to the neuropathic condition as "pain without stain," which helps understand the disassociation between signs and symptoms for this type of DED.[9] Furthermore, neuropathic pain may explain why patients with dry eye and reduced corneal sensitivity have significant dry eye symptoms.[10] This form of dry eye disease requires astute clinical judgment to make the diagnosis since demonstrable signs are not readily visible.

Ocular neuropathic pain is a likely symptom because of the presence of significant corneal nerves, and the unique location of the corneal nerve endings between the superficial epithelial cell and near the ocular surface make them vulnerable to repeated damage from environmental exposure and hyperosmolarity.[11] Research involving a mouse model shows the primary afferent nerves became sensitized on exposure to hyperosmolar and inflammatory solutions.[12]

Diagnosis

Diagnosis involves significant clinical judgment and a good understanding of the disease course. By definition, patients with neuropathic pain will

often experience hyperalgesia or exaggerated pain, spontaneous pain, allodynia (a pain response to normal stimuli such as light or mild air flow) and dysethesias (abnormal sensations). Patients with chronic pain such as neuropathic pain also tend to have higher levels of anxiety, depression and sleep disorders.[13,14]

Although advanced testing such as confocal microscopy potentially can show a damaged sub-basal nerve plexus, most of us do not have access to such technology.[15] The primary method of diagnosis is secondary to symptoms that do not resolve with typical DED treatments. A more specific and effective way to make the diagnosis of neuropathic dry eye is to instill topical anesthetic in the eye. Patients with neuropathic pain will typically mention little to no resolution of their symptoms.[5,16]

Treatment

Unfortunately, these patients rarely respond well to local treatments. Still, we should treat the inflammation possibly contributing to their dry eye, as there may be benefit over time and with long-term use. Furthermore, local treatment may remove the stress and continuous stimuli causing the pain response. However, more standard treatments such as artificial tears do not provide patient-reported pain resolution in most cases.[13] Therefore, consider using options with anti-inflammatory properties, including corticosteroids, Restasis (cyclosporine ophthalmic emulsion, Allergan), Xiidra (lifitegrast, Shire) and oral doxycycline, to name a few. Autologous serum, which includes nerve growth factor among its contents, may affect corneal nerve function.[17]

Treating the ocular surface is essential to minimizing further insult or removing stimuli that is exaggerating pain. Other treatments include those targeting the neuropathic pain centers and descending pathways, such as tricyclic antidepressants, serotonin–norepinephrine reuptake inhibitors (SNRIs) and gabapentinoids.[18] However, tricyclic antidepressants and SNRIs may exacerbate ocular surface issues, given their strong anticholinergic activity and potential ocular drying effects. Nonsteroidal anti-inflammatory drugs (NSAIDs) are another topical option that may affect the nerves because of their ability to decrease peripheral sensitization.[18,19] Thus, treatment with gabapentinoids and topical NSAIDs may affect more nerve pathways than using one alone. Research also shows protecting the ocular surface from environmental stimuli may benefit patients with neuropathic pain, and scleral lenses may help those with neuralgia-type pain.[20]

Catch it Early

However, as with any condition, prevention is the best treatment, and preventing neuropathic pain requires early diagnosis of DED. In the early stages, DED patients typically have persistent stimuli such as hyperosmolarity and inflammation. Eventually, the pain—now neuropathic—remains even after the initiating insult is removed or treated.[21] In the case of lid wiper epitheliopathy, for example, longstanding trauma could eventually result in neuropathic pain and, even if the lid wiper is then treated, the neuropathic pain will persist.[5]

To ensure earlier diagnosis, we must use better diagnostic testing. Research shows NaFl corneal staining is a late disease indicator similar to a visual field defect in a patient with glaucoma.[22] Early disease diagnosis, however, requires uncovering early disease indicators, and newer technologies such as meibography, non-invasive break-up time and osmolarity testing—perhaps the most sensitive test to early DED—can help identify the beginning structural changes.[22]

Neuropathic dry eye is a fascinating yet difficult condition to manage. Doctors need to be aware of this condition to help prevent its development with early intervention of dry eye disease. In cases where DED and neuropathic pain coexist, knowing how to make the diagnosis and treatment options can make a significant difference in the lives of these patients. ■

1. Nichols KK, Nichols JJ, Mitchell GL. The lack of association between signs and symptoms in patients with dry eye. Cornea. 2004;23(8):762-70.
2. Bron AJ, Tomlinson A, Foulks GN, et al. Rethinking dry eye disease: a perspective on clinical implications. Ocul Surf. 2014;12(2 Suppl):S1-31.
3. Spierer O, Felix ER, McClellan AL, et al. Corneal mechanical thresholds negatively associate with dry eye and ocular pain symptoms. Invest Ophthalmol Vis Sci. 2016;57(2):617-25.
4. Arita R, Minoura I, Morishige N, et al. Development of definitive and reliable grading scales for meibomian gland dysfunction. Am J Ophthalmol. 2016 Sep;169:125-37.
5. McMonnies CW. The potential role of neuropathic mechanisms in dry eye syndromes. J Optom. 2016 Jul 15. pii: S1888-4296(16)30034-6.
6. Weiner DK. Office management of chronic pain in the elderly. Am J Med. 2007;120:306-315.
7. Tsantoulas C, Zhu L, Shaifta Y, et al. Sensory neuron down-regulation of the Kv9.1 potassium channel subunit mediates neuropathic pain following nerve injury. J Neurosci. 2012 Nov 28;32(48):17502-13.
8. Goyal S, Hamrah P. Understanding neuropathic corneal pain—gaps and current therapeutic approaches. Semin Ophthalmol. 2016;31(1-2):59-70.
9. Rosenthal P, Baran I, Jacobs DS. Corneal pain without stain: is it real? Ocul Surf. 2009;7:28-40.
10. Benitez-Del-Castillo JM, Acosta MC, Wassfi MA, et al. Relation between corneal innervation with confocal microscopy and corneal sensitivity with noncontact esthesiometry in patients with dry eye. Invest Ophthalmol Vis Sci. 2007;48:173-81.
11. Galor A, Levitt RC, Felix ER, et al. Neuropathic ocular pain: an important yet under-evaluated feature of dry eye. Eye. 2015;29:301-12.
12. Parra A, Gonzalez-Gonzalez O, Gallar J. Tear fluid hyperosmolality increases nerve impulse activity of cold thermoreceptor endings of the cornea. Pain. 2014;155(8):1481-91.
13. Haanpaa ML, Backonja MM, Bennett MI, et al. Assessment of neuropathic pain in primary care. Am J Med. 2009;122:s13-21.
14. Crane AM, Levitt RC, Felix ER, et al. Patients with more severe symptoms of neuropathic ocular pain report more frequent and severe chronic overlapping pain conditions and psychiatric disease. Br J Ophthalmol. 2016 Apr 29. [Epub ahead of print].
15. Spierer O, Felix ER, McClellan AL, et al. Corneal mechanical thresholds negatively associate with dry eye and ocular pain symptoms. Invest Ophthalmol Vis Sci. 2016;57:617-25.
16. Rosenthal P, Borsook D. Ocular neuropathic pain. Br J Ophthalmol. 2016;100(1):128-34.
17. Lambiase A, Micera A, Sacchetti M, et al. Alterations of tear neuromodulators in dry eye disease. Arch Ophthalmol. 2011;129:981-6.
18. Kraff MC, Martin RG, Neumann AC, et al. Efficacy of diclofenac sodium ophthalmic solution versus placebo in reducing inflammation following cataract extraction and posterior chamber lens implantation. J Cataract Refract Surg. 1994 Mar;20(2):138-44.
19. Kalangara JP, Galor A, Levitt RC, et al. Burning eye syndrome: do neuropathic pain mechanisms underlie chronic dry eye? Pain Med. 2016 Apr;17(4):746-55.
20. Parminder A, Jacobs DS. Advances in scleral lenses for refractive surgery complications. Curr Opin Ophthalmol. 2015 Jul;26(4):243-8.
21. Galor A, Zlotcavitch L, Walter SD, et al. Dry eye symptom severity and persistence are associated with symptoms of neuropathic pain. Br J Ophthalmol. 2015;99:665-68.
22. Schargus M, Ivanova S, Kakkassery V, et al. Correlation of tear film osmolarity and 2 different MMP-9 tests with common dry eye tests in a cohort of non-dry eye patients. Cornea. 2015 Jul;34(7):739-44.

AIR OPTIX® CONTACT LENSES AND COSMETICS USE

Melissa Barnett, OD, FAAO

UC Davis Eye Center,
Sacramento, California

Dr. Melissa Barnett was compensated by Alcon for her participation in this advertorial.

For many contact lens wearers, applying moisturizers and/or makeup is a routine part of preparing for the day. But what these patients may not realize is that the action of applying or removing cosmetics, or even the products themselves, can impact the integrity of their contact lenses.[1-3] As eye care professionals whose goal is to help patients see, look and feel their best, we need to educate our patients on the proper way to use cosmetics with contact lens wear.

In a recent study, 9 marketed brands of cosmetics (including mascaras, moisturizing creams, and makeup removers) were tested on 7 brands of soft contact lenses. Several products were found to change the shape, optical performance, and physical characteristics of some of the tested lenses.[1,2] However, AIR OPTIX® AQUA silicone hydrogel (SiHy) contact lenses demonstrated resistance to many of these effects, even when exposed to eye makeup removers.[1] Another study examined cosmetic cleansing oil sorption by hydrogel and silicone hydrogel contact lenses. In that study, AIR OPTIX® AQUA contact lenses did not demonstrate sorption of cleansing oil in either wet or dry conditions.[3]

What sets AIR OPTIX® AQUA contact lenses apart is the proprietary SmartShield™ surface technology, which effectively inhibits hydrophobic silicone from reaching the surface of AIR OPTIX® AQUA lenses[4]

What sets AIR OPTIX® AQUA contact lenses apart is the proprietary SmartShield™ surface technology, which effectively inhibits hydrophobic silicone from reaching the surface of AIR OPTIX® AQUA lenses[4] and may help the lens resist changes from everyday cosmetic product use.

Discussing cosmetics is important, because this information can improve a patient's contact lens experience and satisfaction. In addition, this conversation also offers an exciting opportunity to naturally introduce appearance-minded patients to color contact lenses. As it turns out, many of my patients who frequently use cosmetics also express interest in learning about color lenses! I recommend AIR OPTIX® COLORS contact lenses, which provide color within the lens and offer exceptional breathability.* Patients are able to change or enhance their natural eye color, which they might do for a number of reasons: for a change, to stand out, to feel more confident, to match their fashion style and more. Interested patients are reassured to learn that AIR OPTIX® COLORS contact lenses are available for a wide range of prescriptions, and include the same proprietary SmartShield™ Technology found in other AIR OPTIX® lenses (and therefore offer the same impressive resistance to damage from exposure to cosmetics). Furthermore, with no refit needed for a patient who already wears AIR OPTIX® AQUA,[5] patients can seamlessly alternate between their natural—and natural-looking!—eye color while enjoying the same clean and comfortable lens-wearing experience.[6-8]

I advise my contact-lens–wearing patients to insert their lenses before using hand cream or applying makeup, in order to prevent any residue transfer between their hands and their lenses. I also remind them to maintain proper eyelid hygiene throughout the day. By following these recommendations, patients can successfully wear AIR OPTIX® contact lenses while using cosmetic products around their eyes and on their hands. The entire AIR OPTIX® family of lenses features SmartShield™ Technology, which helps my patients to see, look and feel their best, no matter their lifestyle or visual needs.

*Dk/t = 138 @ -3.00D

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

Important information for AIR OPTIX® COLORS (lotrafilcon B) contact lenses: For daily wear only for near/far-sightedness. Contact lenses, even if worn for cosmetic reasons, are prescription medical devices that must only be worn under the prescription, direction and supervision of an eye care professional. Serious eye health problems may occur as a result of sharing contact lenses. Although rare, serious eye problems can develop while wearing contact lenses. Side effects like discomfort, mild burning or stinging may occur. To help avoid these problems, patients must follow the wear and replacement schedule and the lens care instructions provided by their eye doctor.

References **1.** Luensmann D, Yu M, Yang J, Srinivasan S, Jones L. Impact of cosmetics on the physical dimension and optical performance of silicone hydrogel contact lenses. *Eye Contact Lens.* 2015;41:218-227. **2.** Srinivasan S, Otchere H, Yu M, Yang J, Luensmann D, Jones L. Impact of cosmetics on the surface properties of silicone hydrogel contact lenses. *Eye Contact Lens.* 2015;41:228-235. **3.** Tsukiyama J, Miyamoto Y, Kodama A, Fukuda M, Shimomura Y. Cosmetic cleansing oil absorption by soft contact lenses in dry and wet conditions. *Eye Contact Lens.* 2016. [Epub ahead of print] **4.** Alcon data on file, 2013. **5.** Alcon data on file, 2013. **6.** Eiden SB, Davis RL, Bergenske PD. 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; 39:290-294. **7.** Alcon data on file, 2012. **8.** Alcon data on file, 2014.

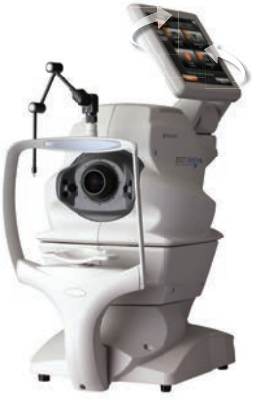
Your dreams came true.

The perfect OCT for your practice is here.

MEET THE MAESTRO.

With its space-saving design, fully automated operation, built-in non-mydriatic fundus camera, 50K A Scans/sec, and wide 12x9mm scan that captures the optic nerve and macula in one scan—the MAESTRO is everything you've been wishing for and then some. No wonder it's one of the best selling SD OCTs in the world.

Simple, precise and surprisingly affordable. Learn more at **newsgram.topconmedical.com/maestro.**



ACADEMY OF OPTOMETRY BOOTH 733

TOPCON

Spotting the Risk: From RP to RD

When a post-op cataract patient experiences retinal detachment, is there a chance it was due to retinitis pigmentosa? **Edited by Paul C. Ajamian, OD**

Q A 68-year-old retinitis pigmentosa patient was seen for the one week post-op visit following cataract surgery in the right eye. Visual acuity was unaided 20/20, but he stated that something was wrong with his vision in that eye. I dilated the patient and observed a large superior detachment that, in the course of the next hour, dropped the vision to 20/80 and then hand motion by the time I got him to the retinal specialist two hours later. What is the risk of RD after surgery, and did the RP predispose him to it?

A "It has been well established that rhegmatogenous retinal detachment (RD) is a potential complication following phacoemulsification," says Mohammad Rafieetary, OD, of Charles Retina Institute in Germantown, TN.[1-3] Although advances in cataract surgery have reduced the relative risk, "by one estimate the risk still looms at 1% compared with a 0.005% risk of retinal detachment in normal eyes," he says.[4] In RP patients, Dr. Rafieetary explains, no evidence currently supports that they are at higher risk for RD following cataract surgery.

"The relative risk for retinal detachment after cataract surgery is higher for younger patients, males and those with long axial lengths.[2] But, within the context of the RP patient, the risk of post-op complications doesn't differ from the general population," Dr. Rafieetary says.[5] He notes that macular edema *(Figure 1)* is a complication of RP, so pay special attention pre- and post-op and start patients on prophylactic NSAID drops.[5]

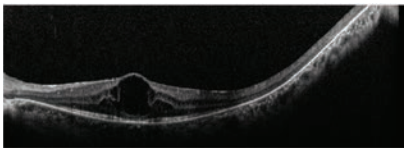


Fig. 1. Macular edema, seen here on OCT, is a complication of RP.

When managing patients with cataract, Dr. Rafieetary explains that the AOA recommends careful dilated retinal examination. "Due to the limitations of imaging devices, including widefield imaging, eye care providers should not substitute fundus photography with a dilated exam of the retina," Dr. Rafieetary suggests.

However, outside of one's standard postoperative protocols, sometimes OCT and visual fields (VF) are imperative, he says. "When unusual subjective symptoms are not explained by changes in the anterior segment, a dilated fundus exam and ancillary imaging such as OCT and automated VF must be used to properly diagnose the etiology," he says.

Q Is there anything new in RP research?

A "RP is an inherited disorder with over 100 genes implicated with its etiology and multiple subtypes," says Dr. Rafieetary. "This can explain the complexities and challenges associated with finding a single effective treatment."[6]

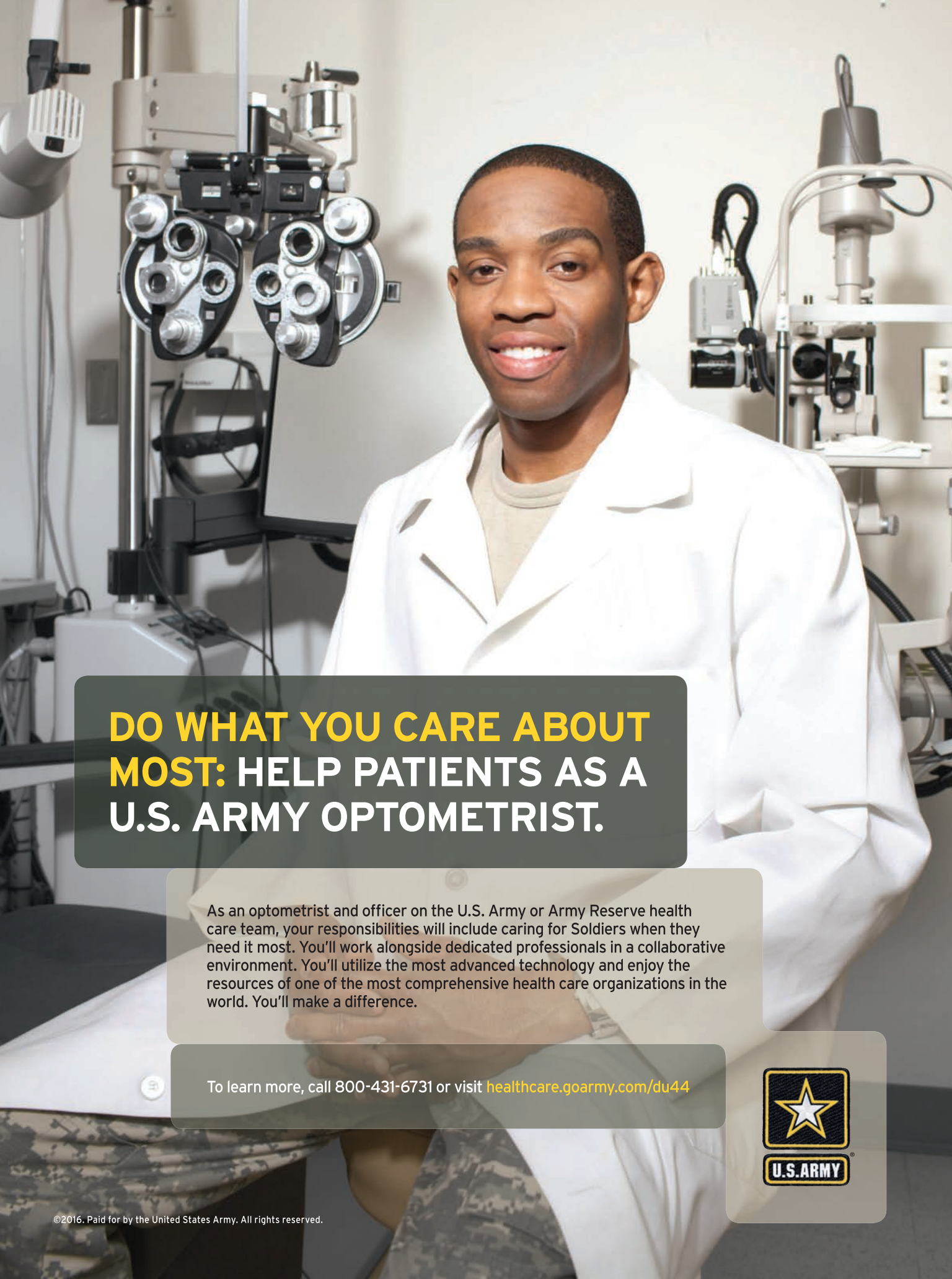
Dr. Rafieetary explains that you can easily follow the progress of RP research online. "The most comprehensive resource to follow the majority of RP clinical and scientific trials is clinicaltrials.gov, which has 117 RP studies listed on the site, in various stages.

Hope on the Horizon

While treatments for RP remain limited, the most promising therapies lie in the realm of regenerative medicine, explains Dr. Rafieetary. "Induced pluripotent stem cells are one of the most promising treatment modalities for RP and other retinal degenerative diseases, such as Leber's congenital amaurosis and Best vitelliform macular dystrophy. These cells can be generated directly from adult cells."[7-9]

The last decade has been a promising era for many posterior segment diseases that previously resulted in devastating visual outcomes, Dr. Rafieetary says, but even greater success could be on the horizon. "Over the next decade, we look forward to new advances for treatment of patients suffering from blinding retinal vascular and degenerative diseases such as RP, diabetic retinopathy and AMD." ■

1. Olsen T, Jeppeen P. The incidence of retinal detachment after cataract surgery. Open Ophthalmol J. 2012;6:79-82.
2. Clark A, Morlet N, Ng J, et al. Risk for retinal detachment after phacoemulsification: a whole-population study of cataract surgery outcomes. Arch Opthalmol. 2012;130(7):882-8.
3. Erie JC, Raecker ME, Baratz KH, et al. Risk of retinal detachment after cataract extraction: 1980-2004: A population-based study. Trans Am Ophthalmol Soc. 2006 Dec;104:167-75.
4. Ivanisevi M, Boji L, Eterovi D. Epidemiological study of nontraumatic phakic rhegmatogenous retinal detachment. Ophthalmic Res. 2000 Sep-Oct;32(5):237-9.
5. Jackson H, Garway-Heath D, Rosen P. Outcome of cataract surgery in patients with retinitis pigmentosa. Br J Ophthalmol. 2001 Aug;85(8):936-8.
6. Hartong DT, Berson EL, Thaddeus PD. Retinitis pigmentosa. The Lancet. 2006;368(9549):1795-1809.
7. Yvon C, Ramsden CM, Lane A, et al. Using stem cells to model disease of the outer retina. Comput Struct Biotechnol J. 2015;13:382-9.
8. Tucker BA, Mullins RF, Streb LM, et. al. Patient-specific iPSC-derived photoreceptors precursor cells as means to investigate retinitis pigmentosa. eLife. 2013;2:e00824.
9. Yang J, Cai B, Glencer P. Induced pluripotent stem cells and outer retinal disease. Stem Cell Internat. 2016;2016. 2850873.



DO WHAT YOU CARE ABOUT MOST: HELP PATIENTS AS A U.S. ARMY OPTOMETRIST.

As an optometrist and officer on the U.S. Army or Army Reserve health care team, your responsibilities will include caring for Soldiers when they need it most. You'll work alongside dedicated professionals in a collaborative environment. You'll utilize the most advanced technology and enjoy the resources of one of the most comprehensive health care organizations in the world. You'll make a difference.

To learn more, call 800-431-6731 or visit healthcare.goarmy.com/du44

U.S.ARMY

Don't Overlook The Obvious

Providing the best care to your patients is also a huge practice builder.

By John Rumpakis, OD, MBA, Clinical Coding Editor

There is always a drive to create a "center of excellence," a "practice of distinction" or some other way to build your practice. However, added complexity in our practices rarely leads to better economics; perhaps it's time to focus on what we have right in front of us. In the October 2013 *Review,* I published "The Economics of Apathy," which focuses on how a few common disease states can impact our patients and our practices.

I recently lectured in Houston about ocular allergy, and in doing my research, I realized the top five areas in the country where pollen forecasts were consistently high were in southern Texas, and Houston in particular.[1] Even more surprising was how few of the audience members had a vibrant allergy or dry eye practice.

Table 1. Economic Impact of Treating Ocular Allergy[2]

Overall incidence of allergies	50%
Incidence of ocular allergies	83% of total allergy sufferers
Number of people in the United States with ocular allergies	134,875,000
Number of people in your practice with ocular allergies/year	1,453
Total number of office visits related to ocular allergy	2,905
Average reimbursement for an allergy-related visits	$73
Annual allergy-related revenue per patient	$146
Total potential revenue/year due to ocular allergy	$212,472
Lifetime economic potential	$9,561,227

Table 2. Economic Impact of Treating Dry Eye[2]

Overall conservative incidence of dry eye (not including lid disease)		25%
Number of people in the United States with dry eye		81,250,000
Number of people in your practice with dry eye/year		875
Average reimbursement for dry eye-related visits		$73
Typical number of visits for nonpunctal occlusion patients per year		3
Typical revenue for Medicare punctal occlusion patient		$737.43
Typical revenue from non-Medicare punctal occlusion patient		$1,302.25
Percentage of patients undergoing punctal occlusion		3%
Percentage of Medicare patients	50%	$9,678.77
Percentage of non-Medicare patients	50%	$17,092.04
Incremental annual dry eye-related revenue		$213,003.54
Lifetime economic potential of diagnosing and treating dry eye		$9,585,159

Patients in Need

Ocular allergy and dry eye are fairly popular topics to discuss, and for good reason. The percentage of the US population that experiences symptoms of ocular allergy is in excess of 40%, and nearly 25% of the population experiences dry eye.[1] However, if you include lid margin disease, the population may be in excess of 60% to 70%.[2] With increased use of handheld devices, symptoms and clinical signs of dry eye are increasing daily. So why do we continually ignore these areas of patient care? Are we lazy, complacent or a combination of both?

Let's look at how taking care of your patients gives them a better quality of life and helps insulate your practice from market pressures.

The average OD works 45 years, making roughly $150,000 per year.[3] Even without the compounding of money factors, that puts the average OD's lifetime earnings at roughly $6,750,000. While that may seem like a lot, inaction in our practices ends up costing us more than what our day-to-day practices provide.

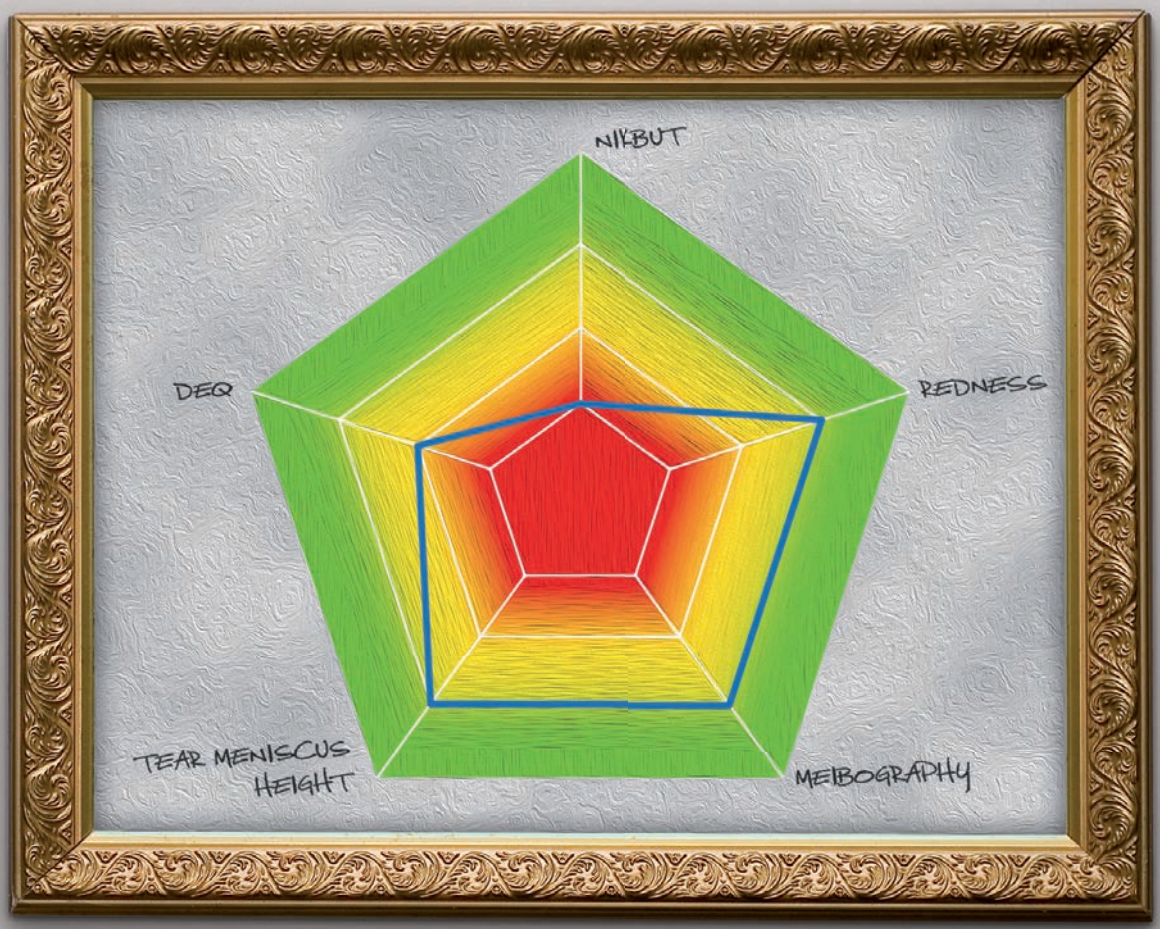
Most of us believe a reasonable patient would tell us they suffer from ocular allergies or dry eye symptoms. However, average patients generally don't keep up with our scope of practice, nor do they know our breadth of clinical knowledge; more importantly, they are often self-treating with over the counter products.

The economic impact is staggering, and practicing to the highest level that our individual licenses allow is critical. We need to be proactive and ask the right questions, perform the right examination and provide the best treatment for efficient and effective outcomes. Health care reform and the eye care model are not mutually exclusive; elevate your business by simply elevating the level of care you provide. ■

Send questions and comments to ROcodingconnection@gmail.com.

1. www.pollen.com.
2. CMS national average figures as of October 2016.
3. Hepp R. 2015 income survey: love what you do. Rev Optom. 2015;152(11):30-4.

A Masterpiece of Dry Eye Diagnostics



OCULUS Keratograph® 5M with JENVIS Dry Eye Report: See all relevant dry eye information at a glance!

The JENVIS Dry Eye Report is a unique feature of the OCULUS Keratograph® 5M that summarizes all data from your dry eye workup and offers an easy-to-grasp print-out for your patient. Excel with your dry eye diagnosis, combine screening and patient education and improve the bottom line with the OCULUS JENVIS Dry Eye Report!

Visit the OCULUS booth #627 at Academy of Optometry 2016 or go to www.dryeyemasterpiece.com

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

OCULUS®

THE STATE OF CATARACT SURGERY IN THE U.S.

Think about today's cataract patients. These patients are active, outgoing, still working, and digitally savvy. What's more, this is not a small group. In 2015, people ages 50 and older represented 45% of the U.S. population.[1] Many cataract patients (estimated around 52%) also have astigmatism,[2] which further impacts their ability to see clearly. That leaves many of our patients with two conditions affecting their sight, both of which can be addressed at the time of cataract surgery.

So, what is an optometrist's part of this conversation? We ought to be key players in diagnosing cataract as well as educating patients about conditions like astigmatism that can, and will, impact their outcomes after cataract surgery. Yet, research shows that we are not as involved as we could be, even with long-term patients whom we have treated and prescribed glasses or contact lenses for years. This article illustrates the opportunity, the potential consequences to our patients and practices, and the road to better outcomes for our patients.

Baby Boomers: America's Silver Tsunami

There exists a growing gap in access to medical eye care. Between 2005 and 2020, demand for eye care services is projected to increase by 28%.[3] During this same period, the supply of ophthalmologists is only expected to increase by 2%.[3]

Now consider this: Only 40% of all cataract patients are referred by optometrists.[4] How did the other 60% bypass an optometrist? What did we do—or not do—to create this disparity?

As primary eye care providers, we should be working to increase these figures, especially when the need for such diagnoses is growing. There were approximately 3.8 million cataract procedures performed in 2015, and that number is expected to increase by 3% in 2016.[5] It's time that we prepare for the significant task ahead of us—both in terms of size and consequence.

The Cataract Patient Journey

One way that we can increase our role with cataract surgery patients is to better comprehend how they experience their condition. Currently, there are three stages in a cataract patient's journey after their diagnosis:

- ***Post-diagnosis:*** Research and education about cataract surgery and lens choices
- ***Pre-surgery consultation:*** A once-in-a-lifetime choice of IOL
- ***Post-surgery:*** Recovery and evaluation of their surgery and lens choice

As the Figure (*see "The Cataract Patient Journey," page 3*) illustrates, patient engagement peaks when the optometrist diagnoses the patient with cataracts. Unfortunately, however, patient engagement soon drops off and doesn't peak again until decision-making time. As such, many opportunities to educate a patient about their condition and inform them of their surgery options prior to the procedure are lost. Were we to engage patients early and often after diagnosis, we could

CATARACT SURGERY = REFRACTIVE SURGERY

Cataract refractive surgery is a customized correction of nearsightedness, farsightedness, astigmatism, and the refractive aging changes of the eye. When cataract surgery is viewed as a refractive procedure, it offers the possibility of optimal vision correction for patients. A key aspect in providing a true refractive correction as part of the cataract procedure is the need to address astigmatism. Without complete and successful treatment of astigmatism, the goal of achieving excellent uncorrected distance vision and reducing spectacle dependence for distance vision cannot be met. Make sure you ask whether the surgeon to whom you refer your patients is a cataract refractive surgeon.

potentially make a positive impact on their final choices and enable them to feel prepared to make this once-in-a-lifetime decision.

Pathway to Satisfaction

Why might your patients become dissatisfied after cataract surgery? It could be that they are not receiving the education they need before undergoing surgery. We cannot be certain that surgeons are making patients aware of all their options or that they know how much time they are spending with each patient. Although most surgery centers have patient counselors, many do not. Education, and patient identification present an opportunity for patients and optometrists alike.

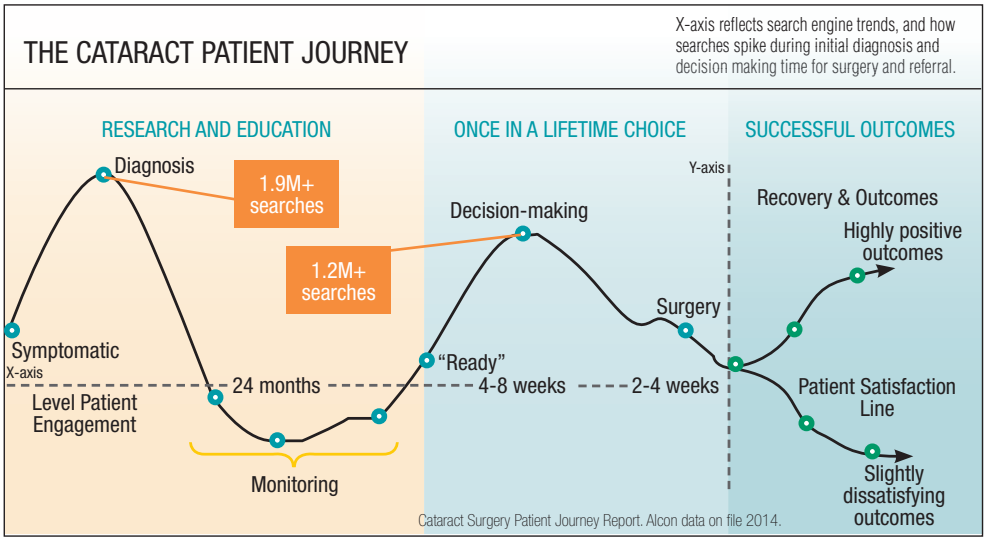
As optometrists, we're in a unique position to engage patients early and ensure they know their surgery options. A simple way we can do this is by identifying appropriate candidates who have astigmatism and should speak to their surgeon about whether they should get a toric IOL; this can be very straightforward in patients with known astigmatism that you have been managing for years. These patients have a corneal astigmatism of .75D or more, a stable ocular surface, and a desire to reduce wearing glasses for distance. It is as simple as discussing the patient's unique vision needs and communicating them with the patient and their selected surgeon.

Understanding your patient's goals after cataract surgery is also a key to achieving optimal outcomes for your patient and should be a topic of conversation with their surgeon.

Light the Way to Visual Freedom

Patients aren't the only ones who are affected by poor outcomes. As primary care providers, optometrists have the history and the relationship with the patient. Consider the impact that an underwhelming outcome can have on your relationship with a patient who you referred. Conversely, how might an outstanding outcome enhance your relationships?

Research illustrates that more than 50% of cataract patients have levels of astigmatism that can be managed with a toric IOL, yet only 7% of patients receive one.[2] While many factors impact the eligibility and appropriateness of a toric IOL—including costs and pre-existing eye conditions—can these truly account for those patients who don't have their astigmatism corrected with a toric IOL?



Indeed, a patient with astigmatism may decide that a toric IOL is not right for them, but don't we have the duty—particularly for our long-term patients—to help them make the best choice possible about their vision for the rest of their lives? This is a once-in-a-lifetime decision that you can directly affect. You can make a big impact by helping your patients make educated, informed choices.

Interestingly, market research shows that optometrists' hesitance to discuss the option of a toric IOL does not stem from an unwillingness to discuss IOLs of any variety. On the contrary, optometrists report discussing monofocal IOLs 72% of the time, but only educate patients on toric IOLs 44% of the time.[6] Furthermore, when toric IOLs are discussed, these conversations tend to be largely positive.[6] Why then are these options not a part of the conversation?

Here's What You Can Do

As an optometrist, there is so much you can you do to help satisfy the patient. Most importantly, you must start the discussion. We have to educate the patient on their condition and discuss the steps needed for cataract surgery. We can also direct them to consumer websites such as mycataracts.com and call centers such as 1-844-MYCATARACT (1-844-962-2827).

We also should talk with the surgeon to whom we refer patients. Communication with all parties is key. We ought to know what technologies our surgeon is using and why. It is important to develop an open communication protocol. Indeed, cataract surgery is an opportunity to work together as a team for the best patient outcome.

1. Audience Insights: Communicating to Boomers (1946-1962). Centers for Disease Control and Prevention website. Available at: http://www.cdc.gov/healthcommunication/pdf/audience/audienceinsight_boomers.pdf (last accessed February 10, 2015).
2. Hill W. Distribution of corneal astigmatism in normal adult population. Keratometry database: http://www.doctor-hill.com/iol-main/astigmatism_chart.htm (last accessed October 11, 2016).
3. U.S. Department of Health and Human Services. Physician Supply and Demand: Projections to 2020. October 2006. http://bhpr.hrsa.gov/healthworkforce/supplydemand/medicine/physician2020projections.pdf (last accessed December 9, 2015).
4. IMS Consulting Group. Alcon Patient Journey US. Workshop 2; December 10, 2013.
5. Alcon data on file, 2015.
6. Alcon. OD and PCP Opportunity Assessment. January 7, 2013.

All In On Dry Eye

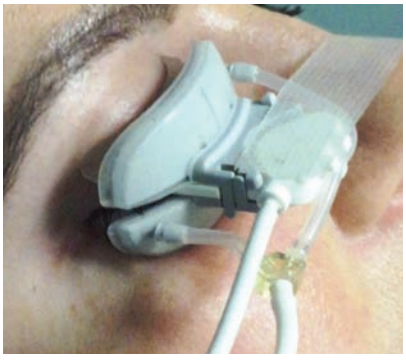
How one practice turned its focus to patients with MGD and built a successful dry eye center. **By Jennifer Lyerly, OD**

How many times a day do you see a patient who says their eyes are red, watering or feel tired and find yourself passing out artificial tears like a bank teller passing out lollipops? How often do you do the very same thing the next year?

With nearly 30 million Americans suffering from dry eye, it is more important than ever for optometrists to offer truly personalized medical care and solutions for our patients.[1] Here's how our practice is augmenting our well-established primary care practice with a center for dry eye diagnosis and management.

Why Specialize?

As Triangle Visions Optometry in Durham, NC, grew from one office to nine since 1974, more and more of our primary care patients came in with complaints of redness, eye fatigue, blurry and fluctuating vision, watering, dryness and epiphora. Like most doctors trying to care for a population of computer users, we employed the full range of our dry eye arsenal. A healthy diet of omega supplements, high quality artificial tears, Restasis (topical cycylosporine 0.05%, Allergan), punctal plugs and daily disposable contact lenses were so commonly prescribed they became drop down menu charting options in the electronic health records. But year after year, patients had the same issues and, even worse, patients who came in with these complaints were getting younger.



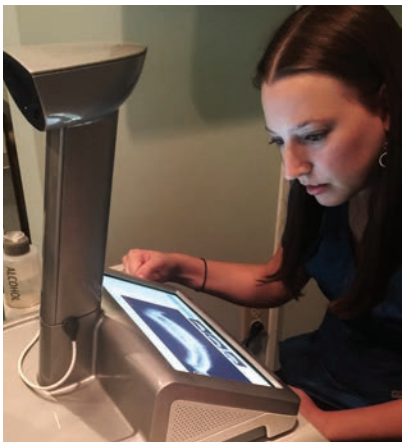
The LipiFlow device sits on either side of the closed eyelid to heat any blocked meibum and massage it free.

With the growing volume of dry eye patients, and with the dismal track record of simple solutions such as artificial tears that every other provider offers, we realized providing more specialized dry eye care could be a true practice builder. Happy patients not only come back, but they tell others about us as well. As we built the dry eye aspect of our clinics, we also saw an increase in medical office visits, and one of our key performance indicators—fill rate—surged.

Gather the Tools of the Trade

In the past few years, clinicians have many more options for dry eye diagnosis and treatment above and beyond artificial tears. Becoming a dry eye center calls for specialized tools that can more accurately diagnose patients with dry eye and meibomian gland dysfunction (MGD), as well as therapeutics that can specifically target each patient's unique causative factors of their dry eye complaints.

Studies show that approximately 40% of the adult population has clinically significant meibomian gland atrophy, and 86% of clinical dry eye patients have associated MGD.[2] To combat this prevalence in our own dry eye center—and to stand out from other local specialists also treating dry eye—we invested in the LipiFlow (Tear Science) treatment device and the meibography imaging LipiView and



Hands-on training gives staff a better understanding of the LipiScan (left) and LipiView (right) technology, and personal experience with the devices means they are better equipped to answer patient questions.

LipiScan devices (Tear Science). We are still considering adding diagnostic aids such as tear film osmolarity testing to our workup, but due to the high rate of MGD among dry eye patients, we prioritized investing in imaging and treatment solutions for that aspect of dry eye. In addition to the LipiFlow system, several other meibomian gland disease treatment options may be useful to have on hand:

- BlephEx (Rysurg) is an in-office treatment for removing collarettes associated with blepharitis.
- Intense pulsed light treatment has shown improvement in tear break-up time and dry eye symptoms in those with ocular rosacea.[3]
- MiBo Thermoflo (MiBo Medical Group) is a handheld heating device that targets expression of blocked meibomian glands. As yet, this treatment does not have any peer reviewed published studies discussing its effectiveness.

Currently there are two studied treatments to open blocked glands: manual expression and LipiFlow, the heated pulsation device that gained FDA approval in 2011. Manual expression can be a painful and lengthy process of forcefully expelling blocked meibum from each gland. The LipiFlow device sits on either side of the closed eyelid to heat any blocked meibum and massage it free. The goal of gland expression is to unblock clogged and dying meibomian glands to allow production of the oil layer and halt the atrophy process. While we are careful to educate patients that it's not a cure for dry eye, studies show that 79% of patients undergoing LipiFlow have improved dry eye symptoms just one month after treatment.[4]

In addition to gland expression treatments, we stock all of the products necessary for keeping glands open and producing. "We no longer advise warm compresses; we specify compresses with Bruder masks," says Tony Clark, OD, co-owner of Triangle Visions Optometry. "We prescribe lid cleansing pads (Ocusoft) and will combine that with Hypochlor spray (hypochlorous acid 0.02%, Ocusoft) for patients with more severe blepharitis or *Demodex* when necessary. We do not simply say, 'take supplements;' rather, we specify BioTears (Biosyntrx) or HydroEye (ScienceBased Health) supplements, which combine omega fatty acids with anti-inflammatory agents such as vitamin D and turmeric. The heightened levels of technological advancement, coupled with the higher quality in available products, will equal greater levels of dry eye treatment success." The HydroEye study showed statistically significant improvements in the dry eye symptoms and signs—including OSDI score, tear break-up time and Schirmer's score—of post-menopausal women with its formula of black currant seed and fish oil at 12 and 24 weeks of use.[5]

The FDA approval of Xiidra (lifitegrast ophthalmic solution 5%, Shire Pharmaceuticals) will add yet another treatment option to our armamentarium. Gaining FDA approval in July 2016, Xiidra is a twice daily eye drop that showed statistically significant improvement in inferior corneal staining and a subjective dry eye survey score at both six and 12 weeks of treatment.[6] Stimulating tear production and reducing inflammation is essential in keeping most dry eye sufferers comfortable, even after their meibomian glands are functional again. Having Restasis, and now Xiidra, brochures and prescribing information ready in office helps us discuss with patients how to use the medication properly to ensure they understand these prescription medications are not simple rewetting drops. We also walk through potential insurance hurdles they may encounter at the pharmacy to increase the chance they will get the medication filled instead of becoming confused or frustrated and walking away without treatment.

Get Staff On Board

Investing in the technology to make our dry eye center successful was no easy undertaking, and it was

just one part of the process. The other crucial aspect, time outside of patient care hours for staff and doctor training, adds up. We had to train our technicians on taking images and performing the treatment itself. They needed to feel comfortable explaining these devices to patients and answering questions about the treatment—and dry eye in general. Front office staff learned how to explain the procedure and some basic information about dry eye to help facilitate questions during phone calls.

Along with staff training, all clinicians learned how to identify ideal candidates for treatment, interpret their meibography images and then educate them on their unique dry eye disease and the keys to successful treatment.

Patient Education is Key

When it comes to keeping patients informed, using meibography imaging to start the discussion about dry eye has transformed the way we connect the disease symptoms and treatment for patients. It's one thing to point to a diagram of a meibomian gland on a wall; it's another to show patients their own meibomian glands and let them see the obvious damage.

"I find that the most remarkable moment for patients is when I show them what ideal, healthy glands should look like," says Adrienne Bender, senior optometric technician who performs treatments and meibography imaging. "Then I show them their own glands. The symptoms they were telling me about when I took their case history was evidence this issue had been there all along, but physically seeing their glands damaged is the most vital and visible proof of a need for change for the patient."

Communicating the need for treatment even before symptoms present is essential to caring for patients with dry eye, but it is also the biggest challenge clinicians face when starting a dry eye center. Patients often resist treating MGD, even with high-quality images proving their glands are showing signs of disease. Like most dry eye sufferers, the intensity of each patient's symptoms is highly variable. Some patients with severe gland disease have very few symptoms, but if their glands are already dysfunctional, they will eventually present with symptoms—presymptomatic, not asymptomatic, we say. In these instances, patient education is especially important. We ensure patients understand if they wait to treat MGD only after symptoms arise, the glands could be so damaged not even the latest innovations can help.

Patients often resist MGD treatment, even with high quality images such as this, showing signs of disease (in this case, severe truncation) in their glands.

"I like to refer to LipiFlow treatment as 'an oil change for the eyes,'" says Ms. Bender. "We help rid the eyes of the old, congealed oil, and the body does the rest."

Another hurdle for patients is cost. Treatment can be expensive, and currently no insurance plan covers the newer modalities. Communicating costs takes sensitivity, but we discuss budgeting options with patients, such as setting aside flex spending money, to help. If we allow the glands to permanently atrophy, no treatment can grow them back, so we always educate that we have a time-sensitive window where treatment will be effective. If we know the meibomian glands are nonfunctional, no other treatment for dry eye will be effective—just a waste of money—until we get the glands working again. Heather Hildebrand, OD, likens her discussion of treating MGD early to intervening for any other chronic degenerative disease.

"Imagine if I could tell a patient who feels fine at today's exam that we see signs they will get rheumatoid arthritis in the next few years," she says. "We have a treatment we can do now that will prevent them from suffering from the symptoms of rheumatoid arthritis. What patient wouldn't want us to help?"

Prepare for Ongoing Care

Doctors and staff know treating MGD isn't the end to managing dry eye—it's just the beginning. After treatment, we prescribe each patient a daily regimen to prevent the glands from reblocking and to treat any additional aqueous deficiency or other causes of dry eye symptoms.

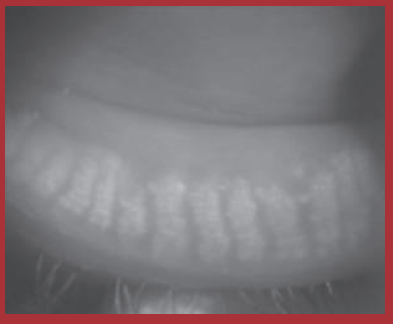
We schedule a medical office visit four to six weeks after treatment for every patient to reassess gland expression and discuss any necessary changes to the patient's daily treatment regimen. These are just like any other dry eye visit and take 10 to 20 minutes, depending on the patient's needs. We set aside

Introducing another great camera from TelScreen

Dry Eye Diagnosis and Treatment is on the rise. . .

TelScreen's IR camera
Infrared Technology

TelScreen's Infrared camera is a great tool for identifying patients who would benefit from Dry Eye treatments.



NEW

When people see their problem, they tend to act on a solution.

A New Approach to Infrared Imaging

Other systems flood the eye with Infrared illumination.

Just as our visible light systems don't need flash or background illumination, our Infrared imaging system does not require additional Infrared illumination.

Introductory Offers!

Add infrared imaging to your EyeRes system for $8,989
(List price: $10,989).

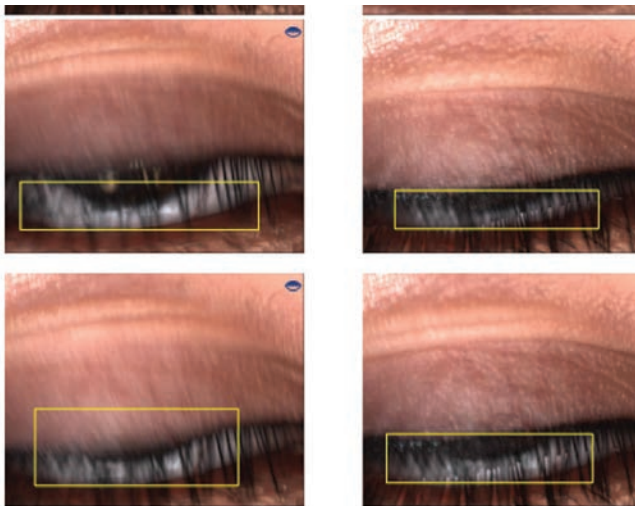
Add a complete Infrared imaging system to your compatible slit lamp for only $12,109
(under $250/month).

Get both visible light and infrared imaging for only $19,983
(List price: $28,433).

8012 Vine Crest Avenue, Louisville, Kentucky 40222 • www.TelScreen.com • 502.515.1518

appointment times for medical or contact lens checks to avoid competition with comprehensive exam slots.

Although patients may ask to get their glands imaged again to see their improvement at this follow-up visit, meibography images will look identical both before and after treatment. The only way to assess success is with gland expression; blocked glands won't express prior to treatment; afterwards, gentle pressure should show expulsion of clear meibum.



Imaging can help clarify underlying causes of clinical symptoms, such as incomplete blink, as seen here.

Clinical Extras

One of the most important prescriptions we write after gland expression treatment is blinking exercises. A significant number of patients with advanced meibomian gland disease are long-hour computer users. Staring at screens all day not only slows blink rate, but programs users to take incomplete, partial blinks.[7] We have noticed a large number of patients with severe gland atrophy have as much as 100% partial blinking rates. If patients never fully close their eyelids, the meibomian glands do not express, and the meibum stays inside the gland, clogging and hardening until a blockage becomes permanent. We prescribe a free blink training app to help teach patients a normal blink motion and educate them to follow the 20/20 rule.[8] Every 20 minutes, break to blink for 20 seconds.

Treating MGD alone can't make a dry eye center. Reports suggest 14% of all dry eye suffers have aqueous deficient dry eye and roughly 36% of dry eye sufferers have both evaporative and aqueous deficient dry eye, referred to as mixed mechanism dry eye.[4] Treating patients with mixed mechanism dry eye requires a multifactorial approach. Our strategy is to attack dry eye from the outside in, starting with eyelid disease by treating any active blepharitis or *Demodex* infections. Then we address meibomian gland health and inspissation. Once the glands are functional and healthy, we focus on aqueous production and goblet cell health. This approach ensures the surface layers of the tear film are functioning properly before we try to improve the interior tear film layer—if the tear film evaporates right off the surface, all of your work on aqueous production will likely be less effective. Some patients who undergo successful meibomian gland treatment still have breakthrough dry eye symptoms and will need aqueous and goblet cell function maximized.[4] We reach for clinically proven aqueous deficient treatments such as Restasis and even punctal plugs for the right patient, and we now have Xiidra to add to our arsenal.

Getting the Word Out

Treating patients successfully and relying on word of mouth for building a dry eye practice is a tried and true path to success; but in today's world, word of mouth travels a lot faster if your patients do it online and through social media. Our marketing strategy for the new dry eye center is a mix of both online and more traditional forms of advertising to reach patients of every age cohort that may be affected by dry eye.

"We have seen excellent success utilizing targeted email blasts to existing patients that we've flagged during their routine exams as someone suffering with dry eye symptoms. We incorporate a variety of videos and free quizzes to help connect their symptoms to the disease condition and our treatment options," says Caleb Clark, Triangle Visions' marketing director. He uses sponsored Facebook ads and Google Adwords to target local patients researching dry eye or the related symptoms online. On our website, a short dry eye questionnaire pops up to help get patients thinking about their eye exam differently.

"It's not just going to be about their new glasses prescription; our doctors will be talking about how your eyes feel and how we can help improve that too," says Mr. Clark. "It creates a value to our eye exam that separates us from area providers who are just focusing on traditional glasses and optical needs."

"I'm also excited to get our toes wet with traditional media," says Mr. Clark. "The demographic who still reads the newspaper is also the demographic most likely to

struggle with dry eye issues. We've discussed tying in traditional media advertising with a free screening event for the senior community, and I think that has immense potential to positively impact both our local community as well as our business."

Building Success

Changing with the times has always been a key to success, but knowing how to change can be challenging. We've all heard the same drums beating for dry eye for years now. 'Become a dry eye center for excellence; it will save lives, promote world peace and make you rich!' Forgetting the hype, here's what we know: an enormous percentage of patients in every primary eye care clinic in America has MGD or dry eye syndrome.

With new technology and new treatment options, we can help improve our patients' health and function in ways we never could before. In so doing, we also develop another vertical revenue column for our practice. It takes some work and focused energy, but it is doable. Anything that can benefit both our patients and our bottom line is a bit of a no-brainer.

There is no silver bullet in treating dry eye, prompting frustrated doctors and patients to reach for the ineffective simple solutions we've been using for years. But with new diagnostic tools we can pinpoint the underlying causes of each patient's dry eye symptoms and make a specific plan of action. We know more about dry eye today than ever before, and as more Americans suffer from dry eye due to diet, lifestyle and digital device use, we are better equipped to offer solutions in our exam chair. ■

Dr. Jennifer Lyerly is an associate at Triangle Visions Optometry in Cary, NC. She is the founder of eyedolatryblog.com, an eyecare education blog, and cohosts the Defocus Media optometric industry podcast.

1. Sullivan DA, Hammitt KM, Schaumberg DA, et al. Report of the TFOS/ARVO Symposium on Global Treatments for Dry Eye Disease: An unmet need. Ocul Surf. 2012;10:108-16.
2. Nichols KK, Foulks GN, Bron AJ, et al. The international workshop on meibomian gland dysfunction: executive summary. Invest Ophthalmol Vis Sci. 2011;52:1922-9.
3. Toyos R, McGill W, Briscoe D. Intense pulsed light treatment for dry eye disease due to meibomian gland dysfunction; A 3 year retrospective study. Photomed Laser Surg. 2015;33(1):41-6.
4. Lemp MA, Crews LA, Bron AJ, et al. Distribution of aqueous deficient and evaporative dry eye in a clinic-based patient population. Cornea. 2012; 31(5):472-8.Lane SS, et al. A new system, the LipiFlow, for the treatment of meibomian gland dysfunction. Cornea. 2012;31(4):396-404.
5. Sheppard JD Jr, Singh R, McClellan AJ, et al. Long-term supplementation with n-6 and n-3 PUFAs improves moderate-to-severe keratoconjunctivitis sicca: A randomized double-blind clinical trial. Cornea. 2013;32:1297-1304.
6. Shire Pharmaceuticals. Xiidra package insert. Lexington, MA.
7.Portello JK, Rosenfield M, Chu CA. Blink rate, incomplete blinks and computer vision syndrome. Optom Vis Sci. 2013; 90(5):482-7.
8. Korb Blink Training App. Available at https://itunes.apple.com/us/app/donald-korb-blink-training/id941412795?mt=8.

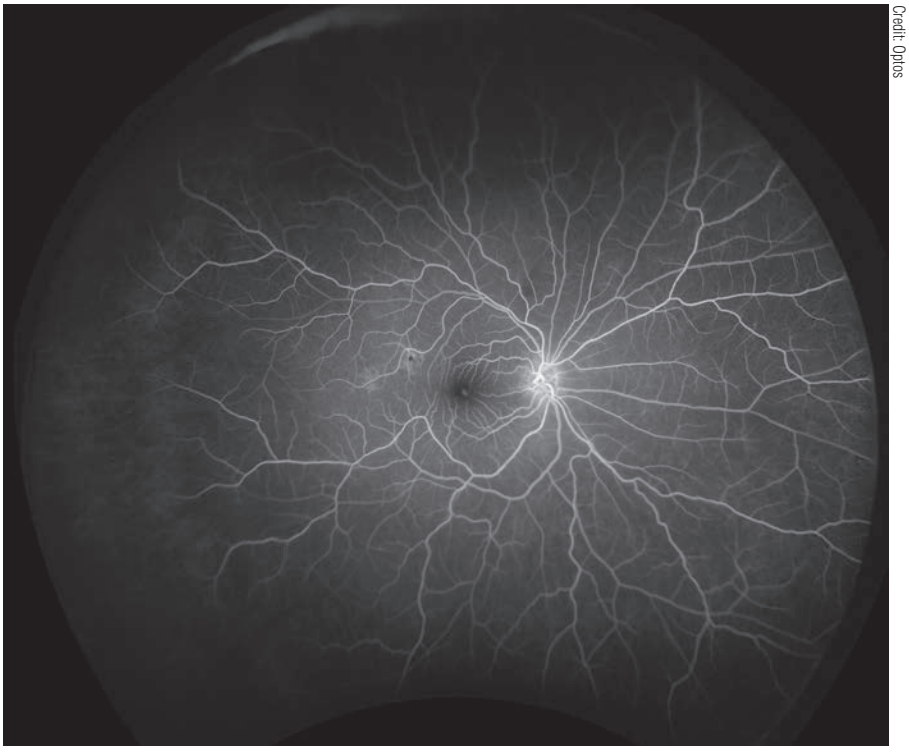
PEER • REVIEWED • RRA • ARTICLE

Four Steps to INCORPORATING DIABETES CARE

You don't need to be an expert to make treating and managing this significant systemic condition a priority in your practice. **By Paul Chous, OD**

The statistics on diabetes in the United States are truly staggering—every optometrist is seeing patients with diabetes or those at high risk for the disease on a daily basis. Recent data from the World Health Organization show that at least 422 million people worldwide have diabetes, more than 30 million of whom live in the United States; another 90 million Americans have prediabetes.[1] The National Health and Nutrition Examination Survey (NHANES) figures show that more than 51% of all American adults had either diabetes or prediabetes in 2012, a figure that jumps to 83% when considering those 65 or older.[2]

Depending on your patient population, at least half of your patient encounters could include people with abnormal blood glucose levels and those at significant risk for diabetes-related eye disease. Given this reality, we have to start thinking systematically about how we care for and educate our patients who have or are at risk for diabetes.



Credit: Optos

Screen patients to detect early signs of diabetes-related ocular conditions such as nonproliferative diabetic retinopathy, as shown here.

We need to partner with patients, their families, other health care and public health professionals and our

communities to reduce diabetes incidence and the complications that come with it.

We need to make diabetes a priority. The task may sound daunting, but every OD can do this with minimal stress by incorporating a few simple strategies.

Step 1: Reduce Risk

The best way to reduce the burden of diabetes-related eye disease is to reduce our patients' risk of developing diabetes in the first place, or at least delay the diagnosis as long as possible. Before we even dig into the weeds of a clinical exam, we should first focus on those at highest risk, and provide the following evidence-based advice to help reduce their modifiable risk factors *(Table 1)*:[3,4]

- Get at least 30 minutes of physical activity each day. For those pressed for time, high intensity interval training (HIIT) is a great alternative.
- Eat a plant-centric diet (Paleolithic or Mediterranean-type diet) with a variety of whole fruits and vegetables (12 or more different types per week).
- The single most impactful dietary strategy is to increase fiber to more than 25g each day.
- Consider fasting on alternate days if they're above ideal body weight and have prediabetes.
- Avoid foods with added sugars, especially high fructose corn syrup.
- Drink water, unsweetened tea or coffee and avoid aspartame (the latter increases insulin resistance by altering the intestinal microbiome).
- Try to get seven to eight hours of sleep every night; turn off all screens at least one hour before sleep to prevent melatonin suppression.
- Get their blood levels of vitamin D tested, with a goal of 40ng/ml to 60ng/ml; if they are below 40ng/ml, take at least 2000 IU additional vitamin D3 daily and retest in three months.
- If you have prediabetes and high blood pressure, ask your physician to take you off of hydrochlorathiazide, a common drug used to treat high blood pressure that worsens insulin sensitivity.
- Reduce consumption of red meat and avoid meat products preserved with sodium nitrate or sodium nitrite (these preservatives are toxic to pancreatic beta cells).

Table 1. Diabetes Risk Factors[4]

Modifiable	Non-modifiable
Increased body weight	Positive family history
Smoking and particulate air pollution	Essential hypertension
High dietary sugar	Older age
Low plant food consumption	Latino/African/Native/Pacific ancestry
Sedentary lifestyle	Caesarian section birth (child)
Low serum vitamin D levels	
Insufficient/excess sleep	
Excess red meat/processed meat consumption	
Use of thiazide diuretics	
Use of lipophilic statins (*atorvastatin, simvastatin, rosuvastatin*)	
Clinical depression	

- Diabetes duration equal to or greater than 10 years (the average patient has had Type 2 diabetes about six years at diagnosis).
- Glycosylated hemoglobin (HbA1c) greater than 7.5%, instability of HbA1c, extended periods of poor glucose control.
- Type 1 diabetes, especially during and following puberty.
- Insulin use (insulin promotes VEGF, so higher dose equals higher risk).
- Systemic hypertension (treated/untreated BP greater than 140/85).
- Ocular hypotension.
- Sleep apnea.

I tell my patients that a one-point decrease in HbA1c reduces their risk of retinopathy progression by about 30% based on the evidence.

I like giving patients a handout with these lifestyle recommendations so they can ask questions, read and re-read, and so I can highlight the things I think are most important.

After a patient is diagnosed with diabetes, be cognizant of the factors that increase the risk of eye diseases such as diabetic retinopathy and diabetic macular edema (DME), and counsel patients accordingly:[5]

- Dyslipidemia.
- Low macular pigment.
- Vitamin D deficiency.
- Latino, African, Native American ancestry.
- Patients of lower socioeconomic status.
- Clinical depression.

Duration and HbA1c are the most important, and I tell my patients that a one-point decrease in HbA1c reduces their risk of

After a patient is diagnosed with diabetes, be cognizant of the factors that increase the risk of eye diseases such as diabetic retinopathy and diabetic macular edema (DME), and counsel patients accordingly.

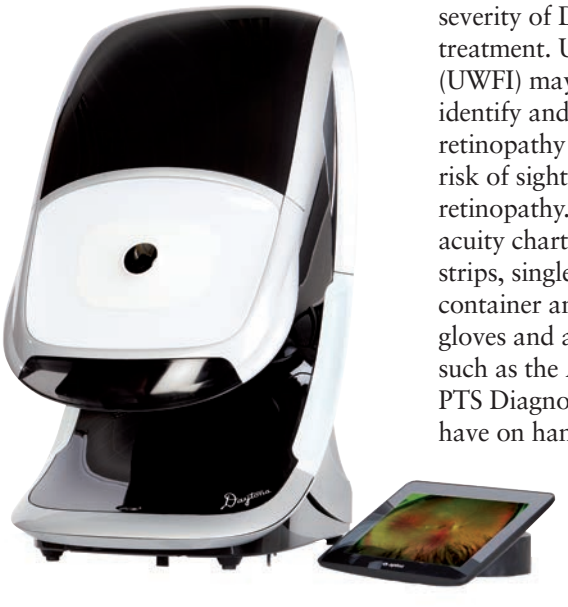
retinopathy progression by about 30% based on the evidence. Modifiable risk reduction strategies will continue to benefit patients long after diagnosis of diabetes. In addition, if we know the pros and cons of various diabetes medicines, we better position ourselves to assist patients in treatment conversations with their primary care doctors and endocrinologists *(Table 2)*.

Step 2: Prepare Your Exam Lane

You already have many of the tools you need to care for patients with diabetes, such as a slit lamp, fundus lenses, etc., and others are simple additions, such as stocking rapid-acting carbohydrates (glucose tablets, glucose gel packs, single serving boxed orange juice or a few cans of sugar-laden soda) in office in the event a patient experiences acute hypoglycemia while being examined. Here are the other essential tools you will need and likely already have:

- Binocular indirect ophthalmoscope
- Tonometer
- Gonioscopy lens
- Mydriatics
- Sphygmomanometer & stethoscope.

Other extremely helpful tools include a fundus camera to document and compare retinal and optic nerve abnormalities over time, and a spectral-domain optical coherence tomographer (SD-OCT) to help you assess for presence and severity of DME and response to treatment. Ultra-widefield imaging (UWFI) may be especially helpful to identify and document peripheral retinopathy linked to increased risk of sight-threatening diabetic retinopathy. A low contrast visual acuity chart, glucose meter and test strips, single-use lancets, sharps container and disposable latex gloves and a rapid HbA1c assay such as the A1cNow Selfcheck from PTS Diagnostics are all helpful to have on hand. Patient handouts on diabetes, diabetic retinopathy, ocular nutrition and science-based supplements can ensure proper education, and



UWFI can help spot peripheral retinopathy.

Table 2. Clinical Pearls for Diabetes Medicines[6,7]

- Oral agents generally lower HbA1c about one point.
- Metformin is cheap, effective (especially for high morning blood glucoses) and cardioprotective compared with insulin and SFU drugs (particularly in obese patients); gastrointestinal side effects can be minimized with (generic) extended release metformin; metformin is weight-neutral.
- Insulin use is typically required when HbA1c is above 10%; earlier use preserves beta cells but promotes weight gain.
- Usually, only patients on insulin or drugs that directly increase endogenous insulin secretion (e.g. sulfonylureas such as glyburide) experience hypoglycemia, which can incapacitate or kill (all health care providers should have a rapid-acting carbohydrate on-hand to treat such emergencies).
- Diabetes agents that assist with weight loss include glucagon-like peptide agonists such as Victoza/Saxenda (Novo Nordisk), Byetta (AstraZeneca), and Trulicity (Eli Lilly) and sodium glucose transporter-2 reuptake inhibitors such as Invokana (Janssen Pharmaceuticals), Farxiga (AstraZeneca) and Jardiance (Boehringer Ingelheim); these drugs appear to lower cardiovascular risk significantly, but are expensive.
- There is evidence that ACE inhibitors, ARB blockers and fenofibrate reduce risk of sight-threatening diabetic retinopathy and macular edema in adults with Type 2 diabetes and mild-moderate nonproliferative diabetic retinopathy.

MacuHealth®
With LM23™
"TRUST THE SCIENCE"

ENRICHING MACULAR PIGMENT FOR OPTIMAL VISUAL PERFORMANCE AND ULTIMATE MACULAR PROTECTION.

Studies show rebuilding macular pigment with all three critical carotenoids, meso-zeaxanthin, lutein and zeaxanthin, result in reduced glare, improved contrast and provide the strongest anti-oxidant effect protecting your vision from oxidative stress over a lifetime.

Who should be supplemented?

- **ATHLETES** and patients with **CRUCIAL VISION REQUIREMENTS**
- **POST CATARACT** patients
- Patients **PREDISPOSED WITH AMD**
- Patients **DIAGNOSED WITH AMD**
- Patients **with ALZHEIMER DISEASE**

Start Rebuilding your Macular Pigment in as little as 8 weeks.[1] To Place An Order Or Learn More Call MacuHealth at **(866) 704-0845** Or email **info@macuhealth.com**

[1] J.M. Nolan et. al., Exp. Eye Res., 2012; 101:9-15

ACHIEVE VISUAL EXCELLENCE

www.macuhealth.com /MacuHealth @MacuHealth

provide patients references when they return home. A few online resources, such as a sight-threatening retinopathy risk calculator (www.retinarisk.com) and a BMI calculator (phone apps are great) will also come in handy when counseling patients with diabetes in your office.

increase the risk of sight-threatening diabetic retinopathy. Although ultra-widefield imaging is 'ultra-fantastic,' it likely will not meet the medicolegal standard of care at the moment.

Don't forget that diabetes can cause problems beyond the retina, including dry eye disease, cataracts, extraocular muscle paresis and ocular hypertension/glaucoma.

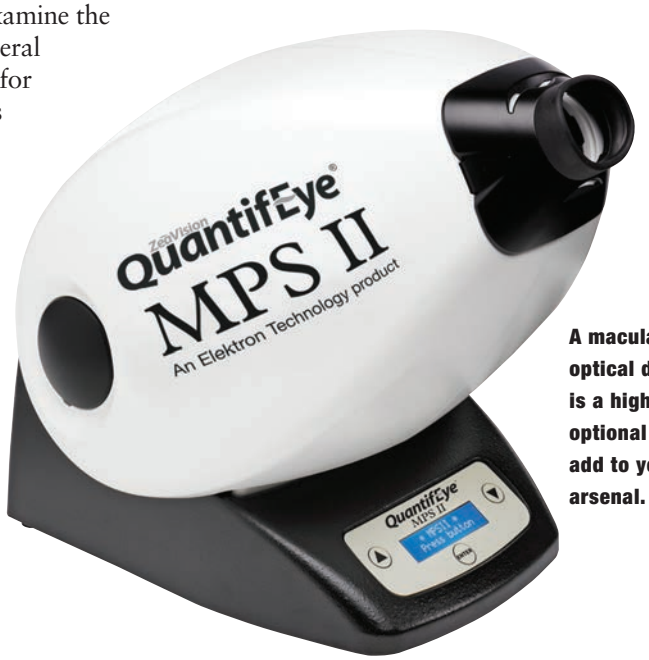
Endeavor to be an advocate, not a critic. If I do criticize something, I let patients know I am criticizing a behavior, not them.

Finally, a few other "hi-tech" tools may be helpful, such as a macular pigment optical densitometer (e.g. the QuantifEye instrument, ZeaVision) and a device for measuring accumulated glucose metabolites in the crystalline lens (ClearPath DS-120, Freedom Meditech).

Once you have the tools you need, you are ready to conduct an appropriate ocular exam in patients with diabetes.

Dilated eye examinations. These are the gold standard because they allow us to stereoscopically assess the disc and macula and examine the peripheral retina for lesions that

Technology is rapidly evolving, and devices are now available to measure accumulated glucose metabolites—a surrogate marker for diabetes risk—in the crystalline lens. Additionally, low macular pigment is associated with diabetes and diabetic retinopathy, and can be easily measured, monitored and improved via heterochromic flicker photometry. As imaging continues to evolve (e.g., adaptive optics, multi-spectral analysis, fundus flavoprotein autofluorescence), we will be able to detect ocular changes caused by diabetes earlier than ever before, allowing us to better monitor and eventually predict just who will or will not develop sight-threatening diabetic retinopathy, as well as the efficacy of new treatments.



A macular pigment optical densitometer is a high-tech, optional tool to add to your exam arsenal.

Step 3: Educate Patients

Risk calculators for diabetic retinopathy are great to have in your patient education arsenal. These allow you to immediately show patients that better control of their glucose and BP will help lower their personal risk of needing photocoagulation or injections into the eyes in the future.

It's helpful to have written materials that explain diabetic eye disease. These can complement your recommendations for follow-up, especially when caring for patients with pre-existing retinopathy and double-especially for those requiring or undergoing treatment by a retinal specialist. It is important for ODs to know that metabolic goals for patients with diabetes (i.e., HbA1c, BP, lipids) are supposed to be individualized based on life expectancy, cognition, comorbidities and complications. For instance, an HbA1c goal of 8% or less may be reasonable for an 80-year-old with little or no retinopathy, heart disease or impaired cognition.

In my experience, patients generally don't respond well to fear until eyesight has been lost, so I endeavor to be an advocate, not a critic. If I do criticize something, I let patients know I am criticizing a behavior, not them. After counseling patients on what we can do in concert with other members of the health care team to reduce the risk of vision loss, I ask patients what they would like to improve before the next eye examination. Be sure

Comfortear®
Lacrisolve™ 180
ABSORBABLE PUNCTUM PLUG

"Many of the absorbable plugs I've used tend to migrate or dissolve too quickly, but the ***Comfortear® Lacrisolve™ 180 Absorbable Punctum Plug*** *is true to its name. This plug is* ***reliable and predictable*** *at the absorption rate."*

Whitney Hauser, OD
Memphis, TN

DEFEAT DRY EYES with
the Comfortear® Family of Punctum Plugs

TO PLACE YOUR ORDER, CALL

(888) 424-1192

Visit punctumplug.com for more information. From outside the U.S., call +1 (503) 206-5733.

M074-1016-01

Paragon BioTeck, Inc. | 4640 SW Macadam Ave, Ste 80 Portland, OR 97239 | (888) 424-1192 | www.paragonbioteck.com

Comfortear is a U.S. Registered Trademark of Delta Life Science, Inc. Lacrisolve is a Trademark of Paragon BioTeck, Inc.

Paragon BioTeck, Inc.

to ask for specificity. For example, "I want to get my HbA1c under 8%," "I want to lose 20 pounds," "I want to walk 8,000 steps every day," I put their goals in the chart ask them to write them down while their eyes are dilating and tell them we're going to assess their progress at follow up—it's all about memory, buy-in and accountability. I also ask my patients with diabetes if they would like to involve family members in these discussions, because good diabetes control and self-management often require support from loved ones. This also reinforces strategies for preventing diabetes in at-risk family members and builds my practice.

If we strive to increase our knowledge of diabetes, proactively educate our patients and communicate clearly with other providers about what we have to offer, each of us can create a growing diabetes specialty niche within our optometric practices.

Step 4: Build Your Practice

With more than 30 million patients with diabetes in need of care, catering to this patient population will undoubtedly help your practice grow.[1] The first step to increasing your patient base is simply telling patients and their family members that you have a special interest in diabetes and preventing associated vision loss.

Additionally, inform every health care provider whom you see as a patient or who is already your patient about your interest in and focus on diabetes. The interdisciplinary Pharmacy/Podiatry/Optometry/Dentistry model endorsed by the Centers for Disease Control is an important tool, and one you should become familiar with and ask other providers to follow.[8] Comanagement is key for these patients, so send a timely (within two weeks) diabetes eye examination report to the primary care provider every time you see a patient with diabetes, even when there are no significant findings; be concise, compare current findings with previous findings (e.g., "our patient has mild nonproliferative diabetic retinopathy without macular edema, and this appears stable compared to my last examination"), don't use abbreviations with which other providers have limited familiarity (e.g. OU, NPDR, DME) and don't forget to be complimentary to providers who are doing a great job.

To ensure patients have access to the right information from everyone in your practice, teach your staff about diabetes and its ocular effects. Armed with the right education, they can reinforce your commitment when patients and potential patients call your office for an appointment.

We all are going to see more patients in our practices who have diabetes or are at risk for the condition. The so-called 'gray tsunami' guarantees increased prevalence of both diabetes and diabetes-related eye disease, despite recent reductions in incidence.[2] This gives all of us a chance to make a difference in public health by collaborating with patients and other stakeholders to make diabetes a priority, consistent with recommendations made in the recent report by the National Academy of Sciences, Engineering and Medicine (formerly the US Institutes of Medicine) to realize the "potential for public and private collaborations at the community, state and national levels to elevate vision and eye health as a public health issue."[9] If we strive to increase our knowledge of diabetes, proactively educate our patients and our communities about diabetes and eye disease and communicate clearly with other diabetes care providers about what we have to offer, each and every one of us can create a growing diabetes specialty niche within our optometric practices. ■

1. NCD Risk Factor Collaboration. Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4·4 million participants. Lancet. 2016 Apr 9;387(10027):1513-30.
2. Centers for Disease Control and Prevention (CDC). Annual number (in thousands) of new cases of diagnosed diabetes among adults aged 18-79 years, United States, 1980-2014. National Health Interview Survey. Available at www.cdc.gov/diabetes/statistics/incidence/fig1.htm. Accessed September 19, 2016.
3. Khavandi K, Amer H, Ibrahim B, Brownrigg J. Strategies for preventing type 2 diabetes: an update for clinicians. Therapeutic Advances in Chron Dis. 2013;4(5):242-61.
4. Ley SH, Ardisson Korat AV, Sun Q, et al. Contribution of the nurses' health studies to uncovering risk factors for type 2 diabetes: diet, lifestyle, biomarkers, and genetics. American J Pub Health. 2016;106(9):1624-30.
5. Lee R, Wong TY, Sabanayagam C. Epidemiology of diabetic retinopathy, diabetic macular edema and related vision loss. Eye and Vision. 2015;2:17.
6. Triggle CR, Ding H. Cardiovascular impact of drugs used in the treatment of diabetes. Therapeutic Advances Chron Dis. 2014;5(6);245-68.
7. Fox CS, Golden SH, Anderson C, et al. Update on prevention of cardiovascular disease in adults with Type 2 diabetes mellitus in light of recent evidence: A scientific statement from the american heart association and the american diabetes association. Diabetes Care. 2015;38(9):1777-1803.
8. Centers for Disease Control and Prevention (CDC). Working together to manage diabetes: A toolkit for pharmacy, podiatry, optometry, and dentistry (PPOD). Available at www.cdc.gov/diabetes/ndep/toolkits/ppod.html. Accessed October 20, 2016.
9. US National Academies of Sciences Health & Engineering 2016. Committee on Public Health Approaches to Reduce Vision Impairment and Promote Eye Health. Making Eye Health a Population Health Imperative: Vision for Tomorrow. Available at www.nap.edu/read/23471/chapter/1. Accessed September 18, 2016.

Tonometry Done Right

Keeler OPTICS

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

Buy Online!
Keelerusa.com

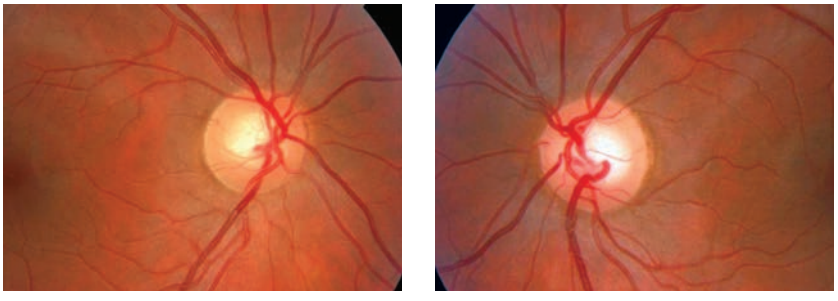
Learn more at www.keelerusa.com

Initiating a Successful Glaucoma Practice

Glaucoma is on the rise, and now is the time to embrace these patients in your practice. **By Deepak Gupta, OD**

A 56-year-old African American patient presents to your office for a routine eye exam to update his eyeglasses prescription. During the course of the visit you discover that his IOP is 22mm Hg OD, 21mm Hg OS and his cup-to-disc ratios are .70 OD and .60 OS. Now what? Years ago, the answer was: refer. But today, optometrists are licensed and capable of diagnosing and treating patients with this condition in all fifty states. Thus, I diagnosed him with glaucoma, started therapy and will be following him regularly.

For those who are already managing patients with glaucoma, you know how rewarding, challenging and lucrative it can be. For those who are not, here are the essentials of creating a glaucoma practice.



The patient's optic nerve head photos help demonstrate the cup-to-disc ratio.

Equipment Upgrades

The first step to incorporating glaucoma care into your practice is investing in the right equipment. The bare minimum equipment to properly assess these patients include:

- Applanation tonometer
- Gonioscope
- Fundus camera
- Threshold visual field analyzer

While necessary, it can be costly and you will have to budget accordingly. This list can cost between $50,000 and $60,000, and it's only the first round of purchases. The next recommendation would be a nerve fiber analyzer—a GDx or an OCT will cost another $60,000 to $80,000—followed by a corneal pachymeter.

Staff Training

This additional equipment also means that you must properly train staff on how to perform the ancillary tests. Commonly, the technician performs the photographs, visual field, etc., and you interpret the results and explain them to the patient. However, staff education should go beyond just teaching them how to perform the tests. They must also be educated on the essentials of glaucoma. Patients will often ask them questions, and having a technician capable of having an intelligent conversation with the patient adds more credibility to your practice.

Patient Protocol

The next step is establishing a protocol for glaucoma suspects and those with glaucoma. In most cases, patients with glaucoma will undergo a variety of different examinations and tests, including a comprehensive examination, visual field examina-

tion, gonioscopy, fundus photography, nerve fiber analysis and corneal pachymetry. Here is my patient protocol, as an example:

Glaucoma suspects:

Visit 1

- Comprehensive, dilated exam
- Gonioscopy
- Corneal pachymetry (if not done before)
- Fundus photography

Visit 2

- Intermediate exam/IOP check
- Nerve fiber analysis

Visit 3

- Intermediate exam/IOP check
- Visual field analysis

For all initial work ups, I separate the visits by three or four weeks each. I also try to schedule these appointments during different times of the day because I want to get an idea of the patient's diurnal variation. Studies show that normal patients exhibit as much as 4mm Hg to 5mm Hg of IOP variation in a 24-hour period. Patients with glaucoma exhibit much more of a fluctuation. If the patient is an established glaucoma suspect, I do these visits roughly every four months.

Be sure to take additional visits into consideration when scheduling. Also, these patients have a potentially blinding disease and may take more time than routine patients.

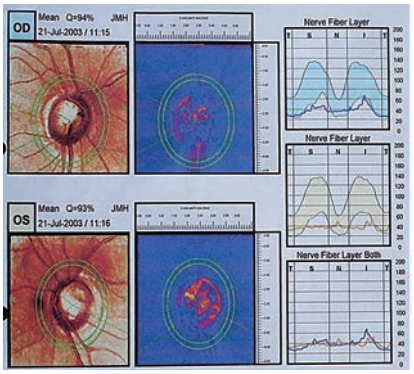
Glaucoma patients:

- Same as above but with a fourth visit for an IOP check, so the patient is in my office every three months instead of four.

To help you provide the optimum level of care for these patients, here's a closer look at the glaucoma protocol.

The Comprehensive Exam for Glaucoma

Any patient suspected of having glaucoma should have a comprehensive eye examination on an annual basis that includes dilation and more specific components such as a careful slit lamp examination, intraocular pressure (IOP) measurement and optic nerve and nerve fiber layer examination.



The patient's GDx printout shows dramatic loss of the retinal nerve fiber layer.

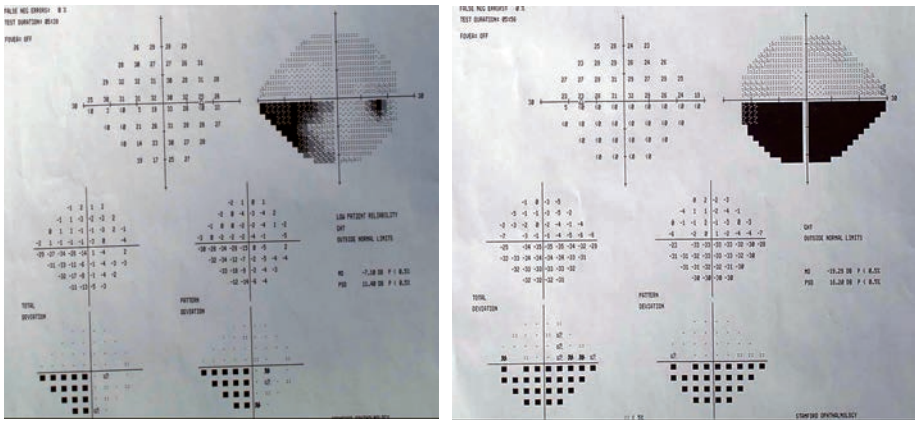
Another important step of the initial assessment is the patient's ocular, medical and family history. For some patients, the information you gather from history taking may be enough to warrant the diagnosis of glaucoma suspect and lead you to order a glaucoma work up. Some of the more common risk factors include advanced age, race (particularly African American and Hispanic), history of elevated IOP, a family history of glaucoma, high myopia and a history of iritis or ocular trauma. Patients who have hypertension, diabetes or any condition requiring the long-term use of corticosteroids are also at higher risk.

These risk factors may help you decide how aggressively you manage these patients. For example, patients with two or more risk factors, or patients with certain risk factors (such as thin corneas), warrant extra attention. For the vast majority of patients, once you have established a diagnosis of glaucoma suspect, you will follow the protocol annually. For a few who are lower risk, you may do every other year, while still seeing them for an annual exam.

When ordering tests and diagnostic procedures to better help you diagnose or manage the condition, your chart documentation must include an order for each specific test requested by you, the treating doctor. Include a short phrase indicating the request, for example, "order visual field in three months" or "gonioscopy/fundus photos performed today." This documents that additional testing is necessary beyond the comprehensive exam.

Billing tip: When coding for the comprehensive exam, you have two basic options: evaluation and management (E/M) codes or eye codes. The difference between E/M codes and eye codes is the level of service provided and proper documentation. In my practice, I use the eye codes almost exclusively. The two most common codes I use for the comprehensive exam are 92004 (comprehensive eye exam, new patient) and 92014 (comprehensive eye exam, previous patient).

Gonioscopy (code: 92020). The visual examination of the anterior chamber angle, or gonioscopy, is part of the standard of care for every glaucoma patient and glaucoma suspect. What we loosely refer to as glaucoma is actually primary open-angle glaucoma—but you can't call it that unless you've visualized the angle with gonioscopy and know that the angle is open. Many practitioners often overlook the procedure because they know roughly 90% of all glaucoma cases are open-angle glaucoma. However, 10% have a secondary mechanism, such as anatomically narrow angles, pigment dispersion or pseudoexfoliation. Perform gonioscopy on all glaucoma patients and suspects every year because the configuration of the



Visual fields show the glaucomatous defects, confirming a diagnosis of glaucoma.

angle can change as a result of pupil size, ciliary tone, iris configuration and crystalline lens size.

Billing tip: For billing purposes, you can perform gonioscopy on the same day as the comprehensive eye exam and the same day you perform other procedures such as fundus photography or visual field analysis. I suggest performing gonioscopy the same day as applanation tonometry because the cornea will already be anesthesized.

Visual field testing (code: 92083). The visual field (VF) test is the most common auxiliary test we order for glaucoma patients. Although many new methods have been developed to assess visual function in glaucoma and glaucoma suspect patients, perimetric evaluation of the glaucomatous visual field remains a cornerstone in the protocol.

Visual field testing depends on the severity of the disease and the risk of progression. You should perform a VF test yearly on glaucoma patients or glaucoma suspect patients who have stable test results. For more progressive or high-risk cases, perform VF testing every six months, and every three months for advanced glaucoma. One of the most common scenarios in which multiple testing is required is if the first VF test demonstrates glaucomatous defects or significant changes from previous tests. The second test verifies the results and checks for repeatable defects.

Billing tip: VF results aren't bundled with other tests, so you can perform them on the same day as gonioscopy and a complete eye examination. You can perform them on the same day as scanning computerized ophthalmic diagnostic imaging (92133) or fundus photography, but you lose 20% reimbursement for multiple procedures done on the same day.

Fundus photography (code: 92250). Stereo photography of the optic nerve head is the minimum standard of care for any glaucoma patient. In most cases, you'll perform fundus photography at the end of the comprehensive eye examination with the pupils dilated. Doing so provides you with an objective measurement of the C/D ratio and a comparison for future photographs. Usually, insurance will not reimburse fundus photography if you perform it on the same day as scanning computerized diagnostic imaging.

Billing tip: Perform stereo photography of the optic nerve head structure annually for most glaucoma and glaucoma suspect patients. Once you have several years of photos, you can use serial analyzing software to help you detect subtle changes.

Optic nerve and nerve fiber analysis (code: 92133). The analysis of the nerve fiber layer is a recent addition to the standard of care in glaucoma workups. Generally, you can perform optic nerve and nerve fiber analysis once a year, or every six months for patients who have advanced glaucoma. You should use a red-free filter on your slit lamp to evaluate the nerve fiber layer, as well as the objective data from a GDx nerve fiber layer analyzer or an OCT for objective, reproducible measurements to help detect subtle changes.

Billing tip: Typically billed bilaterally, these are reimbursable under the code for scanning computerized ophthalmic diagnostic imaging.

IOP measurement. Even though glaucoma is no longer defined by IOP, its measurement is an essential part of diagnosis and management. When done as part of a comprehensive or intermediate eye exam, it's considered an incidental component of the exam with no additional reimbursement. The most common scenario is when you're following a patient who needs their IOP checked after three or four months. The doctor typically checks for any changes in health and vision, updates medications and checks IOP along with a slit lamp examination. Specific items that are cause for concern are elevated IOP (above 21mm Hg), significant asymetric IOP, or a patient with significant fluctuation. Historically, research suggested IOP is highest in the early morning, gradually falling throughout the day. However, newer data suggests that IOP may actually be the highest at night.

Billing tip: The one exception to the rule of not being reimbursed for

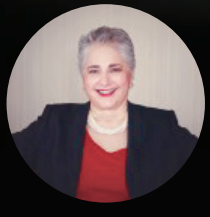
New!

icare ic100

THE **INTELLIGENT** CHOICE

"I really love the Icare tonometer... it is so quick, painless and requires no anesthesia. We use it extensively in our clinics..."

Downey Price, M.D.
Houston Eye Associates



"Using the Icare tonometer is so straight forward that even the newest techs can be trained to use it within minutes."

Jane T. Shuman, MSM, COT, COE, OCS, CMSS, OSC
Eyetechs, Inc./President

Learn more: *www.icare-usa.com* or *info@icare-usa.com*

IOP measurements is serial tonometry (code: 92100). Tonometry is considered serial when you measure IOP at least three separate times during the course of one day. This test is most commonly used in patients who have progressive glaucoma despite the appearance that they are at or below target IOP. Another common situation warranting serial tonometry is if you suspect normal tension glaucoma (code: H40.123).

Corneal pachymetry (code: 76514). With the release of the Ocular Hypertension Treatment Study (OHTS), the corneal pachymeter quickly became part of the standard of care for glaucoma. The OHTS concluded that eyes with relatively thin corneas—555µm or less—had a greater risk of developing glaucoma than those with thick corneas—558µm or greater.

Billing tip: Measure corneal thickness on every glaucoma or glaucoma suspect patient. Unlike other glaucoma testing, which is generally performed on an annual basis, corneal pachymetry is only done once in an individual's lifetime.

Vision vs. Medical

Many optometrists face a dilemma when a patient is in their office for a routine eye exam through a vision plan but they discover some finding leading to a diagnosis of glaucoma suspect. If the patient shares that he is at risk due to his family history, for example, you may consider initiating a medical visit from the beginning. Otherwise, once you proceed with a routine vision exam and uncover suspicious findings, you must have the patient return for glaucoma-specific testing, which will then be billed to the patient's medical insurance. This creates a clear-cut distinction in the patient's mind as to what was routine and what was medical. However, ensuring the patient returns for glaucoma work up can be challenging.

Patient Education

The next step is patient education, which is critical for patient compliance. The informed patient who fully understands the true blinding nature of glaucoma is more likely to adhere to treatment and follow up. Patients who only know what to do without understanding the reasoning will not be as compliant as those who are well-educated.

This can be very simple—a detailed conversation or a brochure. You could also incorporate patient education videos, which can be viewed while a patient is dilating.

Treatment

Our clinical goal is to slow the progression of the disease so the patient does not go blind in their lifetime. We lower IOP as a mere practicality, because it is the only significant risk factor we can change.

There is some consensus as to when a glaucoma suspect patient converts to glaucoma. The most obvious of these is:

1. A repeatable VF with a pattern consistent with glaucoma.
2. A repeatable or progression defect of the nerve fiber layer.
3. Progressive enlargement of the C/D ratio.
4. An IOP level warranting treatment in the absence of the above findings. I treat any patient with an IOP of 28mm Hg or higher and corneas of thin or average thickness.

The first step in treating these patients is to establish a target IOP at which the patient will not demonstrate progression. It is based on many factors such as age, starting IOP, disease severity and thickness of the cornea. However, a target IOP is no more than an educated guess and should be re-assessed at every visit.

The vast majority of patients are started on a prostaglandin such as Xalatan (latanoprost, Pfizer), Lumigan (bimatoprost, Allergan) or Travatan (travaprost, Alcon) OU QHS. These agents demonstrate excellent efficacy and tolerability with great safety profiles. However, they can be very expensive, costing $30 to $80 a month. For patients who cannot afford this, I often start them on a topical beta-blocker, which is usually $5 a month. In my mind, a patient consistently taking a beta-blocker is better than a noncompliant patient on a prostaglandin.

The Long Game

Once patients are diagnosed with glaucoma, they should be in your practice every three months to check for progressive changes. This is also an optimal time to continue educating them about compliance. More than anything, they need your support and positive outlook. Although most patients will be successfully managed, a small handful will still progress even when both of you are doing everything right.

To optimize diagnosis and treatment of patients with glaucoma and glaucoma suspects, ODs must adapt their patterns of practice to accommodate changes in patient flow and testing, scheduling and proper medical billing. In all situations, the key to your glaucoma practice is for you to administer the standard of care and bill appropriately for your services. Mastering both areas will provide the highest level of care for your patients and the best return on investment for your practice. ■

Dr. Gupta is clinical director for The Optometric Referral Center at Gupta EyeCare in Milford, CT.

For your patients with challenging visual conditions, we've brought it all into focus.

Keratoconus • Astigmatism

BAUSCH + LOMB
Specialty
VISION PRODUCTS

PMD • Post-graft • Presbyopia

Scleral lenses and customized soft lenses are now all in one convenient place.

- Customized Lenses
- Lens Care Solutions
- Education and Training
- Specialty Applications

- Specialized, dedicated customized lens sales team
- Fitting consultants with decades of experience – many are FCLSA certified
- Immediate fitting assistance, either by phone or web
- Educational resources – webinars, "hands-on" workshops, online tools

ALDEN OPTICAL™ | **Zenlens™ Scleral Lens**
NovaKone® Soft Lens

Unilens | **C-Vue® Customized Lens**

Custom. Vision. Care.

To learn more, call or visit: **800-253-3669**
www.bausch.com/svp

Working With the Kids Is Alright

A day in the life of a pediatric optometrist. **By Kathleen Foster Elliott, OD**

Pediatric optometry offers the opportunity to experience, to paraphrase Charles Dickens, "the best of times and the worst of times." For the most part, the good outweighs the bad.

A pediatric optometrist can rest easy knowing she is working to change children's lives by correcting their refractive errors, combating amblyopia and even diagnosing early cases of disease such as retinoblastoma—getting a jump on not only saving the eye but, potentially, a child's life. These experiences can be tremendously rewarding.

However, the "worst of times" include dealing with patients who scream and cry when simple drops are administered (something adult-oriented optometrists rarely encounter), negotiating undisciplined children (and parents) and, most seriously, discovering diagnoses with troubling implications such as bilateral retinal hemorrhages, which are often secondary to child abuse.

Getting kids to sit still is a trick of the trade. One solution: ask them to hold on to a little friend, like this young lady and her elephant. It can help soothe anxieties and keep tiny hands busy.

With the advent of the Affordable Care Act (ACA), predictions project an increase of pediatric patients in general practices. According AOA past president David Cockrell, OD, "we all need to become more proficient in pediatric eye care."

This article offers a glimpse into the world of pediatric optometry and what it takes to incorporate this rewarding specialty.

Why Should You be Interested in Pediatrics?

Pediatric patients historically have been underserved. For years, federal agencies and school systems have communicated that a vision screening qualifies as a comprehensive examination by an eye care provider. Nothing could be further from the truth. Optometrists know that vision screenings are not enough to fully reveal undetected vision problems such as amblyopia, latent refractive error, systemic diseases and learning disorders, many of which vision therapy can help remedy. Undetected vision problems require a complete workup and, often, comanagement subsequent to their detection.

It was one of the greatest legislative achievements of our day to have the pediatric comprehensive eye exam (not simply a screening) included in the 10 essential health benefits defined in the law. Now, millions of children in the United States have access to comprehensive ophthalmic examinations, and optometrists have the chance to make history because of our unique ability to provide these greatly needed services.

Adding pediatric services also opens other doors the ACA affords optometry. For one, offering advanced pediatric eye care is smart business. Practices must continue to find ways to diversify their revenue stream to maintain profitability. The medical pediatric model presents a significant opportunity for eye care professionals.

Typically a pediatric patient takes less time than an adult and, with proper scheduling, a clinician can see an increase in gross revenue per month. Another advantage is that families can come in together and have their examinations at the same time, therefore grouping the exams closer and adding more patients per day. Furthermore, by adding pediatric management to your optical, you can outpace competition with a wider frame selection and increased expertise.

An Optical Shop for the Younger Crop

In general practices, most frames are adult sized. Pediatric optometrists must be fiercely intentional about supplying the optical with the necessary sizes and brands—not to mention the staff training to ensure service and profitability.

Many optical frame brands address the pediatric population's specific needs. Lightweight frames are especially vital for pediatric patients, as standard polycarbonate lenses that are +4.00 or greater are thick and heavy on the bridges of tiny noses. Further complicating higher prescriptions, both myopic and hyperopic, is weight and cosmetic effect, which can be remedied by high index or aspheric lens designs. Miraflex and other brands offer frames with a bridge built to accommodate a flatter nose, which most children have. Every optical should be armed with proper staff training in pediatric frame adjustments and additional accessories such as Stayputs—slip on, adjustable devices that fit onto the back surface of the frame temple.

The impact of a proper lens prescription on a child's life is immeasurable. Take, for example, the case of an eight-year-old Hispanic female who presented to my office with uncorrected visual acuities of 20/80 OD and 20/60 OS. She had fallen behind in

Table 1. Childhood Ocular Conditions

Disease Type	Symptoms	Diagnostic Testing	Treatment
Retinopathy of prematurity (ROP): • One of the most common causes of vision loss in childhood. • Can lead to lifelong vision impairment. • Most commonly found in infants weighing less than 2.75lbs (1250g), or born after fewer than 31 weeks. • The smaller a baby is at birth, the more likely that baby is to develop ROP.	Segmented into five stages: • Stage I — Mildly abnormal blood vessel growth. Many children who develop stage I improve with no treatment. • Stage II — Moderately abnormal blood vessel growth. Many children who develop stage II improve with no treatment. • Stage III — Severely abnormal blood vessel growth. The abnormal blood vessels grow toward the center of the eye instead of following their normal growth pattern along the surface of the retina. • Stage IV — Partially detached retina. Traction from the scar produced by bleeding, abnormal vessels pulls the retina. • Stage V — Completely detached retina and the end stage of the disease.	• Fundus examination with binocular indirect ophthalmoscope, lid speculum, scleral depressor and possibly a RetCam (Clarity Medical Systems), as well as optical coherence tomography.	• Peripheral laser ablation destroys the peripheral areas of the retina, slowing or reversing the abnormal growth of blood vessels. • Patients with ROP are advised to avoid sports with a high risk of head trauma, such as soccer, football and boxing.
Amblyopia	• Usually no objective symptoms, making early comprehensive dilated examinations with cyclopentolate vital. Outcomes are best if caught and treated before three years to four years of age. Amblyopia is more challenging to treat after eight years of age.	• Cycloplegic refraction, to obtain accurate refraction. • Spot Vision Screener (Welch Allyn).	• Aggressive and expedient occlusive therapy, with refractive error corrected; including, but not limited to, patching, blur contact lens, patching glasses cyclopentolate/atropine used for occlusion therapy. • Vision therapy
Stargardt's disease (Fundus Flavimaculatus) • Mutations in gene ABCA4 are the most common cause of Stargardt's disease. This gene makes a protein that normally clears away vitamin A byproducts inside photoreceptors. Cells that lack the ABCA4 protein accumulate clumps of lipofuscin, a fatty substance that forms yellowish flecks. As the clumps of lipofuscin increase in and around the macula, central vision becomes impaired. Eventually, these fatty deposits lead to the death of photoreceptors, and vision becomes further impaired.	• Difficulty with adapting to bright light. • Central vision loss. • Color vision changes. Stargardt's disease is usually recessive (although there is also a rare dominant inherited pattern).With both parents carrying the mutation, there is a 25% chance of occurrence.	• Color vision testing • Retinal evaluation	• Currently, no cure exists, but promising avenues of research, including gene, stem cell and drug therapies, are in development. • Embryonic stem cell treatment is being employed to restore some integrity to the diseased retina in Stargardt's. • Anti-VEGF intraocular injections. • UV protection, sunglasses and low vision.
Oculocutaneous albinism • Genetic disorder affecting pigmentation of hair, skin and eyes.	• Iris transillumination, nystagmus and amblyopia. • Notably fair skin/complexion. • White or light-colored hair.	• Careful slit lamp biomicroscope examination with retro illumination for definitive diagnosis. • Genetic testing (types I-IV).	• Sun protection (patients at risk for melanoma). • Recommend Transitions lenses. • Prescription sunglasses. • Low vision evaluation.

school, according to her grandfather. Her cycloplegic refraction was +3.00-4.00x180 OD, and +2.00-3.75x180 OS.

She had bilateral refractive amblyopia and was best corrected to 20/40 OU.

After every pediatric exam, I tell my young patients that I saw their brain and that their brain is very smart. After I recited the line to this particular patient, her eyes welled up with tears. That's when I found out that her classmates had called her "dumb." I told her she was not dumb, but that she was very smart and that her glasses would help her move to the top of the class.

After two months with her prescribed glasses, she returned with 20/25 OU vision, along with a bright smile on her face and a glowing report from her grandfather, who boasted that she'd advanced from being the last in her class to one of the top three.

Being a pediatric optometrist gives you the opportunity to change not only the sight of a child, but change the course of their lives.

The Medical Model for Kids

Primarily, children see an OD to address a refractive error, like in the above encounter. But, like adults, children sometimes develop eye diseases and you may end up saving their vision, or even their life.

Ten years ago, I was doing a routine dilated exam on a six-year-old male patient—incidentally, the patient was my nephew. He was plano in both eyes, had passed the school screening and the pediatric vision screening. However, upon dilation, I noticed a two-disc diameter fleshy tumor of the optic nerve in his right eye. After three trips to three different pediatric retinal specialists, his scans were sent to Wills Eye Institute in Philadelphia, where Jerry Shields, MD, confirmed a diagnosis of active retinoblastoma. We were fortunate, as within two weeks, the tumor had outgrown its blood supply and had become calcific tissue.

Today, he is a healthy teenage boy who still checks in with a retina specialist once every five years. This was a close call, but the potential consequences of overlooking this diagnosis could have been dire.

Although this isn't a common condition, some diseases are more common in children (*Table 1*). For example, pediatric optometrists must always be on the prowl for amblyopia, which must be caught and treated before age eight to 10 to prevent permanent visual impairment; ideally, for the best outcome it needs to be caught by age three. Aggressive occlusive and vision therapy, as well as accurate refraction ascertained from a cycloplegic refraction, are needed to ensure amblyopia is prevented.

Comanagement

Comanagement allows optometry to develop a good working relationship with pediatric ophthalmology and other interdisciplinary avenues of care, such as pediatricians, pediatric neurologists, nurses, PAs, endocrinologists and geneticists.

Pediatricians and family practice doctors benefit immensely from comanagement with optometry. These doctors aren't always comfortable treating pediatric ocular issues. For example, without a slit lamp,

Local Eye Site
JOBS IN EYE CARE

Assessment

Scientifically proven to predict performance in eye care jobs.

Hiring?

Pre-employment testing significantly increases the probability of hiring star performers.

Top scorers are proven to achieve:

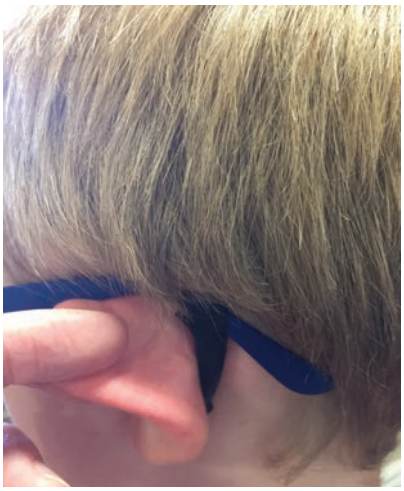
89% More Revenue

Higher Ratings

6x Lower Turnover

localeyesite.com/assessment

919.301.0200



To be a successful pediatric OD, get familiar with products specific to this clientele, such as this device that prevents frames from slipping.

it's difficult to properly diagnose the etiology of a red eye, or remove a foreign body. In regards to a red eye, most default to a broad-spectrum antibiotic, which will not be effective if the cause is viral or the result of an allergic condition, and in regards to a foreign body, most pediatricians, nurses, or PAs are not trained in how to properly remove a foreign body. It behooves you to reach out to other specialties to offer your eye care expertise.

One tool in particular has been a game changer in early diagnosis and treatment of amblyopia and refractive error: the Spot Vision Screener (Welch Allyn). We encourage general and pediatric medical clinics to incorporate this technology in every pediatric exam, regardless of age. Amblyopia can be detected even in newborns by a novice health care worker with this technology.

As optometry develops greater comfort in pediatric care, comanagement doors will open wider for the benefit of the patient and provider.

Challenges in the Chair

Now for the bad news: children can wreak havoc upon an office. Children often touch and maneuver equipment, in spite of posted warning signs. Frustrating as it is, unruliness is natural in children.

But combating unruliness in parents is the real challenge. A certain percentage of parents will actually push back against instilling eye drops in the child's eyes, claiming it burns. In those cases, properly educate the parent that the precision of the refraction and the accuracy of the health diagnosis depends on the cycloplegic dilated eye exam.

Whether pediatric patients struggle with attention deficit disorders or not, visiting the optometrist isn't exactly a day at the park. Children can quickly grow

Remember to invite pediatric patients in by preparing not only your exam lane, but your waiting area as well.

Let Your Office Reflect Your Expertise

If you're considering focusing on a pediatric patient base, obtain the proper education to do so. But one thing you might not learn in your CE courses is how to present the proper office aesthetic. Here are some updates to consider:

1. Make your office more kid friendly by including toys and games in the exam room and dedicating a corner of your waiting area to kids.
2. Consider designing a pediatric exam room. Include a Snellen chart with cartoons and lens bars, and brush up on your skills on retinoscopy with lens bars.
3. Invest in equipment that is germane to the pediatric population: a portable slit lamp, a kid-friendly tonometer such as the Icare brand and amblyopic materials such as eyepatches and brochures explaining strabismus and amblyopia.

You can use the resources provided by the AOA to immediately position your practice as a pediatric optometric practice. "Think about your eyes," and "Infant See" are public health initiatives to educate parents seeking an optometrist to provide care for their children.

impatient and begin to squirm. Here is a pearl I often use: as soon as a child sits down, give him a toy to play with. Next, raise the chair, place the phoropter in front of his eyes with the cartoons used for acuity testing preloaded onto the eye chart. I can then perform retinoscopy. If the child tries to reach up and touch the dials, remind him to keep his hands down and hang on to the toy. Smaller children will have retinoscopy performed with lens bars. I usually give children a toy in the shape of an animal, and we proceed to examine the toy's eyes—afterwards, children are often more accommodating and will allow you to perform the BIO, slit lamp and further testing . It also helps to have young children sit in their parent's lap so you can do a demonstration on the parent. This can help allay children's fears and make them more accommodating. Be sure to apply 1% cyclopentolate and 2.5% phenylephrine 45 minutes before the exam so the child is completely cyclopleged.

If a child is uncooperative on the visual acuity, retinoscope or BIO, we perform a partial exam, prescribe the cyclopentolate 1% *and* 2.5% phenylephrine to the parent and have them return another day with the child already dilated. We instruct the parent to put the drops in one hour before the appointment. The parent is instructed on proper drop installation and dosage before they leave the office.

You can also incorporate a reward system, with parental approval, where pediatric patients are offered a toy from a "treasure box" or piece of candy after the exam, if they can maintain proper behavior.

There's never been a better time to enter the world of pediatrics, and optometry is primed to take the lead in managing this patient group. One in four children has an undiagnosed vision problem, leading to everything from chronic headaches to difficulty in school or trouble reading.[1] Make sure to protect children's vision health by adding a pediatric comprehensive eye exam to your list of services. ■

Dr. Elliott is the optometric director of a high volume pediatric ophthalmology practice in Tulsa, OK, cofounder of www.take10vision.com and a continuing education speaker.

1. Kleinstein R, Jones L, Hullett S, et al. Refractive error and ethnicity in children. Arch Ophthalmol 2003;121:1141-7.

GOOD-LITE®

www.good-lite.com

Good-Lite is the strongest and most comprehensive solution to your ophthalmic testing and evaluation needs.

Why Choose Good-Lite?

MORE OPTIONS

More than 2,000 products for you to choose from

HIGH QUALITY; COMPETITIVE PRICES

We value our customers and are happy to offer the highest quality at competitive pricing!

KNOWLEDGEABLE CUSTOMER SERVICE

Our customer service department understands your needs. Our trained staff of expert ophthalmic-industry representatives will help you make an informed decision based on your goals, type of practice and patient type.

Visit Us at Booth 302 · Buy Early · Save Money · Limited Inventory

Expand your Toolbox: Integrate Low Vision Into Your Practice

Increase patient satisfaction—and your practice's bottom line—by serving this growing patient population. **By Alexis Malkin, OD**

Optometric practices are facing increased demands as the US population ages. Approximately 4% of the US population has vision impairment that is not correctable with surgery or corrective lenses, and 80% of these people are over the age of 65.[1-2] Currently, four million Americans live with vision impairment, and this number is expected to increase by 350,000 people each year.[1-2] The cost associated with vision impairment and eye diseases is expected to reach 770 billion dollars by the year 2050.[3]

Despite the rising need, vision rehabilitation remains an underused service. One report estimates that less than 10% of those who could benefit from vision rehabilitation services used them.[4] If optometrists can increase the availability of low vision services, they can improve the quality of life for their patients and reduce the overall burden on the health care system in the process.[5-6] In addition, providing low vision services can be an area for practice growth, as it often includes the prescription of multiple pairs of glasses, device sales and improved relationships with referral sources such as occupational therapists and ophthalmologists.

We all receive low vision training during our optometric education, yet only a small percentage actually incorporate low vision into our practice. In Massachusetts, where we have an abundance of eye care providers, our estimates show that we are meeting the needs of only about 16% of those people living in the state with vision impairment. This article outlines what you can do to incorporate low vision services into your practice to help meet the needs of all of your patients.

Barriers to Care

A number of issues have been cited as barriers to providing low vision care in optometric practices. For some optometrists the primary barrier is chair time. Low vision exams traditionally take more time than a comprehensive eye health and vision exam. In addition, optometrists may not feel they are up to date on their skillset or on the technology used to care for patients with low vision. Finally, optometrists often express concerns about reimbursement rates for low vision services and device sales.

From the patient's standpoint, barriers include lack of knowledge of low vision services and the benefits these services may provide.[4,7] Even if they are aware, some patients with vision impairment may lack transportation and are unable to access services. Finally, there are not enough low vision doctors to provide care to all of those who need it.

One of the challenges we face is how to define low vision. According to Medicare, low vision is defined as visual acuity worse than 20/70, visually significant scotomas, visual field loss or any combination of these three. However, most low vision practitioners define low vision as any level of vision impairment that impacts a patient's function. Many patients are not aware that their functional complaint can be addressed and do not volunteer information about difficulties they are having with a particular task. Thus, we must ask direct questions to elicit these functional complaints from a patient and begin the conversation about low vision services.

Recommended Equipment

The first two questions doctors ask

The high contrast and backlighting of an ETDRS chart is ideal for low vision patients.

when considering adding low vision to their practices is what kind of equipment they need, and how much it will cost them.

There is a difference between the ideal setup—found at comprehensive low vision centers such as those affiliated with the major hospitals and academic institutions in ophthalmology and optometry—and what a private practitioner requires to provide low vision care. Here are some essentials to get any clinician started with low vision services in their office:

ETDRS chart. This is the ideal acuity chart for low vision patients because of the high contrast and the backlighting. It can be moved to various distances from 4m to 2m and then to 1m, depending on the patient's acuity.

a. There are an equal number of letters per line, allowing the 20/400 patient to have five letters to localize with their eccentric view (EV) rather than the one found on a traditional chart.

b. The ETDRS chart is also logarithmic in its progression from the largest line to the smallest line, as well as with the letter spacing.

c. If an ETDRS chart is not available, a digital acuity chart and a Feinbloom chart are alternatives to a standard projector to measure acuities at greater levels.

Contrast sensitivity chart. Contrast sensitivity can give a better sense of a patient's functional vision than acuity alone. Reduced contrast sensitivity is an independent risk factor for reduced function in all of the visual domains except mobility.[6] There are multiple options for testing contrast sensitivity, including: Mars, Pelli-Robson and iPad apps (with calibrated screen settings).

Continuous text reading card. A continuous text card, such as MNRead or Lighthouse Continuous text card, will help determine a patient's critical print size (i.e., the print at which fluent reading slows). This is different than assessing a threshold print size or a letter acuity, as the critical print is truer to the real-world reading needs of the low vision patient.

Near vision materials. Have magazines, pill bottles, food packages and checkbooks (standard and large print) on hand to test patients' spot reading abilities.

To properly evaluate patients, clinicians must also invest in visual assistive equipment such as:

- ***High plus spectacles/prism half-eyes.*** Powers from +4D with 6 BI to +8D with 10 BI will cover a good range of visual needs.
- ***Hand magnifiers.*** A range

of powers from about 8D to 20D will meet the needs of many low vision patients; LED hand magnifiers tend to be the most well received, but non-illuminated hand magnifiers can be useful for their portability.

- ***Stand magnifiers.*** Consider starting with a dome/bar magnifier and one to two LED stand magnifiers. Don't forget to think about the add power presbyopes require to use a stand magnifier.
- ***Telescopes.*** Monoculars ranging from 2x to 6x are good starting points. Patients requiring more power will likely need more advanced low vision services. Also invest in a 2x to 3x binocular system for television viewing.
- ***Video/digital magnification.*** Demonstrating a portable or desktop CCTV can help patients understand the full range of low vision aids available to them. Although you do not need to obtain all of the brands and styles, familiarize yourself with the various features of the different devices so you can discuss them with the patient.
- ***Filters.*** Keep a range of filters in your optical or low vision supply, especially yellow, plum, amber and gray. Patients with all levels of visual impairment experience symptoms of both indoor and outdoor glare. Having several options available in office will help you determine the most appropriate filter for the patient's comfort and visual function.
- ***Non-opticals.*** Large print checkbooks, bold-lined paper, bold pens and typoscopes are all useful tools to demonstrate to patients.

A Mars contrast sensitivity chart provides a better sense of this patient's functional vision than acuity alone.

Clinicians may also choose to keep some visual assistive equipment in stock so that the devices can be readily dispensed to the patient.

The Low Vision Exam

The low vision functional history is one of the most important components in a low vision exam. We can break the history down into five functional domains: reading, visual information/seeing, mobility, activities of daily living and driving (*Table 1*).

Reading. Most patients who present to low vision clinics come with a chief complaint of difficulty reading.[8] Determining the patient's preferred type of reading will help guide the low vision exam and the strategies used for rehabilitation. Inquire as to whether the patient has primarily spot reading goals (labels, mail, bills) or if they also have continuous text goals (newspaper, books, magazines). Knowing whether the patient is reading electronically (computer, tablet, cell phone) or if the patient reads only standard print materials will impact your treatment approach as well.

Visual information/seeing. This encompasses the patient's ability to see faces at a distance, to see a television and also how the patient functions in different lighting conditions. Patients may express difficulty adapting to changing lighting conditions or with their ability to tolerate indoor or outdoor glare.

Mobility. A decrease of just one or two lines of acuity (from 20/20 or 20/30) increases the risk of falls significantly.[9] Patients may not always understand the interaction between their vision and their mobility, and we can play a very important role in educating the patient and helping prevent future falls. It is essential to ask all low vision patients about their history of falls, fear of falling and what, if any, mobility aids they are using. Referring a patient to an orientation and mobility specialist or recommending they seek an evaluation with a physical therapist may make a substantial improvement to that patient's ability to continue to live safely and independently.

Activities of daily living. Asking patients specific, directed questions about their daily living activities—cooking, cleaning, bathing, dressing and medication management, for example—can help you create the best rehabilitation plan. For patients who have significant

reported difficulty in this particular domain, a referral to low vision occupational therapy can be beneficial.

Driving. Whether a patient has vision impairment or not, we are frequently called upon to sign off on driver's licenses. As low vision specialists, we must be familiar with state laws and understand the intricacies of the medical review board process to best advise patients. In addition, identifying certified driving rehabilitation instructors in the community can help you make more informed decisions about a patient's safety on the road.

The intricacies of a low vision exam go beyond the scope of this article, but keeping these functional domains at the forefront can provide a general guideline for conducting a low vision history and identifying patient goals. Moving through the exam based on the patient's goals will minimize chair time without sacrificing your ability to provide low vision care.

After the patient history, the fundamentals of a low vision exam are similar to the basics of a standard ocular health evaluation.

All low vision exams begin with a measurement of visual acuity at both distance and near. Supplemental testing may include contrast sensitivity and Amsler grid/mapping of central scotomas. These tests are useful in determining the appropriate magnification requirements for patients. They are also helpful when educating patients and their families about the visual condition and visual function. Visual field testing such as facial fields, confrontation fields, Goldmann visual fields or standard perimetry may also be necessary. Understanding the extent of a patient's peripheral field loss will guide the rehabilitation plan and provide valuable information to the members of the rehabilitation team, including the occupational therapist and orientation and mobility specialist.

Low vision refractions are best performed through a trial frame, allowing the patient to eccentrically view with greater ease. Trial frames also facilitate the larger lens changes needed to use the principle of just noticeable differences (JND). Patient education about how much or how little change there is to the glasses Rx will aid in success during the remainder of the low vision exam. Patients are often optimistic that there will be significant changes in their vision after refraction. If there is a minimal change, it is important that the patient understand so that they can be open-minded to the rehabilitative strategies that you will explore.

Trial frames are a must for low vision exams, as they help patients eccentrically view with greater ease.

Rehabilitation/ Team Management

Integrating low vision into your practice requires more than just your expertise—a team approach is key for patients with low vision. To be successful in providing low

Table 1. Addressing Five Functional Domains During the Exam

Functional Domain	Suggested Questions in Case History
Reading	- Are you an avid reader?
Visual Information	- Do you have trouble recognizing faces? From what distance? - Do you have difficulty adapting to variable lighting conditions?
Mobility	- Have you had any falls in the last two years? - Are you afraid of falling?
Activities of Daily Living	- Have you had any accidental cuts or burns while cooking? - Do you have difficulty with medication management?
Driving	- When was the last time you drove? - Are you limiting your driving? - Have you had any accidents or near misses?

vision care, become familiar with the available resources in your community. Each state has resources for those who are legally blind, and, often, these services extend to those with vision impairment as well. Either the department of education or the department of disability usually runs these services.

You must also be prepared to refer patients for low vision occupational therapy as needed. In order to see a patient, an occupational therapist requires a written referral from you. Many occupational therapists have forms that include the covered diagnosis codes as well as any other information essential to their evaluation. You will also sign off on the plan of care as well as the progress notes as the patient continues through their rehabilitation. Whether the occupational therapist works with the patient in the home or in the clinic, they can address activities of daily living the patient struggles with most. Occupational therapists may work in conjunction with speech and language pathologists and physical therapists to provide comprehensive care to the patient with low vision. Occupational therapists can assist with developing strategies to reinforce new learning, home modifications and problem solving. These skills help patients remain independent, even as their visual condition changes. This team approach to care ensures your patient's needs are being met on all fronts.

Billing and Coding

Billing for low vision exams is less complicated than you think. Because all low vision patients enter with a medical diagnosis, you will most likely use the patient's medical insurance and will bill with an E/M code for the visit. The low vision visit is typically not billed on medical complexity but instead is based on the face-to-face time spent with the patient.[10] Medicare provides guidelines for visits during which you spend extended time on education and counseling. To be compliant with this type of billing, document the amount of time spent with the patient and what percentage of the time was spent on education, counseling and coordination of care. These codes are used most frequently when the face-to-face time component is greater than 50% of the exam. It is also important to educate the patient that the refraction is not a covered service in a medical visit, even though it's part of the low vision exam. Low vision refraction fees vary by practice but are often more than the standard refraction given the extra time and complexity. Do not include the refraction in the face-to-face time, since you will bill that portion of the exam separately.

Some insurances are beginning to provide coverage for low vision aids, particularly when patients have vision plans. It is important to discuss this possibility with patients and help them understand, for insurance purposes, the difference between standard spectacles and visual assistive equipment.

Staff Training

Having a well-trained staff is the key to any successful practice, and a low vision practice is no different. Staff responsibilities will shift, and everyone has to be prepared to answer a whole new battery of questions. Here are some tips to ensure your staff is ready to take on the challenges associated with low vision patients:

Technician training:

- Ask patients about their functional vision.
- Ask how patients are coping with a diagnosis.
- Assist with setting patient expectations regarding the low vision exam.

Front desk training:

- Schedule low vision when the patient is referred to retina/glaucoma/cornea specialist for a chronic ocular disease.
- Educate the patient on the phone that the doctor will be spending extra time looking at functional vision.
- Educate the patient that the doctor is going to do a very different exam than the standard eye exam, which is why the appointment needs to be on a specific day/time.

Continuous reading charts help simulate the real-world reading needs of patients with low vision.

Optical training:

- Be prepared to answer questions about replacing magnifier batteries.
- Have a full selection of tints available and schedule time to work through these options with the patient.
- Become familiar with Fresnel lenses and working distances of high adds.
- Prepare for the possibility that some patients may not be 20/20 when they pick up their new glasses.

Although all optometrists have the training to provide low vision care, not everyone decides to become a low vision specialist. Integrating some components of low vision into your practice can help with patient satisfaction and will allow for an easier transition to more advanced low vision care as the patient's disease progresses. Patients are very appreciative when you take the time to provide the detailed trial frame refraction, can provide information about apps/assistive technology and can refer the patient for home safety assessments. Take advantage of the resources in your community, get to know local low vision specialists for the more complex patients and increase patient satisfaction. ■

Dr. Malkin is an assistant professor of optometry at the New England College of Optometry (NECO). She is a graduate of Emory University and completed her optometry training at NECO, where she graduated salutatorian. She completed her residency at the Northport VA Hospital with a focus in primary care optometry, low vision rehabilitation and vision therapy. Dr. Malkin completed the Lions Vision Rehabilitation Fellowship at Johns Hopkins in 2010. She is particularly interested in clinical research, including outcome measures and improved access to low vision care. She has published numerous peer-reviewed manuscripts in low vision rehabilitation and clinical outcomes and has lectured throughout the United States and internationally on a variety of topics including patient outcomes and technology in low vision.

1. Congdon N, O'Colmain B, Klaver CC, et al. Causes and prevalence of visual impairment among adults in the United States. Arch Ophthalmol. 2004;122(4):477-85.
2. Massof RW. A model of the prevalence and incidence of low vision and blindness among adults in the U.S. Optom Vis Sci. 2002;79:31-38.
3. Prevent Blindness. Future of Vision report. The Focus on Eye Health National Summit. 2014. Available at http://forecasting.preventblindness.org.
4. Leonard R, Gordon AR. Statistics on Vision Impairment: A Resource Manual. Research Institute of Lighthouse International. 2002.
5. Stelmack JA, Tang C, Reda DJ, et al. Outcomes of the Veterans Affairs Low Vision Intervention Trial (LOVIT). Arch Ophthalmol. 2008;126:608-17.
6. Goldstein JE, Massof RW, Deremeik JT, et al. Baseline traits of low vision patients served by private outpatient centers in the United States. Arch Ophthalmol. 2012;130:1028-37.
7. Casten RJ, Maloney EK, Rovner BW. Knowledge and use of low vision services among persons with age-related macular degeneration. Journal of Visual Impairment & Blindness. 2005;99(11):720-4.
8. Brown JC, Goldstein JE, Chan TL, et al.; the Low Vision Research Network Study Group. Characterizing functional complaints in patients seeking outpatient low vision services in the United States. Ophthalmology. 2014;121:1655-62.
9. Abdelhafiz AH, Austin CA. Visual factors should be assessed in older people presenting with falls or hip fracture. Age and Ageing. 2003;32:26-30.
10. Verhovshek JG. Go beyond the basics of time-based E/M coding. AACP. Available at www.aapc.com/blog/4615-go-beyond-the-basics-of-time-based-em-coding. Accessed September 27, 2016.

Build a Specialty Contact Lens Clinic From the Ground Up

Now is the time to invest in this ever-growing area of expertise—your patients and your practice will thank you. **By Brooke Messer, OD**

Building a niche practice—any niche—is a challenging process that can yield emotional and financial rewards. A specialty contact lens practice comes with its own unique challenges, including managing the medical condition of a patient, navigating complex insurance coverage and designing and dispensing a custom product. My team and I have learned a lot from our experiences building our contact lens practice, and each year we find ourselves a little further ahead.

To have a successful specialty setting that optimally cares for patients, employees and doctors, you have to balance both clinical expertise and practice management know-how. While no one can argue the clinical skills need to be in place from the beginning, it is equally as important to ensure proper office protocols are in place. Staff education, insurance billing protocols, handling of custom contact lenses, schedule management and contact lens fitting documents or contracts are just a few aspects that are crucial in the management flow of a specialty contact lens practice.

A multifunctional instrument that can assess topography, meibography and tear layer components provides a wide range of uses while maintaining a small footprint in the office.

When I asked myself, "If I were to start the practice all over again, what do I absolutely have to have in place to succeed?" I also considered parts of my practice that I wish were more efficient. While not a comprehensive list, here are some recommendations I have compiled from my experience to help you get your dream contact lens practice up and running.

Make Connections

A beautiful thing about specialty contact lenses is the community is so small. There are many resources

available at your fingertips for both fitting and practice management techniques. To get started, reach out to those who have been there before. Relationships within the contact lens industry are particularly valuable and mutually beneficial. Attend events sponsored by the contact lens sections of major meetings, such as the American Academy of Optometry or American Optometric Association. A growing number of contact lens-specific meetings, such as the Global Specialty Lens Symposium and Vision by Design, are great places to connect with other like-minded practitioners for advice and guidance, not to mention the clinical training through CE courses. You are sure to meet an established contact lens fitter who is willing to share a few war stories and offer advice.

Additionally, connect with at least three specialty lens labs and get to know their consultants. Spend some time on the phone or at their exhibit hall booth and establish a relationship. Choose a lab that can do several types of gas permeable lenses, such as corneal spheres, bitorics and multifocals. Another lab can assist in your scleral lens contact lens fittings. Finally, establish an account with a custom soft lens laboratory.

Other lens options you might want to consider are orthokeratology and prosthetic contact lenses. Many labs can manufacture all types of lenses, but you will learn more working with consultants from a variety of backgrounds rather than a single group.

With this laboratory base, you will be able to order the right lenses for a large percentage of your patients. Great resources for researching lab options are the Gas Permeable Lens Institute (www.gpli.info) and the Scleral Lens Education Society (www.sclerallens.org).

Train Staff

Starting a specialty brings a new dimension to your office, and you'll want to make sure your staff is prepared and on board with the changes in office protocols and patient communication. Take the time to educate your team about the types of lenses you'll be fitting, the patient groups that might enter the practice and the ocular conditions that could benefit from specialty contact lenses. Also, review the basics of the types of contact lens solutions, eye drops, salines and application and removal tools you may be recommending. It's helpful for some of your staff to be familiar with application and removal techniques of soft, corneal GP, hybrid and scleral contact lenses. Spending this time up front will save you, as the prescriber, many hours of phone calls and emails answering questions about contact lens solutions, wear times and whether or not a patient needs to return to the office for a follow-up appointment.

Update Office Protocols

Starting a specialty contact lens clinic within your practice is an investment with great potential. Understanding a few key aspects of running a practice using custom lens technologies will help protect those investments and allow the office to prosper. Here are four protocols to enact that will ensure a smooth specialty lens fitting process:

1. Patient information. The first protocol that should be in place is a fitting services brochure or contract, outlining both the office and patient responsibilities during the fitting process. This should be introduced and referred to during the initial visit. It is important for the patient to know the potential out-of-pocket costs and number of visits required to complete the lens evaluation. I have learned that patients are willing to pay out of pocket for high performing lenses and return for multiple visits when given proper notice. They do not appreciate, however, having to pay an unexpected amount or sitting through multiple evaluations when they had different expectations initially. The fitting brochure should include the price of services, if the office will bill medical or vision plans, when payments are due and if follow-up examinations are considered part of a global fitting period or as office visits billed to insurance. If there is a global fitting period, or a contact lens warranty period for remakes, the timing should be included in the

Simple Adjustment, Huge Impact

Investing in technology can go a long way to building trust and loyalty with your patients. For example, a 38-year-old male with keratoconus presented to our clinic already wearing scleral contact lenses successfully. His only complaint was redness on his nasal conjunctiva during lens wear.

Upon examination, we found his scleral lenses were compressing on a nasal pinguecula in each eye. With a simple depth and diameter measurement with an anterior segment OCT, we were able to order a microvault in a new pair of scleral lenses with near perfect dimensions, which relieved his complaints of redness.

While I could have estimated the dimensions, the photos were great tools to not only efficiently prescribe his new scleral lenses, but also educate him on exactly what was occurring and how we fixed it.

He has told his friends and family about his great experience, and that "we're the office for great contact lenses." As you know, word-of-mouth referrals are priceless.

fitting contract. Consider including examples of acceptable solutions and artificial tears that are compatible with their contact lenses, useful website links for application and removal tips, as well as any emergency contact numbers in case of needed care after hours.

2. Fitting information. To ensure you have the necessary information on the initial evaluation, develop a fitting protocol with the same basic information for every potential custom contact lens patient, regardless of condition. For example, a basic evaluation for someone with keratoconus could include history, visual acuities, refraction, slit lamp and topography. Adding technology such as endothelial cell counts, pachymetry and abberometry are helpful pieces of information.

A clinical pearl I have learned is to always take a baseline refraction for best-corrected spectacle acuities, even if vision is very poor, and be sure to document the best-corrected acuity obtained with a diagnostic contact lens plus the overrefraction results. Occasionally, insurances will request proof of significant vision improvement with a custom contact lens for lenses to be covered. Remind patients that you understand their vision is poor, but the measurements are for insurance purposes and baseline measures.

After baseline testing, make sure patients don't have other questions about the fitting period, any available warranties and exchanges for lenses, and how any potential out-of-pocket costs are handled. Once the patient agrees to the fitting protocol, you can begin the contact lens fitting with diagnostic lenses; the visit could be considered complete if you decide to order empirically. Some practitioners prefer the patient come back for a second fitting visit, while others perform the fitting on the same day. In our office, we gauge a patient's interest in a screening visit only vs. a lens fitting and book time accordingly.

3. Organization. Another key office protocol in managing your contact lens clinic is how to organize and manage lenses delivered to the office. The first step in this process is understanding your laboratory's warranty process. Each lab has a number of lens orders and a certain number of days adjustment lenses can be ordered for little-to-no cost after purchasing the first set of lenses. Some labs require return of each lens set to send adjustment lenses, while others require return of lenses only if the entire order is cancelled within the warranty period. Therefore, it's imperative you keep all lenses accounted for to return lenses needed for credit. Without a proper lens management system in place, fitting specialty contact lenses can get very expensive. A posted chart referencing each lab's exchange policies can be helpful in deciding how lenses should be organized.

Some examples of lens management systems could include a tray or file system, where flat-packs or small bags are kept and labeled with the lens order date, reference number or some other identifying method. I find it helpful to keep the lens invoice with the previous lenses for quick reference of previous and current lens parameters. Once the fitting is complete, we scan all invoices to the patient record. This makes it easy to keep track of which lenses the patient most prefers; it also simplifies returns to the laboratory, if needed. We also keep a flow sheet with the lens tray for quick notes to others in the office. At the end of each visit, anything the rest of the office should know for the patient's next visit can be noted, such as needing to return previous lenses for credit, if the lens fitting is finalized or if the patient has a balance after insurance processing.

As offices move toward paperless records, managing this type of inventory can get tricky, and it is vital to have a solid system in place. A single missing lens, or not returning lenses under warranty in a timely manner can cost the office hundreds of dollars each month. It is not unreasonable to dedicate a staff member to this project as your patient base wearing specialty contact lenses grows.

4. Backup plan. Lastly, be sure to create an office policy on how cases are handled when the patient

Educate and Preserve

When working with specialty contact lenses, we often share good news with patients by showing them they can still see well despite their history of keratoconus, corneal scarring or other condition. However, we occasionally have to share less than favorable findings and educate patients about the maintenance therapy necessary to protect their eyes long term.

For example, a 28-year-old female contact lens wearer mentioned moderate dry eye complaints and decreased contact lens wearing time. We noted significant meibomian gland dysfunction and lid margin telangiectasia. We educated her in office—using meibography photos from our multifunction topographer—about the damage that has already occurred to her meibomian glands, and that diligent maintenance is needed to protect the remaining glands. By comparing her photos to those of normal gland structure, she realized the importance of her care regimen and has committed to a follow-up schedule and filling her prescriptions.

Visit us at
Academy
2016 Anaheim
Booth #600

PRODUCTS AS STRONG AS THE PEOPLE BEHIND THEM.

Built to support your patients, your practice and an entire country.

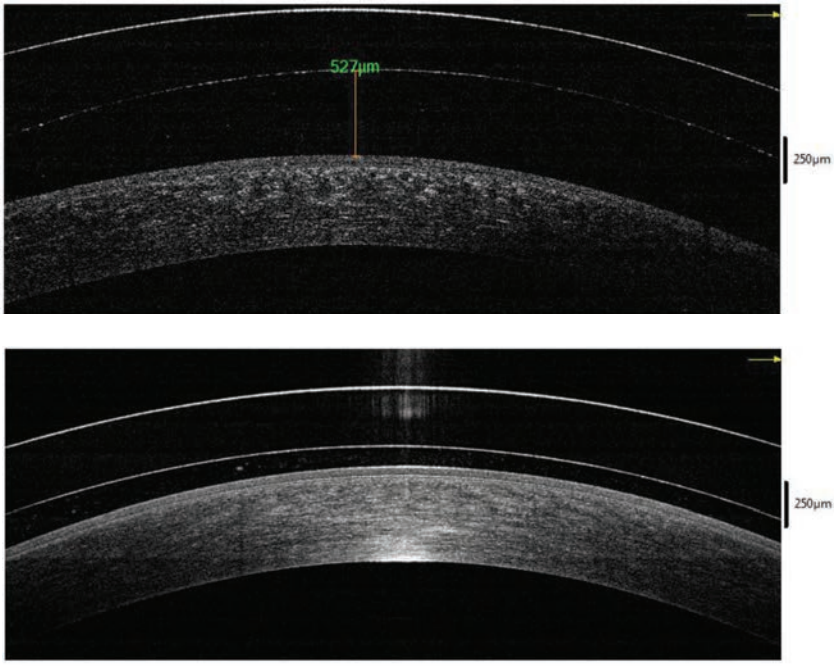
Reliance is more than just our name. It's at the very core of who we are, who our people are. Our industry-leading chairs, stools, exam equipment and accessories are manufactured in the heartland at the hands of dedicated, skilled craftsmen with materials from over 150 U.S. suppliers. Which is why practices around the world rely on us for unrivaled durability, stability, style, and comfort.

Learn more about the reputation we've built with toughest materials and the strength of the American workforce at BuiltToSupport.com.

800-735-0357

RELIANCE INTERNATIONAL

BUILT TO SUPPORT™



OCT images such as these can assist in scleral lens assessment at the corneal apex and limbal areas. The top image depicts extra vault, while the bottom image shows proper vault.

decides they do not want to go forward with the lens fitting. This policy should address if there is any potential for refunds on returned lenses and how service fees are handled if the fitting is not yet complete. Thankfully, this happens far less than patients completing their fitting, but when you do have this situation, it's much easier to handle with a protocol in place beforehand, the entire staff is aware of the policy and everyone knows how to explain it to the patient. A brief explanation of this policy should be in the contact lens fitting brochure or contract.

Build Clinical Skills

Managing the clinical side of the practice is the easy part of a specialty contact lens practice. With the available webinars, hands-on workshops and consultation teams, information is always readily available to assist you in fitting custom contact lenses.

The first step in learning the skills needed to fit custom contact lenses is identifying the right candidates. In general, most myopic children are great candidates for orthokeratology, and many patients with irregular corneas will appreciate the vision and comfort of scleral contact lenses. However, there are groups of patients that are not ideal candidates for certain lens designs. Patients with a low endothelial cell count after a corneal transplant surgery are not ideal candidates for scleral contact lenses due to potential for corneal edema. Numerous resources, including the websites mentioned previously, consultants, research databases and lectures at state and national meetings will often review the ideal candidates for each particular lens design.

With any lens design, consider starting with and learning from a patient who is considered mild in the condition you are treating. For example, begin your orthokeratology fitting with a -2.00 myope rather than a -7.00, as the need for complex troubleshooting techniques will be lower with such a patient. If you would like to start fitting scleral lenses, consider starting with a patient who has mild keratoconus. These types of patients will help you learn and gain confidence at the same time. It is also helpful to discuss your fit with the consultation team, to make sure you are completing the necessary clinical assessments. As you complete more fittings and master the common patient symptoms and how an appropriately fit lens performs, you can broaden your patient base to those with more severe forms of their conditions.

Specialty contact lenses are still contacts, and the fitting assessments should have the same flow as evaluation of a non-custom lens. Measure vision, complete an overrefraction and fitting assessment and evaluate the cornea with sodium fluorescein after removing the lenses. The key is being aware of the evaluation points of the fit and recognizing when findings indicate a refit or routine monitoring of corneal health.

Invest in Technology

Some additional tools can make you more efficient at fitting custom lenses and can assist in troubleshooting difficult fits. These procedures are also billable to medical insurances when a medical diagnosis and applicable plan is involved. Consider investing in these instruments to build your practice and knowledge of how specialty contact lenses affect the ocular surface:

• *Corneal topographer:* This tool is a must if you plan to fully manage corneal diseases and dystrophies. For keratoconus in particular, being able to manage not only the contact

The M&S Smart System®, a Perfect Balance of Quality and Quantity

Backed by experience, precise engineering and attentive support.

Simply put, the **Smart System 20/20** gives you more than any competitor: more features, more reliability, more support and more efficiency.

For over 25 years, we've been focused on one thing and one thing only: developing the best visual acuity testing system in the industry. That means a feature-rich, efficient, long-lasting system that is supported by trained technicians and attentive customer service personnel.

The Smart System 20/20 USA interfaces easily and dynamically with the Topcon CV-5000S

Our recent collaboration with Topcon USA has resulted in a dymanic interface with the CV-5000S Computerized Refraction System. The interface is completely flexible; choose from the complete Smart System 20/20 module or just the modules you use most. Seamless and easy.

See us at the Academy of Optometry Meeting, Booth 521!

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

YOUR FUTURE IN FOCUS

Your job is about more than providing good patient care. To succeed, you have to navigate changing healthcare policies, keep up with the latest clinical advancements, manage your staff and run a successful business. Zero in on the topics impacting every aspect of your practice and career at SECO 2017, the largest conference in the world for optometric professionals and staff, and let us bring it all into sharp focus for you.

SECO2017

WHERE SIGHT MEETS VISION™

MARCH 1-5 | ATLANTA, GA

REGISTER NOW AND SAVE!

Visit www.seco2017.com for more information and start planning today.

lens fitting but also monitor for progression is key. A topographer is also especially helpful in troubleshooting and monitoring orthokeratology fittings. Topographers that can scan the posterior cornea, also known as the posterior float, can be particularly beneficial in explaining why a patient with an irregular cornea and little-to-no corneal scarring has limited visual acuities. New technologies in some topographers allow us to scan the scleral shape to maximize the shape design of the landing zone curves of a scleral lens.

• ***Anterior segment OCT:*** This is a fantastic tool to assess scleral contact lenses beyond what we can see with the slit lamp. Quantifying corneal and limbal vault is remarkably helpful in troubleshooting, as is monitoring for corneal edema over time. I find this instrument particularly helpful in managing patients with corneal transplants to minimize the lens vault, which maximizes oxygen transmission while ensuring complete lens clearance over the graft-host junction.

• ***Specular endothelial microscopy:*** Specular microscopy is another great baseline and troubleshooting measure when working with patients wearing specialty contact lenses. Particularly helpful on patients wanting to wear scleral lenses, these measurements can help you decide if the cornea can handle a scleral lens system without becoming edematous.

Educating Others About Your Practice

There are a few final pieces to add to your specialty practice, especially if you plan to work with other local medical professionals. Stock up on basic brochures about conditions and procedures such as keratoconus, corneal transplants, orthokeratology and scleral lens fittings to quickly answer patient questions and provide a resource they can take with them. A professional brochure about your practice is a perfect way to build referrals from other local optometrists, ophthalmologists and other medical professionals.

As your relationship with local professionals grows, prepare a template to send back to referring providers with information about your findings and the plan for your mutual patient. Respectful comanagement will go a long way in your continued practice growth. Much like our ophthalmology colleagues after cataract surgery, be sure to recommend patients back to their referring provider for services such as back-up glasses and red eye evaluations, especially in instances not related to their contact lens wear.

The Sky's the Limit

As contact lens technology continues to grow, so will our ability to fit lenses with more precision and efficiency. We are already getting a taste of measurements that can customize lens optics to maximize vision, add prism and control aberrations. One day specialty contact lenses may become mainstream in monitoring medical conditions such as diabetes and glaucoma. Thankfully, our offices are ready to handle these innovations now. ■

Dr. Messer received her doctor of optometry degree from Southern California College of Optometry, and completed a cornea and contact lens residency. She now practices in Edina, MN, with special interests in scleral, multifocal and orthokeratology contact lenses.

Dr. Messer is a consultant for CooperVision and Precilens, has research funding from Bausch + Lomb and is a Residency Forum Coordinator for Alden Optical.

BioDOptix AMNIOTIC MEMBRANE IMPROVES POSTOPERATIVE VISUAL OUTCOMES

BY:
AMANDA K. DEXTER, OD

Optimal post-refractive outcomes require a healthy ocular surface. In many instances, the surgeon may complete a flawless, routine laser procedure; however, the desired visual result only will be achieved during the optometric comanagement process. This scenario frequently occurs in the postoperative comanagement of photorefractive keratectomy (PRK) patients.

PRK typically demonstrates a high level of safety and efficacy, and may be employed to treat myopia, hyperopia and astigmatism. Most patients who undergo PRK recover quickly and without incident; however, there are some instances in which the postoperative healing process does not progress as anticipated. Disruption in any part of the intricate healing cascade can yield a persistent epithelial defect or subepithelial corneal haze, or may even precipitate refractive error regression.

Topical medications applied to the eyes during both the intraoperative and postoperative period regulate the wound-healing process, as well as limit undesired side effects. However, complications may still occur, necessitating further action. In this context, the comanaging optometrist plays a critical role in determining which secondary intervention is most likely to facilitate a swift recovery and deliver a high-quality visual outcome.

As previously noted, optometrists must be prepared to manage persistent epithelial defects following photorefractive keratectomy. The PRK procedure requires removal of the corneal epithelial layer by either chemical dissolution or mechanical debridement. The excimer laser is then applied via Bowman's membrane to treat the refractive error. Mitomycin C may be topically administered onto the denuded surface, followed by the placement of a high-oxygen-transmission bandage contact lens. Typically, the corneal surface re-epithelializes under a bandage contact lens within five days. But, when the surface does not heal in a timely and uncomplicated manner, the central epithelium usually is irregular or absent, and the patient complains of discomfort and/or poor vision.

If the patient is uncomfortable or unhappy with their postoperative outcome, a potential treatment option is the placement of an amniotic membrane. The amniotic membrane serves as a biological contact lens for the cornea. Its presence acts as a basement membrane—a scaffold for the progenitor epithelial cells to grow across, as well as an attractant for the patient's own limbal stem cells to collect within.

The amniotic membrane also helps to temper and control the inflammatory cascade via cytokine release modulation. Of equal importance, it suppresses the release of transforming growth factor beta (TGF-B), which in turn inhibits myofibroblast differentiation—the cell type responsible for the formation of scarring or postoperative stromal haze. Further, amniotic membrane provides a microenvironment that is conducive to nerve growth regeneration and overall patient comfort.

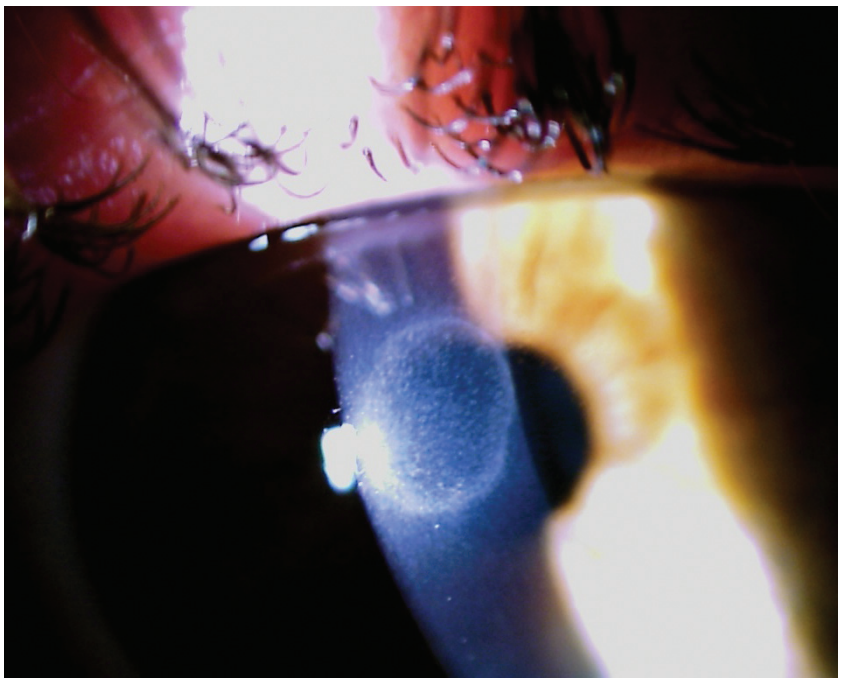
The aforementioned properties clearly illustrate why amniotic membrane use is a more effi-

CASE REPORT: PERSISTENT EPITHELIAL DEFECT FOLLOWING PRK

A 24-year-old male presented with a refractive error of -2.00 OD and -2.00 -0.75D x 180 OS at one-day follow- up after undergoing a routine photorefractive keratectomy procedure. His right eye healed uneventfully during the subsequent five days, and rapidly achieved an uncorrected visual acuity of 20/20. However, the left eye exhibited a central corneal epithelial defect that severely prolonged the healing process. For the next 17 days, the patient was managed with topical medications and frequent bandage contact lens replacements.

On day 18 post-op, the patient exhibited a 2.5mm epithelial defect with mild central corneal haze OS. The thickened and hazy epithelium was gently debrided around the defect edges in the surgery room. Then, a BioDOptix® amniotic membrane was placed on top of the cornea and was allowed to dry before being covered by a bandage contact lens. The patient continued his topical antibiotic regimen.

Within one week, the patient's epithelium had closed and the amniotic membrane had dissolved OS. The bandage contact lens was removed. Two weeks after amniotic membrane implantation in the patient's left eye, his uncorrected visual acuity measured 20/20 OU.



This patient presented with a persistent epithelial defect in his left eye following uncomplicated photorefractive keratectomy. After more than two weeks of nonresolution secondary to topical treatment and bandage contact lens use, a BioDOptix® amniotic membrane was placed in his eye. Within two additional weeks, the epithelial defect was completely resolved and the patient's final visual acuity measured 20/20 OU.

cacious therapeutic option for postoperative refractive surgery patients than a simple, plastic contact lens alone.

Historically, the rate-limiting step to using amniotic membranes in the office setting was accessing a low-temperature storage or keeping fresh tissue in a cost-efficient manner. Fortunately, however, sufficient cold freezer storage or the limited shelf life of fresh grafts may not pose quite the challenges they once did.

BioDOptix® is a dehydrated amniotic membrane that can be stored on a shelf for five years at ambient temperature, and is easily made available when needed. In this instance, the membrane is hydrated simply by adding moisture. BioDOptix® may be secured on the cornea by placing a bandage contact lens over it. Within one week or less, the amniotic membrane will dissolve and the bandage contact lens can be removed. A large-diameter plastic ring to hold the amniotic membrane is not required, and thus the comfort level is dramatically improved.

BioD LLC is a vertically integrated company that makes BioDOptix®. BioD recovers the amnion from prescreened, healthy, live donors during cesarean childbirth. These tissues are processed into their dehydrated state and terminally sterilized for final distribution to the physician's office. The acquisition cost of BioDOptix® is affordable for doctors and is a highly effective, in-office solution to optimize corneas and achieve superior visual results.

Thanks to Mihir Y. Parikh, MD, of NVISION Eye Centers in San Diego, for his contributions to this article.

An Atlas of Conjunctival and Scleral Anomalies

Become familiar with the common clinical presentations of these anterior segment ailments. **By Marc Bloomenstein, OD**

Two of the most protective structures of the ocular system are the conjunctiva and sclera. The conjunctiva, a thin layer of tissue lining the eye and eyelids, contributes to homeostasis of the tear film, provides a layer of protection from foreign material and wards off infection. The sclera, a dense connective tissue made of collagen and elastin, encapsulates the eye, giving it structure and rigidity. Anteriorly, the scleral connects to the cornea at the limbus. Posteriorly, it merges with the meninges at the optic nerve, penetrates the globes and joins with choroidal tissue from the lamina cribrosa.

The integrity of the conjunctiva and sclera is crucial for healthy eyes, and when it is compromised by abnormalities or inflammation, the ocular system quickly becomes chaotic. As primary eye care providers, we often have patients present with symptoms of disorders that affect these structures, and it is our job to provide timely intervention.

This atlas highlights the clinical presentations of some of the most common conjunctival and scleral anomalies so you can quickly identify them in your chair. Prompt diagnosis and treatment will ensure your patients are on the road to recovery in no time.

PINGUECULA

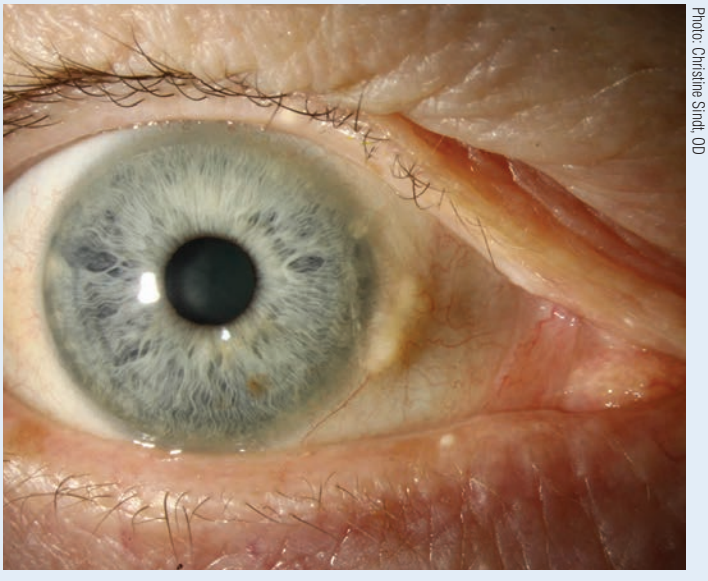


Photo: Christine Sindt, OD

Pinguecula is a common nonmalignant, raised lesion that is most often in the interpalpebral bulbar conjunctiva. This yellowish lesion is a degeneration of elastic tissue and subepithelial collagen with hyalinized connective tissue that does not often involve the cornea.

The etiology appears to arise from the effects of environmental elements such as wind, dust and UV light exposure. Research suggests the more common nasal side occurrence is related to the light passing medially through the cornea, which focuses on the nasal limbus, whereas the nose may reduce the intensity of the UV light to the temporal limbus. Other risk factors include age, gender (M>F), smoking, proximity to the equator and diabetes mellitus.[3-5]

Once the pinguecula is formed, the elevated nature will create symptoms similar to dry eye.

PEMPHIGOID

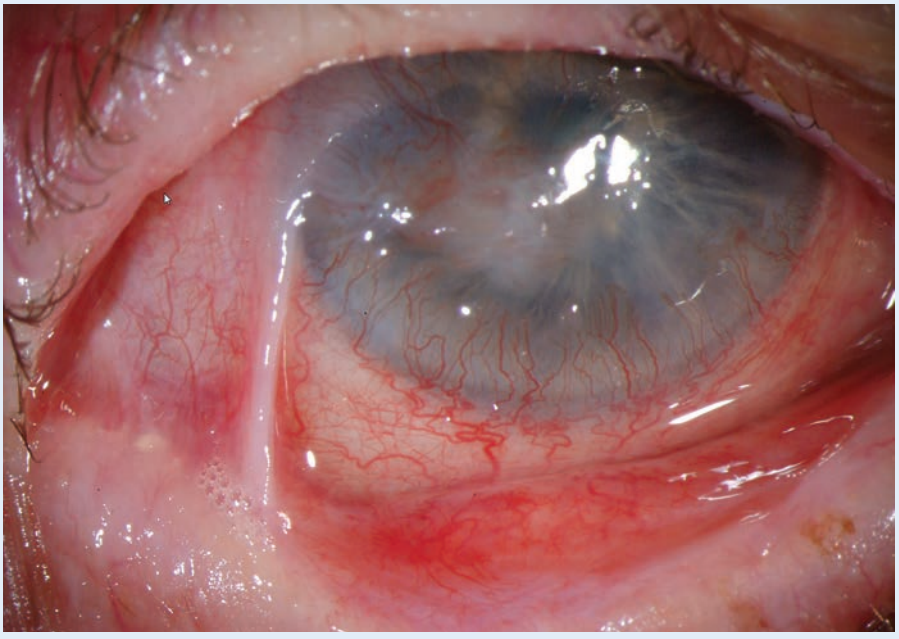


Photo: Christine Sindt, OD

Ocular cicatricial pemphigoid (OCP), commonly known as a subtype of mucous membrane pemphigoid, is a bilateral, progressive autoimmune conjunctivitis, leading to cicatrization and shrinkage of the conjunctiva with opacification of the cornea.

Early symptoms of OCP are hyperemia, discomfort, itching and discharge. However, there are many different stages of this disease, and progression leads to eyelid—most notably trichiasis—and corneal damage and sometimes blindness.

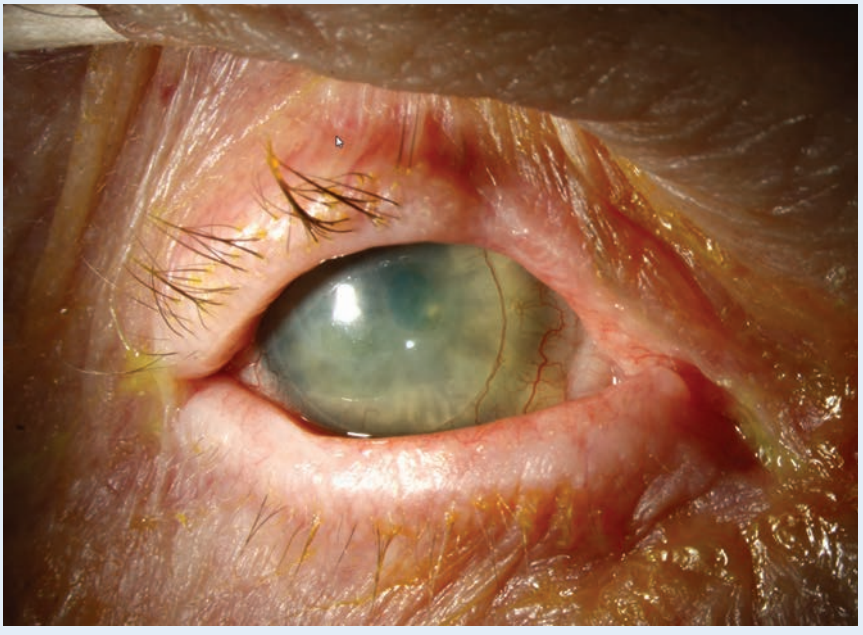


Photo: Christine Sindt, OD

The initial presentation of pemphigoid is often a nonspecific hyperemia, and commonly appears as a chronic conjunctivitis or tear reduction. Without discharge in certain quadrants, the condition progresses to: symblephera (adhesions between the tarsal and bulbar conjunctiva); trichiasis; keratoconjunctivitis sicca; corneal neovascularization, opacification and keratinization; and conjunctival shrinkage. The latter represents end stage, with extensive adhesions of the lid to the globe. Chronic corneal epithelial defects can also lead to secondary bacterial ulceration, scarring and, due to restricted movement and opacification of the cornea, loss of sight.

The differential diagnosis of progressive conjunctival scarring includes previous radiation exposure and atopic disease. Medicamentosa, sequelae of medical allergic response, can result in a psuedopemphigoid, the clinical appearance of which is identical to that of OCP, but with different causation. The clinical diagnosis of cicatricial pemphigoid is made when there is progression of a symblepharon without a history of local radiation, long-term topical medications or severe perennial allergic conjunctivitis.

Conjunctivitis

Conjunctivitis—typically a result of infection, allergy or irritation—is characterized by conjunctival hyperemia, ocular discharge and, depending on the etiology, discomfort and itching. The various etiologies present with differing signs and symptoms, and knowing what to look for will help tailor treatment appropriately:

VIRAL CONJUNCTIVITIS

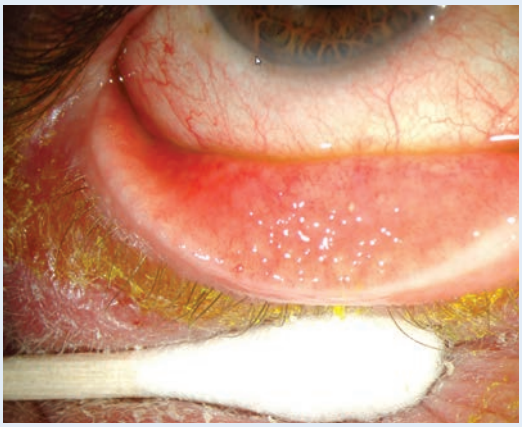
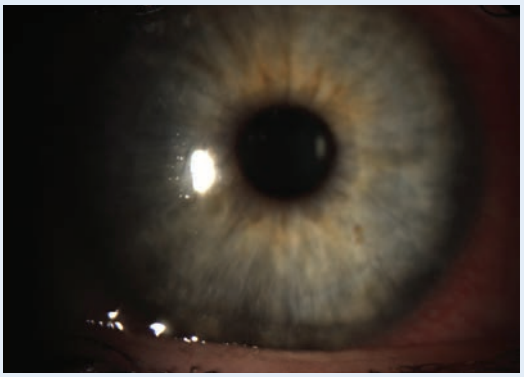


Photo: Christine Sindt, OD



Viral conjunctivitis is often an acute, contagious conjunctival infection usually associated with an infection of the upper respiratory tract or may be related to an adenovirus. Symptoms, which often are limited to one eye at a time, include irritation, photophobia and watery discharge.

Conjunctivitis may accompany the common cold and other systemic viral infections, especially measles, but also chickenpox, rubella and mumps. Localized viral conjunctivitis without systemic manifestations usually results from adenoviruses or enteroviruses.

Epidemic keratoconjunctivitis (EKC), pharyngoconjunctival fever and acute hemorrhagic conjunctivitis—a rare conjunctivitis found in Africa and Asia—result from adenovirus serotypes. The virus is often seen with conjunctival hyperemia, watery discharge and ocular irritation, usually beginning in one eye and spreading rapidly to the other. Although the signs may seem common, they vary from patient to patient. Characteristically, follicles may be present on the palpebral conjunctiva, and a preauricular lymph node is often enlarged and painful.

Adenoviral conjunctivitis presents with photophobia and foreign body sensation due to corneal involvement. Chemosis, pseudomembranes of fibrin and inflammatory cells on the tarsal conjunctiva, focal corneal inflammation, as well as the subepithelial infiltrates (SEIs) are a direct result of the virus. Corneal opacities occasionally result in decreased vision and significant halos and starbursts. However, extreme redness, light sensitivity and discomfort are the driving symptoms. Even after the conjunctivitis has resolved, SEIs may be visible with a slit lamp, and the threat of dormant virus reactivating the inflammation is common.

Adult Inclusion Conjunctivitis

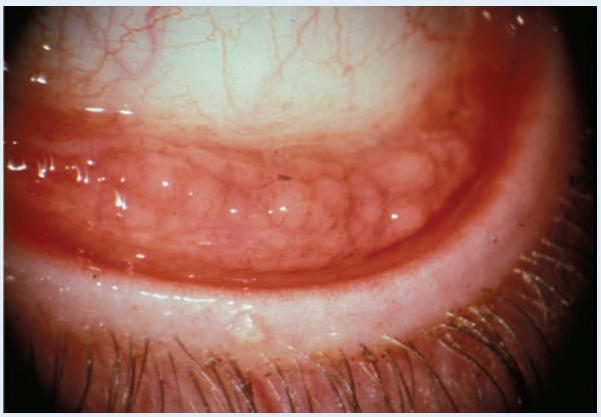


Photo: Paul M. Karpecki, OD, and Diana L. Shechtman, OD

Adult inclusion conjunctivitis is caused by *Chlamydia trachomatis* and has an incubation period of two to 19 days. Most patients have a unilateral mucopurulent discharge, as well as a follicular and hyperemic tarsal conjunctiva response. Preauricular lymph nodes may be swollen on the side of the involved eye. Often, symptoms have been present for weeks or months while nonresponsive to topical antibiotics.

A chronic conjunctivitis, mucopurulent discharge, marked tarsal follicular response and failed treatment with topical antibiotics should alert the clinician to the differential of adult inclusion conjunctivitis.

LOMBART CS-5 CHAIR & STAND

Quality, Style & Value

Package includes:

- The ***Lombart CS-5*** Chair & Stand
- ***Topcon VT-10*** Refractor
- ***Topcon SL-2G*** Slit Lamp

- ***Lombart CVS Essential Visual Acuity System*** with RF Remote Control
- Additional upgrades & configurations available.

$13,995

Or lease for $277/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

BACTERIAL CONJUNCTIVITIS

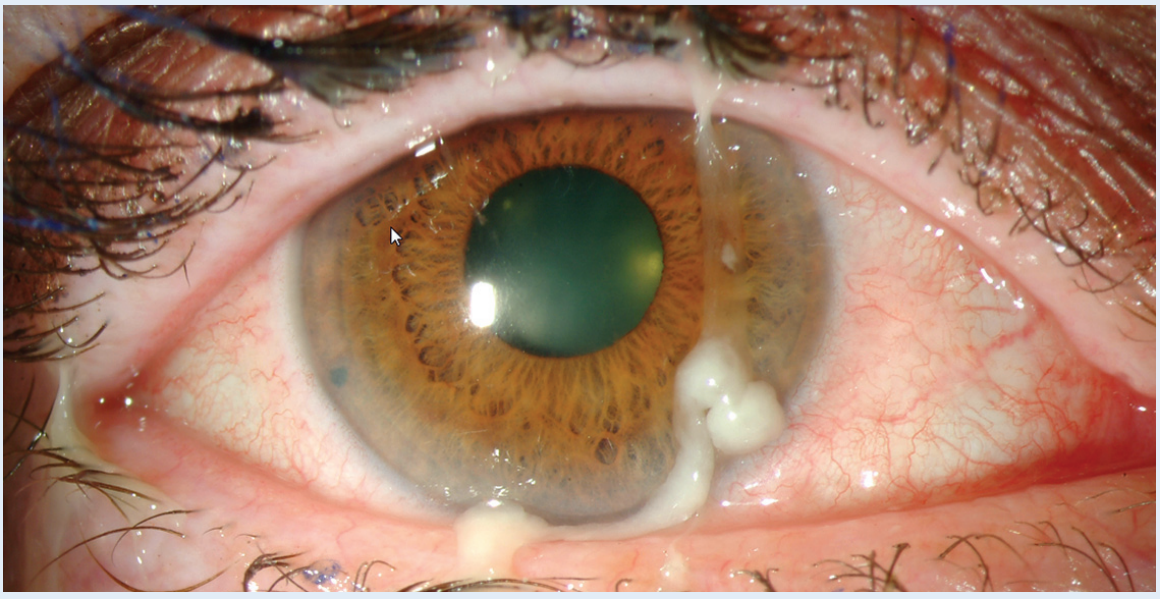


Photo: Christine Sindt, OD

Bacterial conjunctivitis is often a byproduct of the natural flora of the individual and is most commonly caused by *Staphylococcus aureus*, *Streptococcus pneumonia* and *Haemophilus*. Most bacterial conjunctivitis is acute; however, *Chlamydia* and, rarely, *Moraxella* may cause a chronic bacterial conjunctivitis. Chlamydial conjunctivitis includes trachoma and adult or neonatal inclusion conjunctivitis.

Symptoms are typically unilateral, but frequently spread to the opposite eye within a few days. The bulbar and tarsal conjunctivae can become intensely hyperemic with a mucopurulent discharge. The condition often has a papillary reaction on the palpebral conjunctiva.

In cases of adult gonococcal conjunctivitis, symptoms include eyelid edema, chemosis and a prolific purulent discharge.

Symptoms of ophthalmia neonatorum secondary to gonococcal infection are bilateral and include intense papillary conjunctivitis with eyelid edema, chemosis and mucopurulent discharge.

TRACHOMA

***Chlamydia trachomatis*, or trachoma, is endemic in poverty-stricken parts of the world, with most blinding occurring in Africa, and rarely occurring among Native Americans and immigrants in the United States. The disease tends to affect children three to six years of age.[1]**

The clinical manifestations are often divided into active disease and those associated with repeat infections. Active disease is symptomatic for a follicular conjunctivitis, and follicles are dome-shaped and the center is avascular. Large follicles located near the cornea may leave depressions known as Herbert's pits, cicatrial scars of follicles at the limbus, and are considered pathognomonic for trachoma. The active disease will also show signs of papillary hypertrophy and corneal pannus. In contrast, the repeat infections have significant conjunctival scarring, cicatricial entropion with trichiasis and the potential to induce corneal opacification.

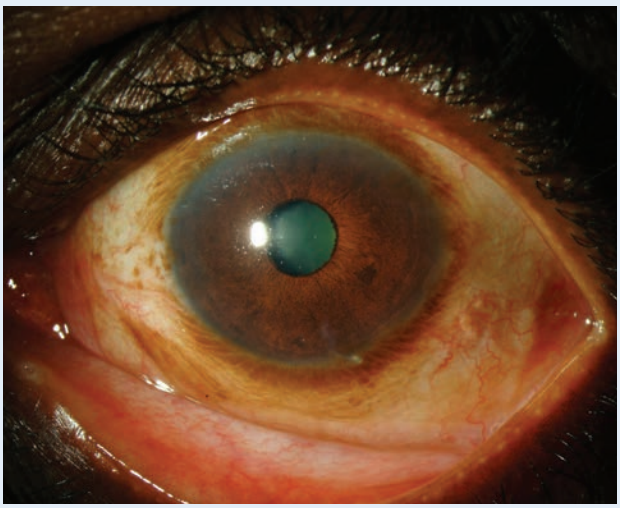
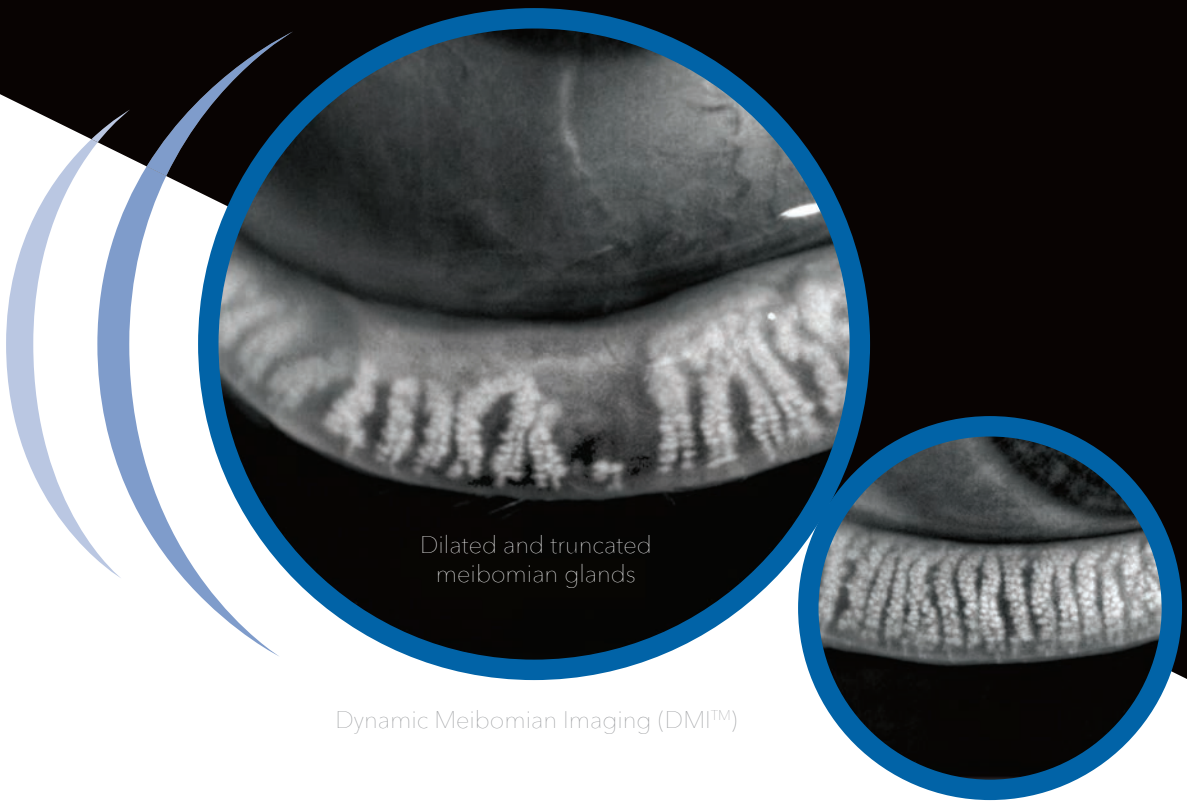


Photo: Christine Sindt, OD

Call TearScience today

New. Fast. Affordable.

Reveal meibomian glands in HD



Dynamic Meibomian Imaging (DMI™)

Rapid HD Meibomian Imaging

Pre-test workup imaging to fit your workflow.

- Images fast, in 10 seconds
- Under normal lighting
- User friendly
- Small footprint
- Pretest and satellite locations

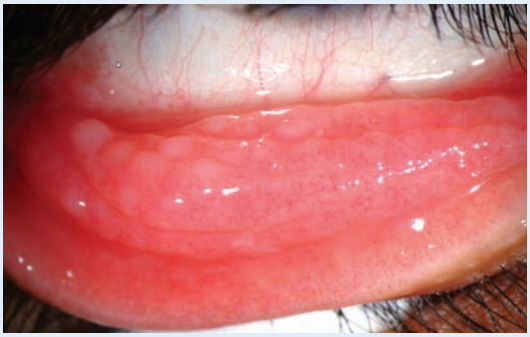
LipiScan™

Treat MGD with LipiFlow® by TearScience

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

ALLERGIC CONJUNCTIVITIS

Photo: Christine Sindt, OD

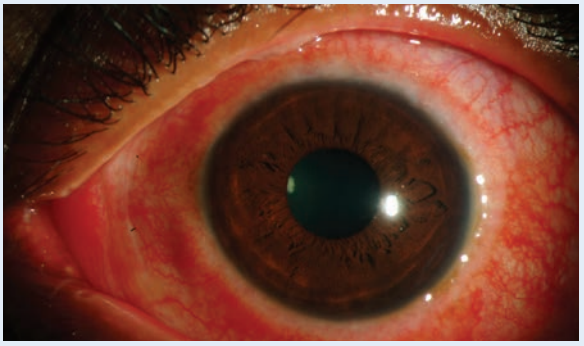


Allergic conjunctivitis is due to a type I hypersensitivity reaction to a specific antigen. Statistics show that more than 50 million Americans suffer from allergies each year.[2] Airborne mold spores and the pollen of trees, grasses and weeds cause seasonal allergic conjunctivitis. Perennial allergic conjunctivitis (atopic conjunctivitis, atopic keratoconjunctivitis) is caused by dust mites, animal dander and other nonseasonal allergens.

The symptoms of allergy and dry eye tend to overlap; however, patients report bilateral, mild to intense ocular itching, conjunctival hyperemia, photosensitivity, eyelid edema and watery or ropy discharge. Concomitant rhinitis is common.

VERNAL KERATOCONJUNCTIVITIS

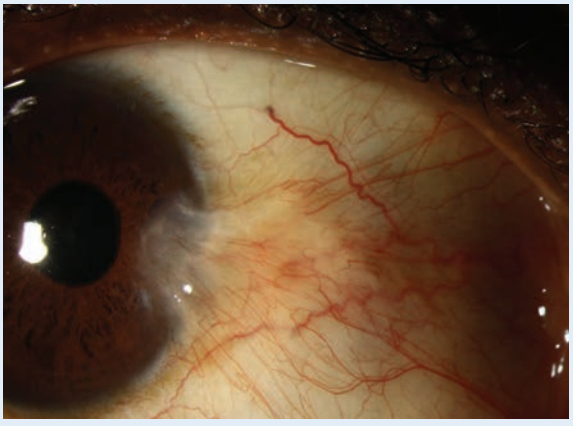
Photo: Christine Sindt, OD



Vernal keratoconjunctivitis is a more severe form of conjunctivitis and is most likely allergic in origin. It is most common among males ages five to 20 who also have eczema, asthma or seasonal allergies. Trantas' dots in the superior limbus represent an aggregation of epithelial cells and eosinophils.

PTERYGIUM

Photo: Christine Sindt, OD

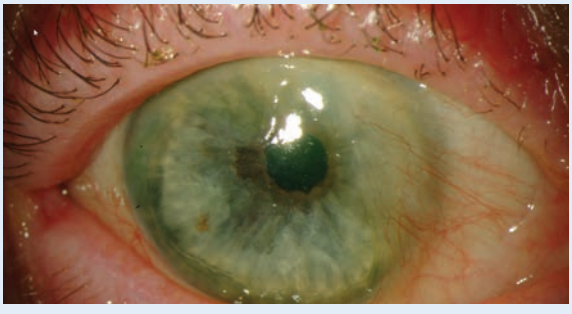


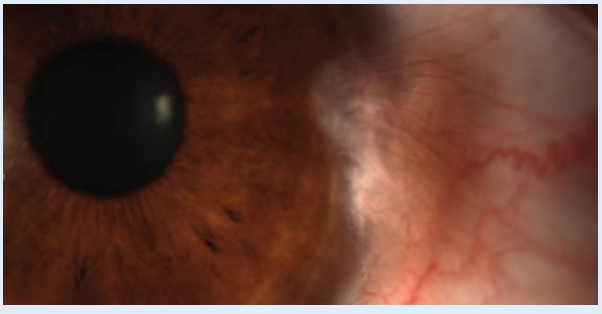
Pterygium is a common conjunctival fibrovascualr lesion originating from the limbus within the palpebral fissure. The nasal limbus is more common for this triangular growth. The growth may spread across the cornea, inducing astigmatism and ultimately affect the visual acuity.

The actual etiology of the pterygium is unknown, but a higher incidence is noted in areas closer to the equator or a higher incidence of UV light exposure.[6-9] Pterygia are an accumulation of degenerated subepithelial tissue, destroying Bowman's layer.

A slit lamp exam will reveal the typical limbal growth pattern. The thin translucent membrane or thickened, elevated mound may be in the presence of a pinguecula. Because the lesions tend to stay in the interpalpebral zone, lesions resembling a pterygia outside this area are highly suspicious.

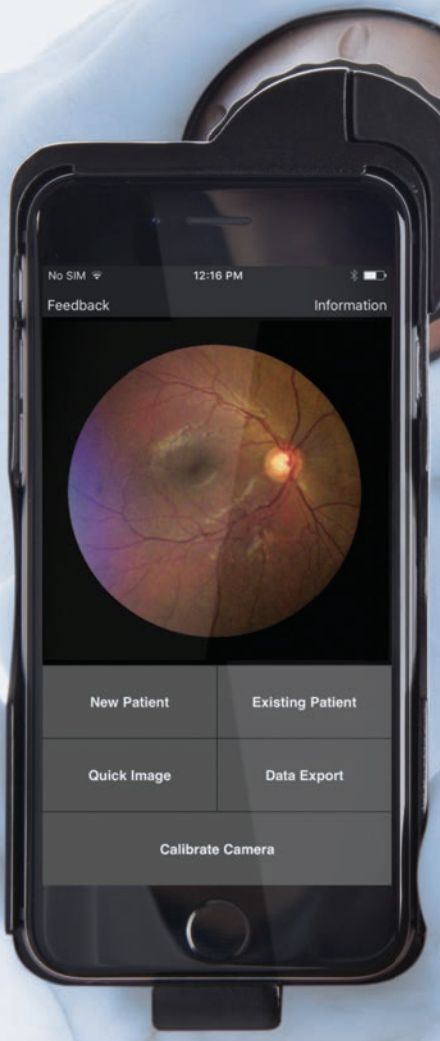
Photo: Christine Sindt, OD





VOLK®
IMAGING

Seeing is **believing.**



VOLK® iNview

iPhone fundus camera

Create a **Doctor-Patient Dialogue** with iNview.

Trusted Volk Optics. iPhone Convenience.

BUY TODAY 10% OFF **volk.com/offers**

Limited time offers. Online customers only.

EPISCLERITIS

Episcleritis is an idiopathic inflammation of the episcleral tissue and occurs in young adults, more commonly among women.[10] It is usually idiopathic and can be associated with connective tissue diseases and, rarely, with serious systemic diseases.[11] Episcleritis is classified as either nodular or simple. Nodular episcleritis has discrete, elevated areas of inflammation; simple episcleritis is present without nodules.

Episcleritis is distinguished from conjunctivitis by hyperemia localized to a limited sector or diffuse areas of the globe, much less lacrimation and no discharge. It is distinguished from scleritis by lack of photophobia and lack of severe pain. The condition is often described as self-limited, and a diagnostic assessment for systemic disorders is not routinely warranted.

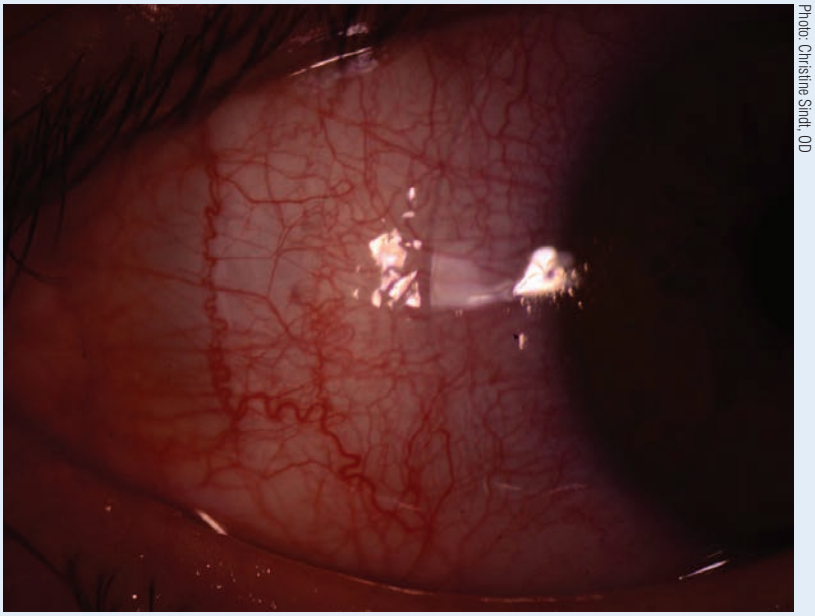


Photo: Christine Sindt, OD

SCLERITIS

Scleritis is a destructive, vision-threatening inflammation involving the deep episclera and sclera. Fourteen percent of patients with scleritis lose significant visual acuity within a year, and 30% lose significant visual acuity within three years.[12]

Scleritis is most common in females in their third to fifth decade, many of whom have connective tissue diseases.[12] About half of the cases of scleritis have no known cause.

Symptoms are moderate to marked pain, hyperemia of the globe, tearing and light sensitivity. Scleritis most commonly involves the anterior segment and occurs in three types: diffuse, nodular and necrotizing.

Pain, often characterized as a deep, boring ache, is often severe enough to interfere with sleep and appetite. Hyperemic patches develop deep beneath the bulbar conjunctiva and with a more violet color compared with those of episcleritis or conjunctivitis. The involved area may be focal (usually one quadrant of the globe) or involve the entire globe and may contain a hyperemic, edematous, raised nodule (nodular scleritis) or an avascular area (necrotizing scleritis).

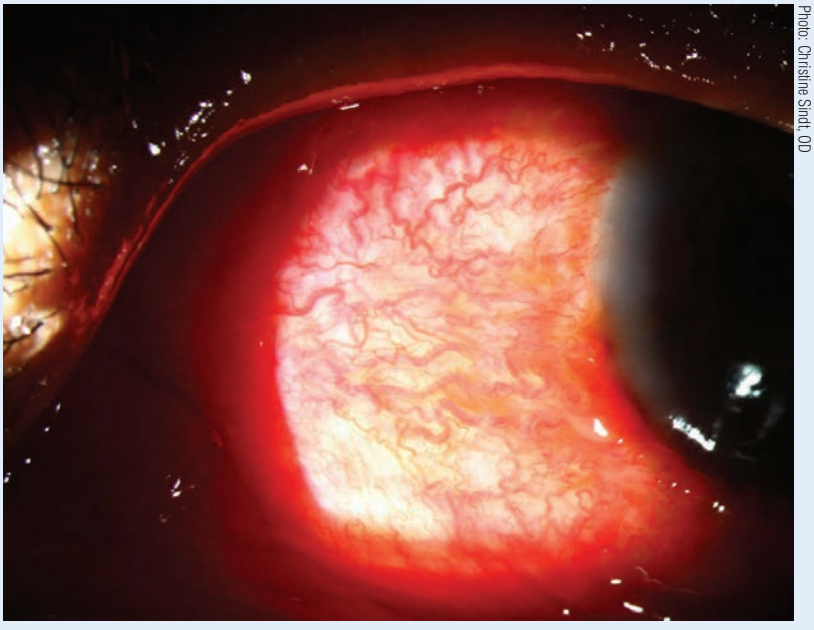


Photo: Christine Sindt, OD

Posterior scleritis is less common and is less likely to cause red eye but more likely to cause blurred or decreased vision.

In severe cases of necrotizing scleritis, perforation of the globe and loss of the eye may result. Necrotizing scleritis in patients with connective tissue disease signals underlying systemic vasculitis. Patients with necrotizing scleritis and underlying systemic vasculitis have a mortality rate mostly due to myocardial infarction.

HAI Ophthalmic

NEW YEAR, NEW DEALS, LET'S WORK SIDE BY SIDE

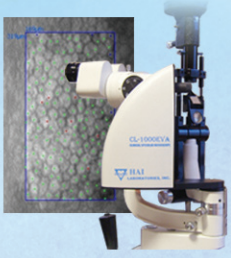
2016 SHOW SPECIAL PRICE

	LIST PRICE	SHOW PRICE
Basic Exam Lane Package	~~$12,998~~	$12,850
SL-5000LED Slit Lamp	~~$5,500~~	$3,580
SL-5000p Plus Slit Lamp	~~$6,300~~	$4,950
SL-5000s Standard Lamp	~~$4,800~~	$2,990
SL-5000bx Basic Slit Lamp	~~$3,800~~	$1,990
SL-5000h Handheld Slit Lamp	~~$3,150~~	$2,380
VC-170 17" LED Vision Chart	~~$2,500~~	$1,590
AB-6100 A/B Ultrasonic Scan	~~$18,500~~	$8,000
SL-5000p digital Slit Lamp	~~$12,000~~	$15,800
CL-1000eva specular w/ Slit Lamp	~~$17,800~~	$15,990

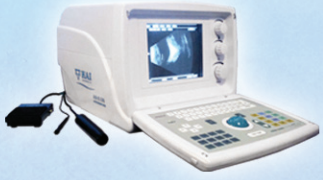
AAOPTO booth #315

Basic Exam Lane Package

*Basic Exam Lane Package including: HAI SL-5000bx, HAI VC-170 LED Vision Chart, S4OPTIK CB-1800 Chair/Stand Combo and SL-Y100 Refractor



HAI CL-1000eva Endothelium Viewing Attachment

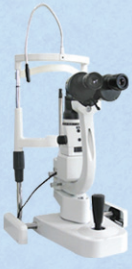


HAI AB-6100 A/B Ultrasonic Scan

VC-170 LED Vision Chart

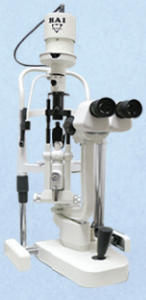
HAI SL-5000 Digital Video Slit Lamp

HAI SL-5000h Hanheld Slit Lamp



HAI SL-5000bx Basic Slit Lamp

HAI SL-5000s Standard Slit Lamp



HAI SL-5000p Plus Slit Lamp

HAI SL-5000e Elevate Slit Lamp

Slit lamps • Specular microscopes • Corneal topographers • Keratometers • Ultrasounds • Vision charts and testers • Autorefractors Lensmeters • Pachymeters • Tonometers • Trial lens sets • Chairs and stands • Digital imaging solutions

HAI Laboratories, Inc. | TOMEY CBD • TOMEYUSA | Inami Ophthalmic Instruments | S4OPTIK

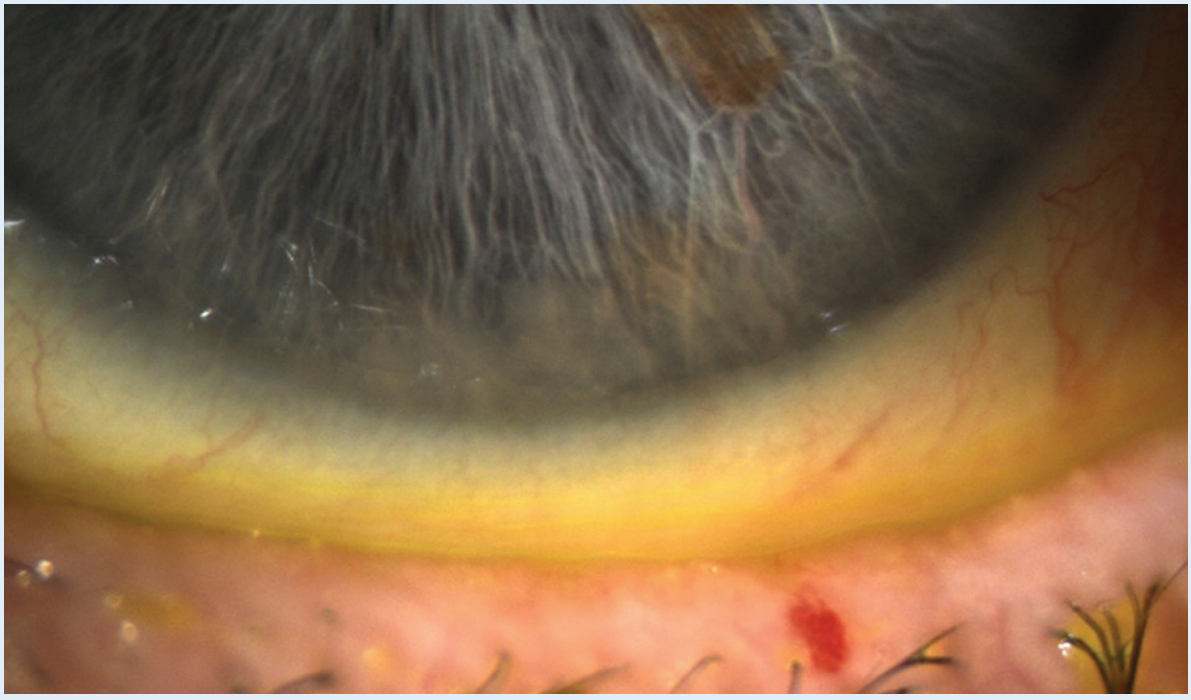
HAI Ophthalmic is a division of HAI Laboratories, Inc., an ISO 13485-certified US manufacturer.

Visit us at www.hailabs.com and www.haiophthalmic.com

Call **(781) 862-9884** or e-mail **sales@hailabs.com** for a free consultation.

HAI 20 YEARS ANNIVERSARY

CONJUNCTIVOCHALASIS



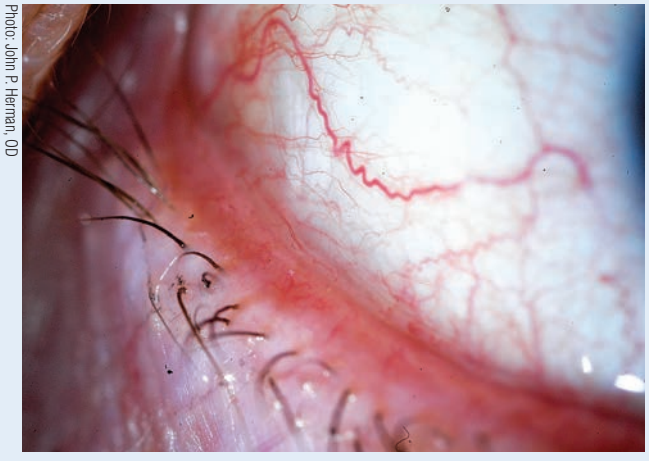


Photo: John P. Herman, OD

Most conjunctivochalasis is thought to be caused by the gradual thinning and stretching of the conjunctiva that accompanies age as well as a loss of adhesion between the conjunctiva and underlying sclera related to the dissolution of Tenon's capsule. The resulting loose, excess conjunctiva may mechanically irritate the eye and disrupt the tear film and its outflow, leading to dry eye and excess tearing. A correlation may also exist between inflammation in the eye and conjunctivochalasis; however, it is unclear if this correlation is causal. Conjunctivochalasis may be associated with previous surgery, blepharitis, meibomian gland dysfunction (MGD) and aqueous tear deficiency.

Symptoms range from dry eye, epiphora and irritation to localized pain, foreign body sensation, subconjunctival hemorrhage and ulceration. Symptoms often worsen due to vigorous blinking.

You will see any number of these issues in your chair, and being able to recognize them quickly will give you a head start on diagnosis and management. ■

Dr. Bloomenstein is director of optometric services at Schwartz Laser Eye Center in Scottsdale, Ariz.

1. International Trachoma Initiative. The world's leading cause of preventable blindness. Available at http://trachoma.org/about-trachoma. Accessed Sept. 13, 2016.
2. American College of Allergy, Asthma, and Immunology. Allergy Facts. Available at http://acaai.org/news/facts-statistics/allergies. Accessed September 13, 2016.
3. Ozer PA, Altiparmak UE, Yalniz Z, et al. Prevalence of Pinguecula and Pterygium in Patients With Thyroid Orbitopathy. Cornea. 2010;29(6):659-63.
4. Yam JC, Kwok AK. Ultraviolet light and ocular diseases. Int Ophthalmol. 2013 May:14(2)187-94.
5. Mimura T, Obata H, Usui T, et al. Pinguecula and diabetes mellitus. Cornea. 2012 Mar;31(3):264-8.
6. Moran DJ, Hollows FC. Pterygium and ultraviolet radiation: A positive correlation. Br J Ophthalmol. 1984;68:343-6.
7. Taylor HR, West S, Munoz B, et al. The long-term effects of visible light on the eye. Arch Ophthalmol. 1992;110:99-104.
8. Ang LP, Chua JL, Tan DT. Current concepts and techniques in pterygium treatment. Curr Opin Ophthalmol. 2007;18:308-13.
9. Singh G. Pterygium in the tropics. Ophthalmology. 1990;97(5):542-3.
10. Homayounfar G, Nardone N, Borkar DS, et al. Incidence of Scleritis and Episcleritis: Results From the Pacific Ocular Inflammation Study. Am J Ophthalmol. 2013 Oct;156(4):752-8.
11. Akpek EK, Uy H, Christen W, et al. Severity of episcleritis and systemic disease association. Ophthalmology. 1999;106(4):729-31.
12. Roque MR. Scleritis. Medscape. Available at http://emedicine.medscape.com/article/1228324-overview#a6. Accessed September 13, 2016.

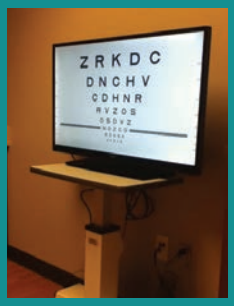
Acuity Pro
VisionScience Software

REDEFINING LOW VISION

"The ETDRS chart is a gold standard for work in vision rehabilitation. Until recently, changing charts was a cumbersome task of sliding the plastic chart sheets in and out of the light box. Using Acuity Pro, combined with a 40 inch LCD monitor, we can change charts with the ***push of a button****. The optotypes are sharp with no detectable pixelation, even at 1 meter test distance. The brightness of the monitor can be adjusted to match the recommended luminance so testing is truly identical to the original light box method.*

After using Acuity Pro, ***I can't imagine practicing without it.****"*

David Lewerenz, OD, FAAO
Clinical Diplomate in Low Vision, American Academy of Optometry
Northeastern State University Oklahoma College of Optometry



EXAM ROOM AT THE NEW OKLAHOMA COLLEGE OF OPTOMETRY LOW VISION CENTER IN BROKEN ARROW, OK

LESLIE L. WALLS
LOW VISION CENTER

LARGE SCREEN LED TESTING
The 40" monitor will allow the entire top 20/200 row to be displayed at 4 meters. Plus all the rows down to 20/40 are visible in one view.

MOVABLE TARGET
By mounting the LED monitor on a table with wheels, testing can be done at 4 meters, 2 meters, and 1 meter

ALL ETDRS CHARTS INSTANTLY
Just press the "I" button to cycle through the three original and the three 2000 Series ETDRS charts in their true optotype sequence

Acuity Pro
VisionScience Software

Visit us at AAO Booth #720

- Acuity Pro Digital Acuity Software Suite Version 9 for Windows
- Software only or all in one systems available
- Includes custom remote, pathology images, Looney Tunes clips, Macro app, and Marco TRS compatible

877-228-4890 info@acuitypro.com acuitypro.com

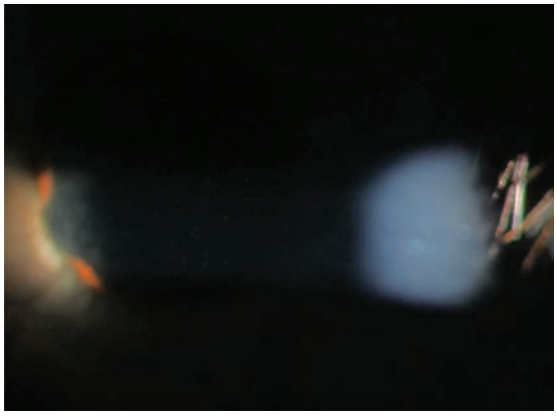
2 CE Credits
(COPE approval pending)

Identifying Systemic Sources of Uveitis

Nearly half of all uveitis cases are caused by systemic disease. Learn the root causes and improve—sometimes save—the lives of your patients. **By Aaron Bronner, OD**

Uveitis is a challenging ocular condition to manage due to its link to a wide variety of systemic inflammatory and infectious processes, as well as its often under-appreciated potential to cause vision loss. Approximately 40% to 50% of all uveitis cases are caused by an underlying systemic condition. We recognize some of the clinical variability among etiologies, but our findings don't always mean what we think. While lab studies are sometimes critical for diagnosis, ordering a huge battery of tests is neither cost effective nor particularly useful.

This article reviews the signposts we can use to whittle our differential diagnosis down to a smaller set of conditions prior to testing. Because anterior uveitis accounts for 70% of cases, it is the primary focus of this review.



Cell in the anterior chamber is the diagnostic finding of anterior uveitis.

Clinical Clues

First, consider factors in the presentation and patient demographics that might allow you to narrow your focus.

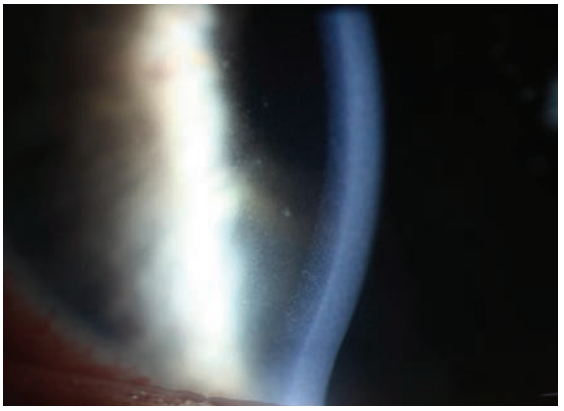
Certain underlying etiologies have very well described propensities for manifesting in certain ways—granulomatous forms of uveitis generating mutton fat keratic precipitates (KPs), for example. However, these associations are not set in stone; HLA-B27, in rare cases, may cause mutton fat KPs, while sarcoidosis-associated uveitis does not present with mutton fat KP or iris nodules—classic signs

Release Date: November 2016
Expiration Date: November 15, 2019
Goal Statement: Uveitis is often associated with serious ocular and systemic conditions requiring immediate treatment. This course provides an overview of the most common etiologies underpinning uveitis in the United States. The various causes of uveitis as well as creating a workable differential diagnosis in the different cases are discussed.
Faculty/Editorial Board: Aaron Bronner, 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.
Disclosure Statement: The author has no relationships to disclose.

of granulomatous disease—in many cases.[2,3] Additionally, recognizing trends in KP distribution can be enormously beneficial. KPs classically present in Arlt's triangle, while diffuse, very localized or linear KP distributions are strongly suggestive of specific etiologies.[4,5]

Although we tend to think of uveitis symptomology in terms of photophobia and a deep achy eye, this is only characteristic of acute anterior uveitis (AAU). Patients with deeper inflammation, such as primary vitritis, choroiditis or chronic anterior disease, do not experience these classic symptoms; rather, they complain of reduced vision or floaters.[5] These groups all have separate etiologies with only minimal spill over. For this reason, asymptomatic uveitis patients should generate a separate differential than those with classic symptoms.

Given the wide array of systemic conditions associated with uveitis, identifying available clues can help you more easily determine the cause. More specifically, looking at the intersection of both the clinical picture of the episode and the patient can give you a good start towards a diagnosis. For example, one study on tubulointerstitial nephritis and uveitis syndrome (TINU), demonstrates that 1.7% of the patients presenting to this uveitis center had TINU, making it a rare source of disease.[6] However, when the authors looked at only uveitis cases that were acute, bilateral and anterior and occurred in patients younger than 20, they found that TINU accounted for 32% of all cases.[6] By simply assessing an obvious patient feature (age) and clinical features (acute vs. chronic and laterality), the researchers increased the likelihood they were dealing with TINU by 19 times, making this rare disease the primary differential in the correct setting.[6]



Fine KP dusting of the endothelium was seen in a case of HLA-B27 AAU. This is frequently encountered in cases of nongranulomatous anterior uveitis.

While not all sources of uveitis are as easily differentiated, demographic factors such as age and race play important roles in determining likely etiologies. And, be aware that the etiologies presented here are common to the United States, which vary in frequency across the globe.

Etiologies

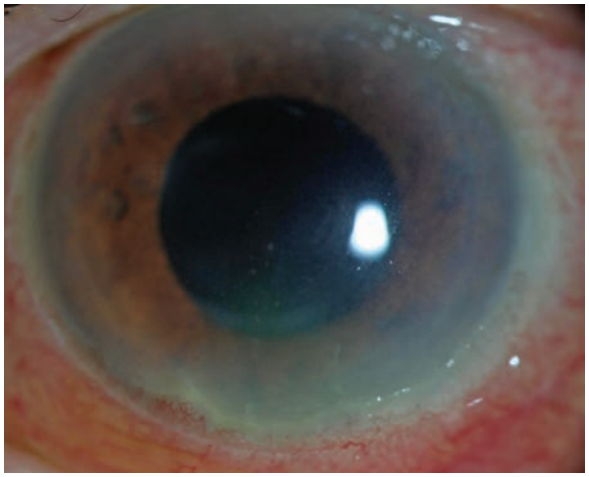
Much like "dry eye" is a catch-all phrase for a heterogeneous assortment of often unrelated conditions, so too is "uveitis" an umbrella term with poor specificity. Below we review particular etiologies with an emphasis on how to recognize their unique attributes.

• ***HLA-B27.*** Specific human leukocyte antigens (HLAs)—cellular markers associated with the immune response—are associated with an increased risk of specific disease processes. HLA-B27 is associated with an increased risk for developing diseases such as ankylosing spondylitis, reactive arthritis, psoriatic arthritis and inflammatory bowel disease. Additionally, its presence may be associated with local forms of inflammation such as isolated aphthous ulcers (canker sores), erythema nodosum or anterior uveitis.

A person who carries HLA-B27 has a 100-fold greater relative risk for AAU over a population-matched non-carrier of the marker.[5] HLA-B27's association with uveitis accounts for approximately 50% of AAU cases in Caucasian populations, superseding idiopathic causes.[5,7,8] This association increases to 70% in patients with recurrent AAU.[10] Patients with HLA-B27 and an associated systemic disease have a 25% lifetime risk of developing an episode of AAU, and those with a history of HLA-B27 and AAU have a 50% risk of developing an associated disease.[1,5,6]

Patient setting. The patient who develops an initial bout of HLA-B27 uveitis will generally be a teen aged to middle aged Caucasian, and one study reports that six out of 138 uveitis patients over the age of 60 developed HLA-B27 uveitis as a primary episode.[14] The incidence of HLA-B27 among Caucasians is approximately 8%, and its rate is even higher among certain indigenous American populations such as Haida Native Americans (50%) and American Inuit populations (25%).[1] In African Americans, the incidence drops to 2%, and if it exists at all in an unmixed African racial profile, it's only at extremely low levels.[9] While most cases of uveitis across all populations are idiopathic, among Caucasians with AAU, HLA-B27 is the most common etiology.[1,5,7]

Clinical picture. On history and exam, a patient with HLA-B27 uveitis will generally look like the classic case of moderate-to-severe unilateral acute, non-granulomatous uveitis. The patient will be symptomatic with photophobia and vague deep discomfort. The exam will show ciliary flush and white cells predominating in the anterior chamber with significant flare as well. Fibrin and hypopyon occur in a high percentage of patients and vitreal spillover is frequently seen, though anterior chamber inflammation is always primary.[1,7,10]



Seen here are early granulomatous KP respecting Arlt's triangle in a case of sarcoidosis-related uveitis. Arlt's triangle distribution is a general finding and not suggestive of any specific etiology.

While cystoid macular edema (CME) and epiretinal membrane (ERM) may develop, especially with recurrences, the prognosis of HLA-B27 uveitis is generally good if treated early, with only idiopathic disease having better outcomes.[5] Each attack lasts for an average of six to eight weeks, and treatment should continue over that time frame. Recurrences may freely alternate between eyes, and their timing varies dramatically, with the average recurrence being every one to two years.[1]

• ***Juvenile idiopathic arthritis (JIA)***. Although the precise mechanism of JIA is not known, patients can generally be subclassified based on joint involvement and serologic testing. The condition exhibits significant heterogeneity, so several subtypes are possible, which include the pauciarticular (fewer than five joints involved within the first six months), polyarticular (five or more joints involved within the first six months) and the systemic form, which is paired with features such as fever, rash and internal organ involvement.

The pauci- and polyarticular forms may be further subtyped as rheumatoid factor positive or negative and anti-nuclear antibody (ANA) positive or negative, with ANA(+) pauciarticular patients having a 30% risk of uveitis.[5,10,11] Conversely, patients with rheumatoid factor-associated disease or systemic JIA have a low risk of developing uveitis.[10]

Finally, a JIA cohort exists that is predominated by older male patients who develop a disease form similar to HLA-B27 uveitis, and may go on to develop associated spondyloarthropathies.[10-12]

Patient setting. Very young patients with asymptomatic uveitis tip the clinician off to JIA. These patients most typically develop arthritis between the ages of three and six; when uveitis develops, it generally follows within the year, though in approximately 5% of cases the uveitis precedes the arthritis.[12] Uveitis in teenage males—a subgroup of JIA-associated patients—tends to be more acute and symptomatic. This group is often associated with HLA-B27 and may develop an associated adult form of spondyloarthropathies.[10]

In any juvenile patient with white blood cells within the eye, particularly those that are asymptomatic, keep the masquerade syndromes (discussed below) such as leukemia on the differential list.

Clinical picture. In JIA uveitis, the inflammation is confined to the anterior segment, most typically bilateral, mild and chronic in nature. In contrast to the significant symptomology seen in HLA-B27-associated disease, JIA patients are asymptomatic in most cases.[12] The eyes are often white, which may belie an anterior chamber reaction that varies from very mild to severe.[5,12] KPs reflect the nongranulomatous nature of the disease and are fine or medium sized. Because the uveitis generally follows the arthritis, a systemic diagnosis of JIA is often pre-existing.

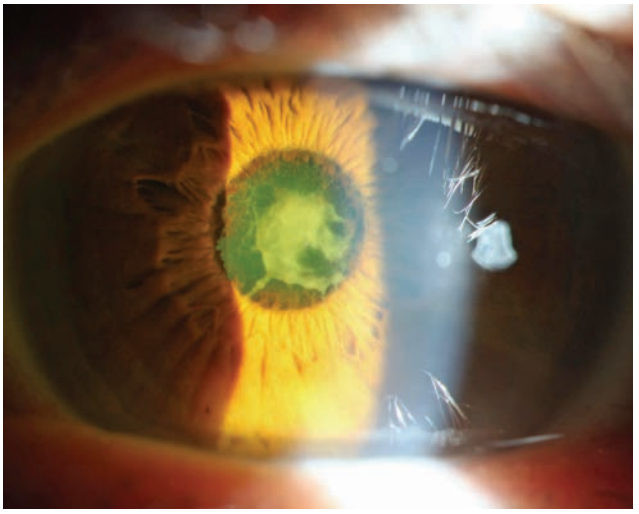
As with all chronic forms of uveitis, potential sources of vision loss in JIA patients include glaucoma, band keratopathy and cataract, with the latter developing in approximately 25% of eyes with JIA.[11] Due to the link to sight-threatening conditions and the fact these patients will often be asymptomatic, ANA(+) JIA patients will require follow up every three to four months and ANA(-) patients every six months, even in the absence of diagnosed uveitis, to rule out its development. Patients with the systemic subtype may be followed yearly to ensure no insidious causes of vision loss are developing.

The older male subtype of JIA is more in line with seronegative spondyloarthropathies in adulthood than younger cohorts, and this uveitis behaves more like that associated with HLA-B27.

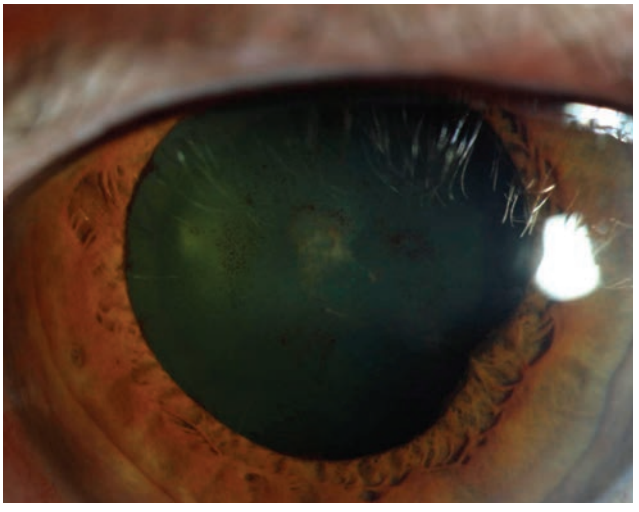
• ***Viral uveitis***. While viral causes such as herpes simplex virus (HSV) 1 and 2 and varicella zoster virus (VZV) can cause pathology in any age group, VZV especially tends to be more prevalent in patients older than 60 years, while newly acquired autoimmune conditions tend to decrease beyond the age of 60. Therefore, we should be increasingly suspect about infectious etiologies of uveitis, particularly those etiologies which affect an aging population, in an elderly patient with new uveitis. Herpes zoster and herpes simplex do account for a higher percentage of uveitis cases as we age, with one retrospective review showing viral sources of uveitis as being the causative etiology in 5% of their cases overall, but approximately 18% of cases in patients older than 60.[14]

Patient setting. In patients over the age of 60 with a first episode of anterior uveitis and no other risk factors (particularly for masquerade syndromes—see below), think "viral uveitis" until proven otherwise. As viral forms of uveitis are often paired with corneal edema, patients with herpetic uveitis will frequently have a chief complaint of blurred vision.

Clinical picture. While almost exclusively unilateral, viral uveitis may be either granulomatous or nongranulomatous; reports are inconsistent as to which form predominates.[4,5] In HSV, acute, recurrent episodes are the norm, while VZV uveitis tends to be more chronic. Most typically, it manifests as a keratouveitis, with keratic precipitates and concomitant corneal edema, both in a circular distribution in the central cornea. However, alternate forms with diffuse and linear KPs are also encountered and are strong indicators of a herpetic etiology. Diffuse KPs are only typical of viral uveitis, toxoplasmosis and Fuchs' heterochomic iridocyclitis.[5] It can be encountered in the absence of corneal involvement, in which case deeper clues to the diagnosis may be found.[15] IOP is elevated in up to 90% of cases.[16] Sectoral iris atrophy occurs most classically in VZV uveitis, but is also found in HSV cases.[4,5,16] Posterior synechiae are less common in viral uveitis than other forms of AAU.[4] Panuveitis as either progressive outer retinal necrosis or acute retinal necrosis is rare, but mandates immediate retinal referral.



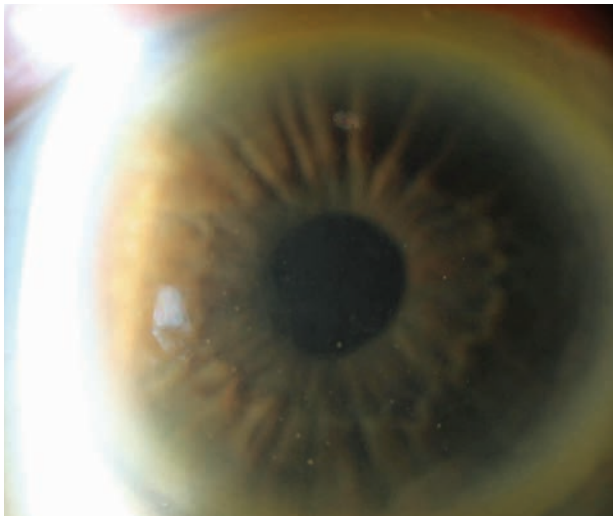
The fibrin pupillary plaque seen here resulted from HLA-B27 uveitis. The fibrin in the anterior chamber is common in this etiology.

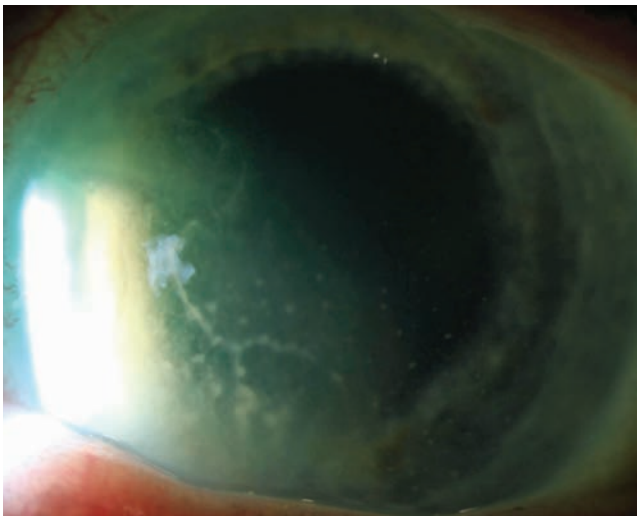


Resolution of the plaque should occur after episode has been controlled, but cataract may result.

Most herpetic uveitis cases can be diagnosed clinically based on the associated findings and patient demographics with good reliability, though PCR of aqueous offers a slightly invasive option to confirm diagnosis.

• ***Sarcoidosis***. This is a multisystem disorder of unknown etiology, though an infectious trigger is widely suspected, which results in the development of non-necrotizing granuloma formation throughout the body.[17] Classically, the lungs are the most often involved organ, and ocular involvement is close behind with 25% to 60% of cases affecting the eyes; however,





At left, this diffuse distribution of KP was seen in a case of Fuchs' heterochromic iridocyclitis (FHI). This KP distribution is diagnostically suggestive of herpetic disease, FHI and anterior spill-over of toxoplasmosis, and is also seen in corneal allograft rejection. At right, linear endotheliitis was seen in a case of viral keratouveitis—the dendritic pattern seen is actually a KP pattern on the endothelium. This is also encountered in corneal allograft rejection.

most patients with sarcoid will be asymptomatic.[17,18,20] Systemic involvement, and the precise degree of ocular involvement, varies among age groups and ethnic profiles.[3]

While uveitis is only one of the many ocular manifestations of sarcoid, it is also the most common. Ocular involvement may occur in the absence of systemic involvement (which is nonspecific), and pulmonary findings may wax and wane throughout the course of the disease, making it a difficult disease to diagnose.[20] Because of this, some epidemiologic studies and classification systems will list both confirmed and suspected sarcoidosis as causes of uveitis, and at least one expert suspects undiagnosed sarcoid accounts for a significant percentage of idiopathic uveitis.[14,18,20] In a large retrospective review on chronic uveitis etiologies, sarcoid was the second most common after idiopathic at 14%.[19] This figure only takes into account sarcoid presenting as the more common chronic form, but sarcoid also appears less frequently as a source of acute disease.

Patient setting. Sarcoidosis can affect any population, though women are affected more frequently than men. In the United States, African Americans develop sarcoid 10 to 17 times more than Caucasians. Although sarcoid has become the chief source of ocular inflammation in Japan, North American patients of Asian ancestry have a lower risk than those living in Japan.[3,5,18,21]

Age of incidence varies depending on race. Caucasian patients are more likely to have sarcoid uveitis develop after the age of 50, while African Americans tend to develop it roughly a decade earlier.[3,21]

In juvenile-onset sarcoidosis, the disease presents with more arthritis symptoms and is on the differential for any childhood arthritis or uveitis.[13]

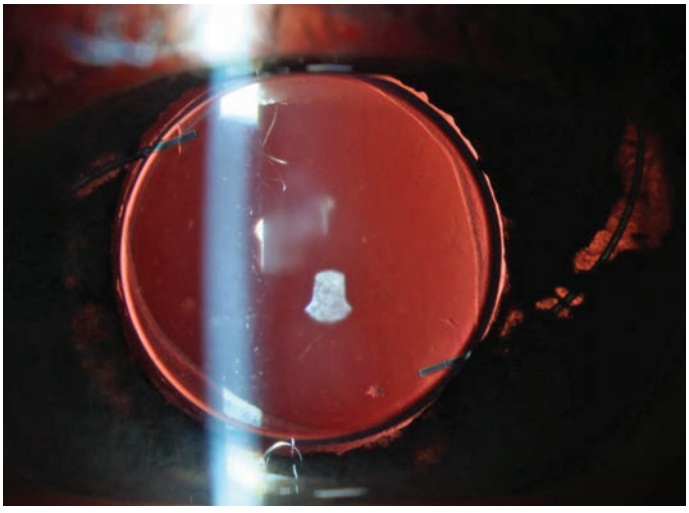
Clinical picture. Sarcoid is often an asymptomatic disease, and when symptoms occur they are most often nonspecific such as a dry cough or fatigue.[20] In 80% of cases, sarcoid uveitis presents bilaterally.[18,19]

While sarcoid is the chief source of granulomatous uveitis and may present with mutton fat KPs, iris nodules or both, it frequently presents without either of these key markers.[3,21] Iris nodules, when present, may occur at the pupillary margin (known as Koeppe's nodules) or within the stroma of the iris (busacca nodules). Berlin's nodules, a less frequently recognized form of anterior segment nodules associated with granulomatous disease, occur at the trabecular meshwork (TM). These can be difficult to detect as they are often white and very small.[18] Tent-shaped peripheral anterior synechiae (PAS) in an eye with concomitant uveitis is also suggestive of sarcoid.[18]

Despite sarcoid being a granulomatous disease, granulomatous findings are not always present. For instance, in one study, 66% of patients with sarcoid-linked uveitis had nongranulomatous anterior uveitis on presentation.[21]

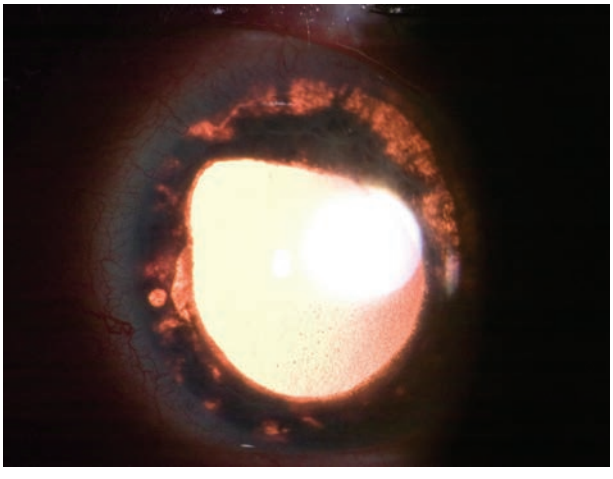
Note that anterior uveitis is only one of sarcoid's uveal

manifestations. It also causes intermediate uveitis, and is the chief systemic differential for patients presenting with primary vitritis, which manifests as white cells in the anterior vitreous and accumulation of acellular inflammatory debris (i.e., "snowballs") in the inferior vitreoretinal interface—known as snowbanking—the latter of which is quite suggestive of sarcoid.[18] Retinal vasculitis, primarily exudative periphlebitis (inflammation of retinal venules), is a classic sign of sarcoid, a finding often described as candlewax drippings.[18] In severe cases, exudative periphlebitis may culminate in a retinal vein occlusion.[5,18] Choroiditis may manifest as solitary choroidal nodules or smaller, multifocal-type choroiditis. These small, yellow choroidal lesions may resolve completely or result in small, punched-out scars, similar to those seen in histoplasmosis.[5,18,20]



This uveitis masquerader was caused by obvious mechanical chafing of IOL haptics. These may go on to cause micro-hyphema and elevated IOP at which point it would be classified as uveitis glaucoma hyphema (or UGH) syndrome.

Sarcoid uveitis may present with any or all of these findings. However, racial differences influence which are found. African Americans may have a significantly higher rate of anterior segment granulomatous disease when compared with Caucasians, with research showing 72% of African Americans with sarcoidosis and uveitis exhibit anterior granulomatous findings compared with 25% of Caucasians.[3] Since a significant percentage of cases occur in the absence of its classical presentation, awareness of the alternate, deeper forms of sarcoid uveitis are very important for clinical decision making.



This sectoral iris atrophy was seen in a patient with zoster iritis.

Prognosis for sarcoid uveitis follows, to a degree, the depth of uveal involvement. Patients who develop CME or multifocal choroiditis are at the greatest risk for vision loss, and patients with anterior only disease have a relatively good visual prognosis.[5,22,23]

• ***Syphilis***. This is no longer a primary source of uveitis, with most estimates of luetic disease accounting for 0.5% to 3% of all uveitis cases.[25,26] Still, its varied presentation and link to severe ocular and systemic morbidity keeps it on the differential list in any adult patient.[20,23,26-28] Ocular manifestations of syphilis are extremely varied, and syphilis serologies should be part of essentially any systemic inflammatory workup. Though ocular involvement is uncommon, uveitis is the most frequent ocular manifestation and may be found in 2.5% to 5% of all cases of tertiary disease.[23,25] In its acquired form, luetic uveitis may occur in the disease's primary stage but is more frequently encountered during secondary or latent stages and may also be associated with neurosyphilis.[5,23,27,28] Coinfection with HIV may cause altered pathologic patterns of the disease, including its uveal manifestations.[25,27]

Patient setting. Of the approximately 36 million people worldwide living with syphilis, 90% are in developing countries.[28] In the United States, men account for roughly 85% of all cases of acquired syphilis.[24,28] A spike in infections during the 2000s was driven primarily by male-

Using Lab Tests to Hone the Differential

Labs are a critical component of uveitis management. The following is a brief primer on a handful of labs commonly used to manage anterior uveitis.

• *HLA-B27.* A positive result (which is 99% sensitive and specific for the antigen) indicates the presence of one of the HLA-B alleles. The test itself is not diagnostic for any specific disease; however, a positive result in the presence of unilateral acute anterior uveitis is diagnostic. Patients with a positive HLA-B27 test without a previously diagnosed systemic disease should be counseled appropriately on their systemic risks.[42]

• *Antinuclear antibodies.* Most often, ODs think of ANA as a test for lupus. However, these are associated with a variety of systemic diseases and even exist in the normal population. While ANA is associated with lupus, isolated uveitis is uncommon in systemic lupus erythematosus (SLE). ANA testing for SLE (a rare cause of uveitis) has a low positive predictive value and should not be tested for on a routine basis.[43,44] ANA's value in uveitis management is part of a broad systemic workup or when subclassifying JIA.

• *Rheumatoid factor (Rf).* Like ANA, Rf's utility in uveitis screening should be, in most cases, reserved for classification of JIA. Rheumatoid arthritis seldom produces isolated uveitis.

• *Angiotensin converting enzyme (ACE).* This is a byproduct of macrophages interacting with granulomas.[20] Elevated serum ACE, while associated with sarcoidosis, is not specific for the disease. It's also not particularly sensitive for sarcoid, with only 40% to 60% of sarcoid patients having elevated levels.[3,18] The best testing for sarcoidosis combines serum ACE testing and a chest X-ray or CT. ACE inhibitors limit the reliability ACE testing.[18]

• *Chest X-rays.* These are most frequently used to test for sarcoid and tuberculosis. When used for sarcoidosis, bilateral hilar lymphadenopathy (BHL) is the significant finding. As with ACE testing, chest X-ray is not highly sensitive for the sarcoid with only 50% to 80% being positive.[3] Further, it is possible for the chest X-rays to alternately be positive or negative throughout the course of the disease.[20] When combined with serum testing, however, sensitivity of the two tests together rises to 89%.[3] BHL may also occur in lymphoma.[18]

Though chest X-rays will be more frequently ordered for sarcoid, lobe cavitation is the key finding when used for TB, though less than half of TB patients with ocular symptoms will have pulmonary involvement.[30]

• *Syphilis testing.* The syphilis organism *Treponema pallidum* can't be cultured and is difficult to see under light microscopy, so testing relies on the body's reaction to elements associated with the bacterium.

Testing can be broken down into to two general types:

Non-treponemal tests: Venereal Disease Research Laboratory (VDRL) and rapid plasma reagin (RPR).

Treponemal tests: Fluorescent treponemal antibody absorption (FTA-ABS) and microhemagglutination for *Treponema pallidum* (MHA-TP).

The non-treponemal tests are less expensive and less specific; they are often negative during primary and latent syphilis, though will also be negative after successful treatment. Treponemal tests, which look for specific antibodies to the organism, are more sensitive and will be positive during primary syphilis and latent syphilis. They will also remain positive after treatment.[24]

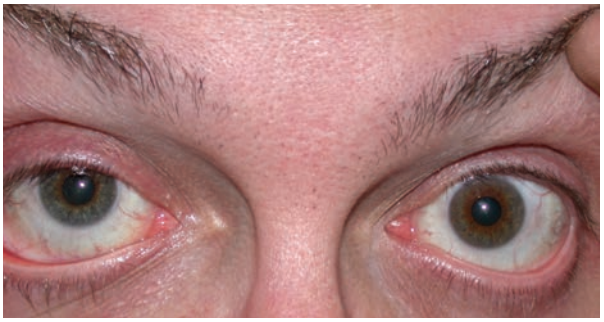
• *Tuberculosis skin prick.* This uses a type IV hypersensitivity reaction to *Mycobacterium tuberculosis* (TB) antigens. Infected patients develop a large area of induration (swollen, hardened skin). As expected, patients who are immunosuppressed will have a less vigorous zone of induration and may generate false negatives. Patients from countries where the Bacillus Calmette-Guerin (BCG) immunization is given for TB may also have false negatives.[34] Since TB is an uncommon source of uveitis, routine testing is unnecessary unless history and exam are supportive.

male intercourse.[24,28] This group accounted for 64% of cases of patients with primary or secondary syphilis in 2004.[23] The disease has also disproportionately affected African Americans, with a rate of 9.0/100,000 (compared with the national rate of 2.7/100,000).[24]

Clinical picture. Any and all layers of the uvea are possible targets for the bacterium and no findings are pathognomonic: anterior, intermediate, posterior and panuveitis presentations are all possible.[23,30] Granulomatous and nongranulomatous cases are split nearly equally, as are bilateral and unilateral cases.[30] Findings somewhat suggestive of a luetic source in a uveitis eye are optic disc edema and retinal vasculitis. But, these findings are only present in posterior segment manifestations and are not helpful in the 20% to 55% of cases that present with simple anterior uveitis.[23,26,29] Iris abnormalities will occasionally accompany anterior disease and may manifest as vascular abnormalities (roseola and papules) or nodules (nodosa) and can help direct clinical suspicion.[5] The best clue may be the response to therapy—as a manifestation of a systemic infection, syphilitic uveitis will not respond definitively to corticosteroids.[27]

Syphilis testing is imperative in cases of unresponsive uveitis. If identified and treated appropriately, good outcomes are likely, even in those with deep disease or coexisting HIV infection.[28] Treatment is somewhat controversial, as some authors suggest ocular syphilis be treated as secondary or tertiary disease with three weekly intramuscular injections of penicillin G while others advocate for treatment as neurosyphilis with IV penicillin G, particularly when the deep globe is involved.

• ***Tuberculosis (TB)***. Worldwide, nearly two billion people are infected with *Mycobacterium tuberculosis*, of which 10% develop frank tuberculosis.[30] The vast majority of these cases occur in developing countries where poverty is a primary risk factor.[31] TB remains the primary source of infectious morbidity and mortality across the globe.[31,32]



Heterochromia as encountered in FHI is seen here. Heterochromia may not be present or may manifest as more irregular iris depigmentation.

As the organism is an obligate aerobe, it most commonly affects parts of the body with high oxygen concentrations, such as the lungs.[31] Not surprisingly, uveitis is the most common ocular manifestation of TB, as the highly vascularized uveal structures (particularly the choroid) are among the most highly oxygenated tissues of the body.[31] Despite this, in the United States, TB accounts for only 0.5% of cases at uveitis clinics.[32]

Patient setting. History-based risk factors are essential and are advocated as part of clinical criteria for diagnosing ocular tuberculosis. Immune suppression is the primary risk factor for TB in the United States and developed countries, though emigration from endemic areas or previous exposure to those with active pulmonary TB are equally important.[31,32] While pulmonary symptoms may help steer clinical suspicion, general symptoms such as fever, night sweats or fatigue may be of some help in the absence of pulmonary findings.[31]

Clinical picture. Posterior and panuveitis are the most common forms of TB uveitis.[32] Serpiginous choroiditis, retinal vasculitis with or without choroiditis and broad-based posterior synechiae are suggestive of the disease, being quite specific but only weakly sensitive for TB.[30] Their presence strongly merits evaluation for TB in an at-risk population, but their absence does not rule out the condition. In its anterior form, TB most commonly causes chronic recurrent uveitis characterized.

While TB is a granulomatous process, mutton fat KPs or iris nodules do not occur much more than in a non-TB uveitis population.[30] Though TB is not on the primary differential for most episodes of uveitis, its presence should be considered prior to initiating any treatments that rely upon a mechanism of systemic immune suppression.

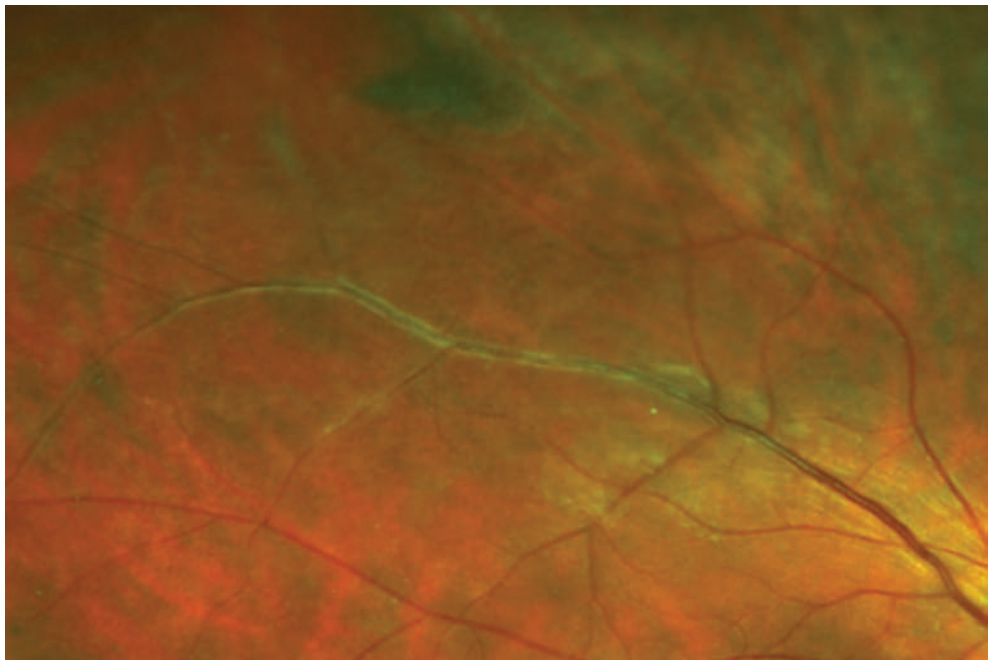
• ***Fuchs' heterochromic iridocyclitis (FHI)***. Proposed mechanisms of FHI include infectious sources (toxoplasmosis, HSV or VZV), disorders of adrenergic innervation, genetic and autoimmunity to anterior segment self-antigens.[35] More recently, researchers have made a very compelling case for a rubella etiology, by showing that patients with FHI demonstrate a universal immunoglobin activity against rubella.[5,36,37] A high percentage of Fuchs' patients have rubella RNA recovered from their anterior chamber, and FHI incidence is lower in countries with rubella vaccination programs compared to countries without programs.[5,32,37]

Patient setting. While FHI may occur in any demographic, it's most commonly seen in a young and middle-aged adult population.[33,34] It affects Caucasian and African American populations, though heterochromia is less prominent in deeply pigmented populations, possibly leading to underreporting in the African American population.[38]

Clinical picture. The vast majority of cases are unilateral and chronic with mild anterior chamber reaction and a white eye. Despite being a frequently missed diagnosis, FHI is not rare; one study reports it as the second most common etiology of chronic uveitis behind sarcoidosis, accounting for 12% of chronic cases at one uveitis clinic over 35 years.[19] Patients are nearly always asymptomatic, and researchers suggest that, even prior to evaluation, the picture of a young, healthy adult with asymptomatic uveitis and good vision should alert the clinician to FHI as a possibility.[33]

FHI is associated with iris heterochromia. In a patient with light eyes, the darker iris is involved; in dark eyes, the lighter iris is involved. While heterochromia is an important—nearly pathognomonic—finding, as many as 30% of FHI cases lack this feature.[34]

Diffuse, white stellate KPs may be found in up to 100% of FHI patients.[5,34] Less common are iris nodules, which occur more frequently in African American FHI populations; posterior synechiae are almost always absent.[34,38] Vitritis is also frequently encountered, though it does not produce snowbanking as seen in pars planitis or other forms of intermediate uveitis, and retinal



Here, venous sheathing was caused by early periphlebitis as seen in sarcoid uveitis. The vasculitis in these cases may become occlusive and lead to BRVO.

scars are often seen.[33,34]

Later in the course of the disease, cataracts almost always manifest as mid-lens opacities or posterior subcapsular changes.[33,34] Additionally, glaucoma occurs in 9% to 50% of eyes with FHI over time.[34] Due to its asymptomatic nature, delays in diagnosis are common, with patients often aware of heterochromia years prior to visual concerns that are linked to cataract or glaucoma.

Given its proposed infectious etiology, FHI does not respond, or responds incompletely, to corticosteroids.[33,34] Treatment should focus instead on preventing vision loss from lens and vitreous opacities, as well as the timely diagnosis and management of glaucoma.[5,33,34]

Masquerade Syndromes

These conditions produce inflammation or pseudoinflammation in the eye, but stem from noninflammatory conditions such as lymphoma, exogenous sources or surgical complications. Among each relevant population, consider the possible masquerade syndromes when formulating a differential diagnosis.

• ***Intraocular lymphoma.*** Vitritis is a universal finding in intraocular lymphoma. The form most associated with ocular findings is non-Hodgkin's CNS lymphoma, which tends to present between ages 50 and 70.[5,15,38] Retinal findings are variable and only 20% of cases may have anterior uveitis.[15] Any case with chronic vitritis in a patient older than 50 warrants suspicion for lymphoma. Workup includes head MRI, lumbar puncture and diagnostic vitrectomy.[5,38]

• ***Intraocular leukemia.*** Although this masquerades as uveitis less commonly than lymphoma, acute leukemias account for up to 5% of pediatric uveitis cases.[39] Because the presence of uveitis is a poor prognostic sign—it usually indicates spread to the CNS or bone marrow relapse—it is enormously important to consider intraocular leukemia as the etiology in this population.[39] Hypopyon, pseudohypopyon and increased IOP as a result of leukemic infiltration of the TM are all possible findings. When IOP is sufficiently high, the patient may be symptomatic; otherwise, the patient will be asymptomatic.

• ***Retained lens material after cataract surgery.*** In any patient with a recent history of cataract surgery preceding chronic intraocular inflammation, retained lens fragments must be suspected as a primary differential. While uncomplicated cataract surgery and IOL placement almost universally produces white cells in the chamber, this is not an immune reaction in the typical sense. Rather, the mechanical trauma of the surgery disrupts the blood/aqueous barrier, resulting in white cells leaching into the anterior chamber. Under most circumstances, the blood/aqueous barrier is reestablished within two to four weeks, even without corticosteroids, and the chamber becomes quiet. Any lingering inflammation should produce a strong suspicion that lens material, which is an antigenic source of uveitis, may be causative. Thoroughly evaluate the angle with gonioscopy and the posterior chamber with dynamic viewing through a dilated pupil. If a fragment is still not visible, refer for ultrasonic biomicroscopy. Aspiration of any long-lasting lens fragment is necessary.

• ***Uveitis glaucoma hyphema (UGH) syndrome.*** This is most

common with anterior chamber IOLs, caused when chafing of the IOL on the iris results in a mechanical iritis, hyphema and inflammatory glaucoma. Lenses placed in the ciliary sulcus have a lower risk, though chafing and UGH may still occur. IOLs placed fully in the capsular bag present no risk. Cycloplegics (to reduce chafing associated with pupillary movement), corticosteroids and IOP-reducing medications are palliative, but IOL repositioning is the long-term treatment when feasible.

• ***Chronic infectious endophthalmitis.*** Chronic cells in the anterior chamber and vitreous following cataract surgery, where lens fragment and IOL positioning issues have been ruled out, means chronic infectious endophthalmitis should be considered. This masquerade condition produces a milder intraocular inflammation than the acute form. Assessment of the lens capsule for suspicious white plaques can be helpful to steer suspicion. Diagnosis is confirmed through anterior chamber and or vitreous tap and culture.

• ***Brimonidine side effect.*** An infrequently encountered though important late side effect of brimonidine use is acute granulomatous uveitis. This occurs approximately one year after initiating therapy and responds to discontinuation. The uveitis will recur if the medication is reintroduced and occurs only in medicated eyes.[41]

• ***Retained intraocular foreign body.*** Iron-based foreign bodies are antigenic and will produce a cellular response. In some cases, patients will be unaware of the injury but will come in with complaints of uveitis weeks or months later. Consider this: if uveitis is not responding permanently to therapy, question patients for a history of welding, grinding or frequent use of a striking instrument. Gonioscopy can help identify material in the angle. The uveitis in these cases will be unilateral, chronic and generally mild. It will respond to steroids temporarily but will recur upon cessation.

Despite the enormous variety in uveitic etiologies, often relatively narrow differentials can be made when clinicians carefully consider the clinical appearance and the patient setting of the disease. By becoming familiar with and assessing these features, the appropriate diagnosis and management of uveitis, though still difficult, can become less opaque and simpler to achieve. ■

Dr. Bronner is an attending optometrist at Pacific Cataract and Laser Institute in Boise, WA.

1. Chang JH, McCluskey PJ, Wakefield D. Acute anteriror uveitis and HLA-B27. Surv Ophthalmol. 2005;50:364-88.
2. Miserocchi E, Modorati G, Di Matteo F, et al. Visual outcome in ocular sarcoidosis: retrospective evaluation of risk factors. Eur J Ophthalmol. 2011;21(6):802-10.
3. Birnbaum AD, Oh FS, Chakrabarti A, et al. Clinical features and diagnostic evaluation of biopsy proven sarcoidosis. Arch Ophthalmol. 2011;129(4):409-13.
4. Jap A, Chee SP. Viral anterior uveitis. Curr Opin Ophthalmol. 2011;22(6):483-8.
5. Van Gelder RN, Prasad AG. Review of Uveitis. Thorofare, NJ; Slack Inc; 2008.
6. Mackensen F, Smith J, Rosenbaum J. Enhanced recognition, treatment and prognosis of tubulointerstitial nephritis and uveitis syndrome. Ophthalmology. 2007;114(5):995-9.
7. Brewerton DA, Hart FD, Nicholls A, et al. Ankylosing spondylitis and HLA-27. Lancet. 1973;1:904-7.
8. Ramsay A, Lightman S. Hypopyon uveitis. Surv Ophthalmol. 2001;46(1):1-18.
9. Khan MA. Race related differences in HLA association with ankylosing spondylitis and Reiters disease in American blacks and whites. J Nat Med Assoc. 1978;70(1):41-2.
10. Smet MD, Ongkosuuwito JV. HLA-B27 related uveitis. In: Krachmer JH, Mannis MJ, Holland EJ, eds. Cornea. 2nd ed. St. Louis: Mosby; 2004:1375-81.
11. Kotaniemi K, Savolainen A, Karma A, Aho K. Recent advances in uveitis of juvenile idiopathic arthritis. Sur Ophthalmol. 2003;48:489-502.
12. Tugal-Tutkun I, Quartier P, Bodaghi B. Disease of the year: juvenile idiopathic arthritis-associated uveitits–classificaiton and diagnostic approach. Oc Immunol Inflamm. 2014;24(1):56-63.
13. Hu-Torres S, Foster CS. Disease of the year: juvenile idiopathic arthritis – differential diagnosis. Oc Immunol Inflamm. 2014;22(1):42-55.
14. Chatzistefanou K, Markomichelakis NN, Christen W, et al. Characteristics of uveitis presenting for the first time in the elderly. Ophthalmology. 1998;105(2):347-52.
15. Zamiri S, Boyd S, Lightman S. Uveitis in the elderly – is it easy to identify the masquerade? Br J Ophthalmol. 1997;81(10):827-31.
16. Santos C. Herpes simplex uveitis. Bol Asoc Med P R. 2004;96(2):71-4.
17. Kawagoe T, Mizuki Nobuhisa. Sarcoidosis. Curr Opin Ophthalmol. 2011;22(6):502-7.
18. Herbort CP, Rao NA, Mochizuki M, et al. International criteria for the diagnosis of ocular sarcoidosis: results of the first international workshop on ocular sarcoidosis. Oc Immunol Inflamm. 2009;17:160-9.
19. Birnbaum A, Little DM, Tessler HH, Goldstein DA. Etiologies of chronic anterior uveitis at a tertiary referral center over 35 years. Oc Immunol Inflamm. 2011;19:19-25.
20. Rosenbaum JT. Round 20: Why a Rheumatologist needs to Understand Uveitis. Johns Hopkins Arthritis Center. Available at www.hopkinsarthritis.org/physician-corner/rheumatology-rounds/round-20-why-a-rheumatologist-needs-to-understand-uveitis/. Accessed August 18, 2016.
21. Evans M, Sharma O, LaBree L, et al. Differences in clinical findings between caucasians and african americans with biopsy proven sarcoidosis. Ophthalmology. 2007;114(2):325-33.
22. Lobo A, Barton K, Minassian D, et al. Visual loss in sarcoid related uveitis. Clin Exp Ophthalmol. 2003;31(4):310-6.
23. Barile G, Flynn T. Syphilis exposure in patients with uveitis. Ophthalmology. 1997;104(10):1605-9.
24. Gaudio PA. Update on ocular syphilis. Curr Opin Ophthalmol. 2006;17(6):562-6.
25. Chao JR, Khurana RN, Fawzi AA, et al. Syphilis: reemergence of an old adversary. Ophthalmology. 2006;113(11):2074-9.
26. Yap SC, Tan YL, Chio MT, Teoh SC. Syphilitic uveitis in a singaporean population. Oc Immunol Inflamm. 2014;22(1):9-14.
27. Emmons WW, Church LW. Syphilitic uveitis. West J Med. 1994;161(2):168-71.
28. Cunningham E, Eandi CM, Pichi F. Syphilitic uveitis. Oc Immunol Inflamm. 2014;22(1):2-3.
29. Amaratunge BC, Camuglia JE, Hall AJ. Syphilitic uveitis: a review of clinical manifestations and treatment outcomes in human immunodeficiency virus-positive and negative patients. Clin Exp Ophthalmol. 2010;38(1):68-74.
30. Gupta A, Bansal R, Gupta V, et al. Ocular signs predictive of tubercular uveitis. Am J Ophthalmol. 2010 Apr;149(4):562-70.
31. Shakarchi FI. Ocular tuberculosis: current perspectives. Clin Ophthalmol. 2015;9:2223-7.
32. Tabbara KF. Tuberculosis. Curr Opin Ophthalmol. 2007;18(6):493-501.
33. Mohamed Q, Zamir E. Update on Fuchs' uveitis syndrome. Curr Opin Ophthalmol. 2005;16(6):356-63.
34. Velilla S, Dios E, Herreras JM, Calonge M. Fuchs' heterochromic iridocyclitis: A review of 26 cases. Oc Immol Inflamm. 2001;9(3):169-75.
35. La Hey E, de Jong PTVM, Kijlstra A. Fuchs; heterochromic cyclitis:review of the literature on pathogenic mechanisms. Br J Ophthalmol. 1994;78:307-12.
36. de Groot-Mijnes JD, de Visser L, Rothova A, et al. Rubella virus is associated with fuchs heterochromic iridocyclitis. Am J Ophthalmol. 2006;141(1):212-4.
37. Birnbaum AD, Tessler HH, Schultz KL, et al. Epidemiologic relationship between fuchs heterchromic iridocyclitis and the United States rubella vaccination program. Am J Ophthalmol. 2007;144(3):424-8.
38. Tabbot BR, Tessler HH, Williams D. Fuchs' heterochromic iridocyclitis in Blacks. Arch Ophthalmol. 1988;106:1688-90.
39. Rothva A, Ooijman F, Kerkhoff F, et al. Uveitis masquerade syndromes. Ophthalmology. 2001;108(2):386-99.
40. Sharma T, Grewal J, Gupta S, Murray PI. Ophthalmic manifestations of acute leukaemias: the ophthalmologist's role. Eye (Lond). 2004;18:663-72.
41. Byles DB, Frith P, Salmon JF. Anterior uveitis as a side effect of topical brimondine. Am J Ophthalmol. 2000;130:287-91.
42. Wakefield, Chang JH, Amjadi S, et al. What is new HLA-B27 acute anterior uveitis? Ocul Immunol Inflamm. 2011 Apr;19(2):139-44.
43. Rosenbaum JT, Wernick R. The utilitiy of routine screening of patients with uveitis for systemic lupus erythematosus or tuberculosis. Arch Ophthalmol. 1990;108:1291-3.
44. Gallager K, Viswanathan A, Okhravi N. Association of systemic lupus erythematosus with uveitis. JAMA Ophthalmol. 2015;133:1190-3

OSC QUIZ

You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form *(page 103)*, and return it with the $35 fee to: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. 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.reviewofoptometry.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. Which of these subtypes of uveitis classically cause photophobia?
a. Chronic anterior uveitis.
b. Acute anterior uveitis.
c. Acute posterior uveitis.
d. Any form of uveitis will cause photophobia classically.

2. Which is true regarding granulomatous and non-granulomatous findings with uveitis?
a. Mutton fat KP will always be present in cases of uveitis caused by sarcoidosis.
b. Iris nodules will always be present in cases of uveitis caused by sarcoidosis.
c. Mutton fat KP are never present in cases of uveitis caused by HLA-B27.
d. Sometimes cases of classically granulomatous disease will present in the absence of granulomatous indicators and vice-versa.

3. Which of the following is NOT true regarding HLA-B27?
a. HLA-B27 causes up to 50% of acute anterior uveitis cases in a Caucasian population.
b. HLA-B27 causes up to 70% of recurrent acute anterior uveitis cases.
c. HLA-B27-linked diseases (such as ankylosing spondylitis) are the actual etiology of uveitis in this setting, rather than being related to HLA-B27 antigen specifically.
d. HLA-B27 is nearly non-existent in a racially unmixed African black population.

4. Which is NOT true?
a. If you are HLA-B27 (+), you have a 50% lifetime risk of developing acute anterior uveitis.
b. If patients are HLA-B27 (+), they have 100 times the relative risk of developing acute anterior uveitis as someone without the antigen.
c. If patients are HLA-B27 (+) and have an associated systemic disease, they have approximately a 25% lifetime risk of developing acute anterior uveitis.
d. In a Western population, HLA-B27-related uveitis is a more frequently encountered cause of acute anterior uveitis than idiopathic etiology.

5. Which is true of the clinical picture of HLA-B27 linked uveitis?
a. The uveitis is chronic and mild.
b. The visual prognosis with HLA-B27 uveitis is poor.
c. The disease typically is bilateral.
d. Fibrin occurs frequently in this disease.

6. Which is a correct characterization of juvenile idiopathic arthritis? It:
a. Is a single homogenous group of childhood arthritides.
b. Develops most frequently between the ages of 10 to 12.
c. May be linked to Rheumatoid factor (Rf) or antinuclear antibodies (ANAs).
d. Frequently leads to uveitis in the Rf positive subgroup.

7. Identify which is NOT true. A patient with uveitis caused by JIA:
a. Will typically be young (between the ages of three and eight).
b. Will most frequently have bilateral disease.
c. Will most frequently be in the pauciarticular ANA (+) subgroup.
d. Will develop uveitis prior to the arthritis in most cases.

8. The clinical picture of JIA will show which of the following features?
a. Chronic and few symptoms.
b. Unilateral disease.
c. An obviously inflamed red eye.
d. Mutton fat KP.

9. Which is NOT true about care of JIA based uveitis patients?
a. Even without uveitis, ANA (+) JIA patients require frequent follow-up.
b. Vision loss occurs frequently as a result of CME and exudative retinal detachment.
c. Patients will often not seek care due to lack of symptoms.
d. Cataracts develop in approximately 25% of those impacted by the disease.

10. Viral uveitis:
a. Occurs most commonly in a young population.
b. Often manifests as a keratouveitis.
c. Generally, is bilateral.
d. Most frequently appears a fibrinous acute uveitis.

11. Which is NOT part of the clinical picture of herpetic uveitis?
a. Diffuse KP.
b. Linear KP.
c. Fine or granulomatous KP.
d. All of these are possible with herpetic disease.

12. What is a finding that is frequently seen with herpetic uveitis?
a. Elevated IOP.
b. Iris nodules.
c. Panuveitis.
d. Broad based posterior synechiae.

13. Which is true about a patient with systemic sarcoidosis?
a. These patients will often be asymptomatic.
b. There is a greater likelihood that they will be Caucasians.
c. An African American with sarcoid will usually be older at the time of disease development compared to a white patient.
d. The skin and lungs are more frequently involved than the eyes.

14. Which is NOT true of sarcoid uveitis?
a. African Americans are more likely to have anterior granulomatous disease than Caucasians.
b. It can be a challenging etiology to definitively diagnose.
c. Tent-shaped PAS are suggestive of the disease.
d. Retinal vasculitis is primarily of the arterioles.

15. Which is not a type/location of

OSC QUIZ

granulomatous iris nodule?
a. Busacca/iris stroma.
b. Berlin/chamber angle.
c. Dalen Fuchs'/pigmented epithelium.
d. Koeppe/pupillary margin.

16. Of this group, which findings are most suggestive of a syphilitic source of uveitis?
a. Bilateral granulomatous uveitis.
b. Unilateral multifocal choroiditis.
c. Retinal vasculitis with optic disc edema.
d. Chronic serpiginous choroiditis.

17. When would ordering tests of tuberculosis be most indicated in a uveitis patient?
a. A patient with nongranulomatous acute anterior uveitis.
b. A patient with serpiginous choroiditis.
c. A patient with intermediate uveitis.
d. A patient with optic disc edema and retinal vasculitis.

18. Which is NOT true of Fuchs' heterochromic iridocyclitis (FHI)?
a. The condition is not responsive to steroid.
b. Heterochromia is felt to be present in all cases.
c. Diffuse stellate KP are the most frequently seen finding.
d. Glaucoma and cataract are frequent sources of vision loss.

19. Which is a speculated etiology of FHI?
a. Rubella.
b. Scarlet fever.
c. Rheumatoid arthritis.
d. Systemic lupus erythematous.

20. When working up a patient with chronic uveitis, the clinician should consider which of the following?
a. Onset of uveitis relative to cataract surgery.
b. History of cancer.
c. History of vocational or avocational hazard for intraocular foreign body.
d. All of these would be appropriate considerations in this case.

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

Examination Answer Sheet

Valid for credit through November 1, 2019

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.

Identifying Systemic Sources of Uveitis

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, 440 9th Avenue, 14th Floor, New York, NY 10001.

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.

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 113659 RO-OSC-1116

The Challenge of Vector-borne Disease

Many epidemiologists believe the emergence and spread of Zika and other arboviruses was inevitable. Optometrists must now care for those affected.

By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

With ebola, and now Zika, making headlines around the globe, it's clear vector-borne infection has become a significant public health threat. Travel and changing land use have increased the risk of new pathogens emerging, as well as existing agents previously confined to small, remote areas re-emerging and crossing borders.

Infection, Transmission and Disease

In infectious disease, the absence or presence of the etiological agent is the main determining factor in the epidemiology of the condition. While disease cannot occur in the absence of the agent, disease may not always result from the presence of the agent. The distinction between infection and disease is important:

- Infection is the invasion of a living organism (the host) by another living organism (the agent).[1]
- Disease is a derangement in the function of the whole body of the host, or any of its parts.[1]

A vector is an invertebrate animal that actively transmits an infectious agent between infected and susceptible vertebrates.[1] To prevent and control vector-borne disease in humans, we must first understand the agent and its vectors, modes of transmission between vectors and humans and among humans, and the natural history of the resulting disease.

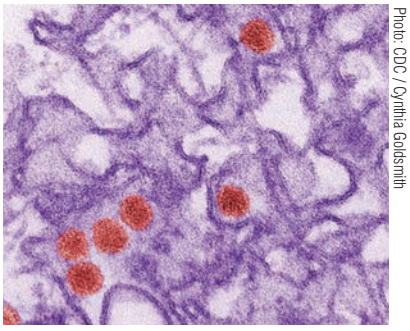


Photo: CDC / Cynthia Goldsmith

Fig. 1. This is a transmission electron micrograph of ZIKV. Virus particles (in red) are 40nm in diameter, with an outer envelope and an inner dense core.

Vectors can transmit infectious agents in two ways. For mechanical transmission, the vector serves as a vehicle whereby the infectious agent is conveyed from one host to another without undergoing a stage of development or multiplication.[1] In biological transmission, the agent undergoes some stage of development or multiplication in the vector.[1] In the latter, the vector serves either as an intermediate or definitive host.

The Zika Challenge

Zika virus (ZIKV) is a mosquito-borne *Flavivirus* isolated from the Zika forest of Uganda in 1947.[2] *Flaviviridae* is a family of positive, single-stranded, enveloped RNA viruses found in arthropods such as ticks and mosquitoes, and can infect humans (*Figure 1*). Members of this family belong to a single genus, *Flavivirus*. Mosquito species within the *Aedes* genus (primarily *Ae. aegypti*) carry the ZIKV. *Aedes* mosquitoes usually bite during the day with peaks during early morning and late afternoon/evening.[2,3] Sexual transmission of ZIKV has been reported.[2,3] A pregnant mother can transmit the virus to her fetus, and other modes of transmission such as blood transfusion may be possible.[2-4] ZIKV does not appear to spread from casual contact.

Until now, ZIKV had remained an obscure agent confined to a narrow equatorial belt running across Africa and into Asia. In 2007, the first large outbreak was reported from the Island of Yap (Federated States of Micronesia).[2,3] The Pan American Health Organization issued an alert in May 2015 regarding the first confirmed ZIKV infection in Brazil.

In February 2016, the World Health Organization declared ZIKV a Public Health Emergency of International Concern because of clusters of microcephaly and other neurological disorders in some areas.[2-4] ZIKV has now circled the globe, arriving not only in the Americas—including the United States—but also in Cape Verde in West Africa, near its presumed ancestral home (*Figure 2*). There have been 43 locally acquired mosquito-borne cases reported in

All-new!

Vc
VisionChek™
Digital Exam Suite

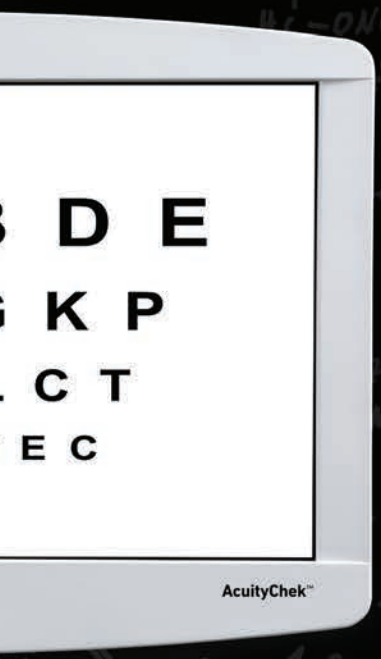
Sc
SightChek™
Digital Phoroptor®

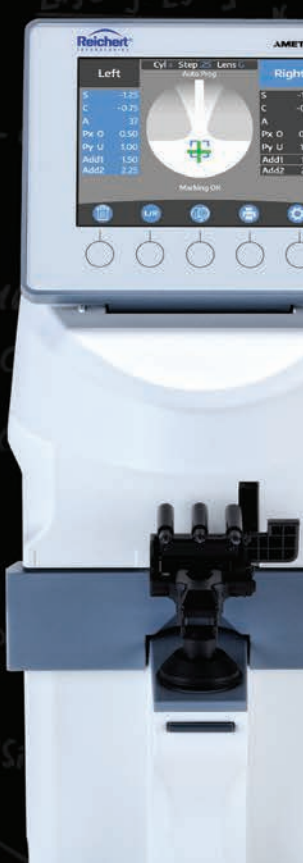
Oc
OptoChek™ *Plus*
Auto Refractor +
Keratometer

Ac
AcuityChek™
Digital Acuity Chart

Pro
Lc
LensChek™ *Pro*
Digital Lensometer®
PD + UV

Plus
Lc
LensChek™ *Plus*
Digital Lensometer®





Elements of Refraction.

VisionChek™ Digital Exam Suite

Reichert® combines technology, simplicity,
and value at the core of your exam.

AAO Anaheim #**604** · reichert.com/visionchek

Reichert®
TECHNOLOGIES

AMETEK®

the continental United States, all of which occurred in Florida (*Figure 3*).

Symptoms and Signs

More than 80% of ZIKV infections are asymptomatic. In those that do develop symptoms, the incubation period is typically a few days.[5,6] The systemic symptoms of ZIKV infection are usually mild and last for several days to a week after being bitten by an infected mosquito. Symptomatic patients typically have a mild course of disease characterized by: fever, maculopapular skin rash, joint pain, red eye or a combination of all of these. Severe disease and fatalities due to ZIKV are rare.[5,6]

Guillain-Barré syndrome has been reported in patients with suspected ZIKV infection.[6] There may also be a link between ZIKV and acute disseminated encephalomyelitis, an autoimmune disorder that affects the brain and spinal cord similar to multiple sclerosis.[7] ZIKV infection during pregnancy can cause microcephaly, which stunts proper formation of the head and brain, causing severe and sometimes fatal brain damage, miscarriage or stillbirth.[4,5] Pregnant women in areas where ZIKV have been reported are advised to take steps to prevent mosquito bites.[6]

Ophthalmic Complications

Investigators in Brazil have reported macular and optic nerve abnormalities in a study of 29 infants with microcephaly associated with ZIKV congenital infection.[4,5] In another report, ophthalmic findings included gross macular pigment mottling, chorioretinal atrophy, optic nerve hypoplasia, increased cup-to-disc ratio, iris coloboma and lens subluxation.[8] Additional findings of blood vessel changes, hemorrhagic retinopathy and torpedo maculopathy in infected infants have been described. It is not known if these findings are a direct result of the ZIKV infection or a consequence of microcephaly. Infected adults have presented with clinical signs of uveitis and a nonpurulent conjunctivitis (*Table 1*).[9,10]

Photo: CDC

Fig. 2. All countries and territories with active ZIKV transmission.

Diagnosis and Management

Individuals should see their health care provider if they have any of the symptoms described and have visited an area with reported ZIKV.

Detecting ZIKV RNA is the gold standard of confirmation of infection. ZIKV can be isolated in cell culture from urine, semen, saliva and breast milk. More recently, investigators detected and isolated ZIKV from conjunctival fluid.[11] Also order blood tests to look for similar viruses such as dengue and chikungunya.[6,10]

A person diagnosed with ZIKV infection should try to prevent mosquito bites for the first week of the illness. The virus usually requires non-specific treatment. Patients should be advised to get plenty of bed rest, drink clear fluids and treat pain and fever accordingly.[10] There is no specific antiviral treatment for ZIKV at this time.

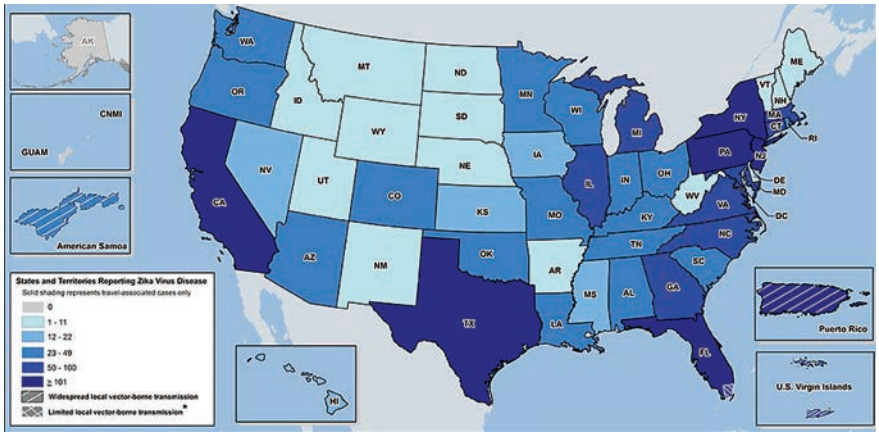


Photo: CDC

Fig. 3. Laboratory-confirmed Zika virus disease cases reported to ArboNET by state or territory as of September 14, 2016.

Our Role Beyond Clinical Care

The CDC encourages all providers to report suspected cases of ZIKV infection to their state health department to help reduce the risk of local transmission. As part of an examination of all patients with possible congenital ZIKV infection, the CDC recommends an eye examination be performed, including retinal evaluation, either in the hospital or within one month after birth.[6,10]

Pregnant patients during any trimester should postpone travel to an area with ongoing ZIKV transmission.[6,10] Testing for the virus can be offered to any asymptomatic pregnant patients who have traveled to areas with ongoing ZIKV transmission, pregnant patients who exhibit two or more symptoms consistent with ZIKV disease and pregnant patients who reside in areas with ongoing ZIKV transmission. The CDC Laboratory and several state health departments are performing ZIKV virus testing.

Table 1. Ophthalmic Signs of Zika Viral Infection

ZIKV in adults

- Uveitis
- Conjunctivitis (nonpurulent)

ZIKV in infants with microcephaly

- Macular pigment mottling
- Loss of foveal reflex
- Chorioretinal atrophy and scarring
- Hemorrhagic retinopathy
- Torpedo maculopathy
- Optic nerve hypoplasia
- Iris coloboma
- Lens subluxation

Currently there is no vaccine. The arbovirus pandemics suggest the one-bug-one-drug approach is inadequate.[12] Broad-spectrum pharmacotherapies effective against whole classes of viruses are needed. ■

1. National Institutes of Health, Biological Sciences Curriculum Study. NIH Curriculum Supplement Series. 2007. Understanding Emerging and Re-emerging Infectious Diseases. Available at www.ncbi.nlm.nih.gov/books/NBK20370/.
2. Hennessey M, Fischer M, Staples JE. Zika virus spreads to new areas—region of the Americas, May 2015–January 2016. Am J Transplantation. 2016;16(3):1031-4.
3. Kindhauser MK, Allena T, Franka V, et al. Zika: the origin and spread of a mosquito-borne virus. Bull World Health Organ. 2016:171082. [Epub].
4. de Miranda HA, Costa MC, Frazão MAM, et al. Expanded spectrum of congenital ocular findings in microcephaly with presumed Zika infection. Ophthalmology. 2016;123(8):1788-94.
5. Meissner HC. Fragile but potentially deadly: Mosquitoes transmit illnesses to millions every year. AAP News. June 2016.
6. Petersen EE, Staples JE, Meaney-Delman D, et al. Interim guidelines for pregnant women during a Zika virus outbreak—United States, 2016. MMWR Morb Mortal Wkly Rep. 2016;65:30-3.
7. Mécharles S, Herrmann C, Poullain P, et al. Acute myelitis due to Zika virus infection. The Lancet. 2016 Apr 2;387(10026):1481.
8. de Oliveira Dias JR, Rosenfeld PJ. Zika Virus and the Eye. Retina Specialist. 2016 June:14-15.
9. Furtado JM, Espósito DL, Klein TM, et al. Uveitis associated with zika virus infection. N Engl J Med. 2016;375(4):394-6.
10. The Centers for Disease Control and Prevention. Key Messages—Zika Virus Disease. September 14, 2016. Available at www.cdc.gov/zika/pdfs/zika-key-messages.pdf.
11. Sun J, Wu D, Zhong H, et al. Presence of Zika virus in conjunctival fluid. JAMA Ophthalmology. September 15, 2016. [Epub].
12. Fauci AS, Morens DM. Zika virus in the Americas—yet another arbovirus threat. N Engl J Med. 2016;374(7):601-4.

Up to 16-20 CE Credits*

NEW TECHNOLOGIES & TREATMENTS IN EYE CARE 2017

REVIEW OF OPTOMETRY
EDUCATIONAL MEETINGS OF CLINICAL EXCELLENCE

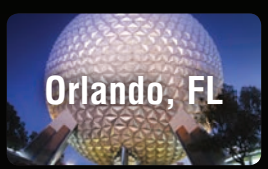
2017 MEETINGS

February 17-21, 2017
Winter Ophthalmic Conference
(Formerly SkiVision)
Program Chairs:
Murray Fingeret, OD & Leo P. Semes, OD

March 24-26, 2017
Program Chair:
Paul Karpecki, OD

Hands-on Workshops†

April 20-23, 2017
Joint Meeting: NT&T/OCCRS
Program Chair:
Paul Karpecki, OD



June 8-11, 2017
Program Chair:
Paul Karpecki, OD

November 3-5, 2017
Program Chair:
Paul Karpecki, OD

CHECK OUR WEBSITE FOR THE LATEST INFORMATION

Online: www.reviewofoptometry.com/events
E-mail: reviewmeetings@jobson.com
Call: 866-658-1772

OCCRS OPTOMETRIC CORNEA, CATARACT AND REFRACTIVE SOCIETY

14th Annual Education Symposium
Joint Meeting with NT&T In Eye Care
San Diego, CA, April 20-23, 2017

†Workshops not available for "Aspen, CO" meeting. See Review website for any meeting schedule changes or updates. Stock Images: ©iStock.com/JobsonHealthcare

Administered by
Review of Optometry®

cope
*Approval pending

Orange and Green at UM

Can this patient's medical history help explain his fading vision?

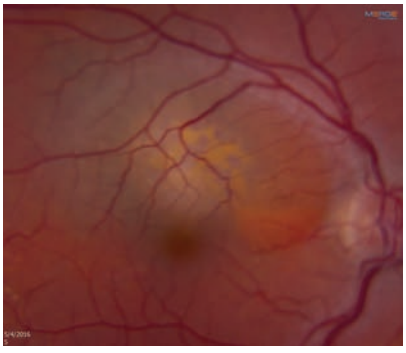
By Elliott Brafman, OD, and Mark T. Dunbar, OD

A 71-year-old Hispanic male presented for an evaluation of a cataract in his left eye. He had undergone cataract surgery in the right eye three years earlier with excellent visual results. However, over the past year he said he has noticed more difficulties seeing out of both eyes. He denies any flashes, floaters, double vision or ocular pain. His past medical history was significant for a recent diagnosis of prostate cancer. The prostate was removed three months prior without radiation. His most recent prostate specific antigen (PSA) test was 0ng/mL one month prior.

Evaluation

Upon examination, his uncorrected vision was 20/25 OD and 20/25 OS, which was correctable to 20/20 in both eyes. Confrontation visual fields were full to careful counting in both eyes. Pupils were equally round and reactive to light; no afferent pupillary defect was found. The intraocular pressure (IOP) was 14mm Hg in both eyes.

A slit lamp examination revealed mild meibomian gland dysfunction and posterior blepharitis in both eyes and a well centered PCIOL without PCO in the right eye. The left eye had a mild nuclear sclerotic cataract. Dilated fundus exam of the left eye was normal. The right eye revealed a 1.5DD elevated lesion (*Figure 1*). An OCT (*Figure 2*) and ultrasound imaging, including B-scan (*Figure 3*) and standardized A-scan (*Figure 4*), were also obtained.



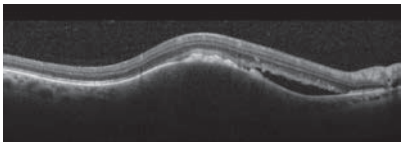


Fig. 1. At left, the patient's left eye. Note the lesion superior to the macula.
Fig. 2. Above, OCT slightly superior of the macula.

Upon further questioning, he was not certain about his ocular history other than having cataracts. Given the history and suspicious appearance of the lesion, we referred him promptly to the ocular oncologist.

Take the Quiz

1. How would you describe characterize the A-scan findings?
a. Essentially normal.
b. Highly reflective.
c. High and low reflectivity.
d. Low to medium reflectivity.

2. What high-risk characteristic does the lesion in the fundus photo display?
a. Orange pigment.
b. Subretinal fluid.
c. Proximity to the fovea.
d. a and b only.

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

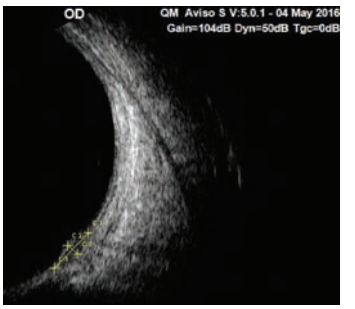
4. Given the location of the lesion, how will this patient likely be managed?
a. Observation.
b. Enucleation.
c. Plaque therapy.
d. External beam radiation.

5. Which statement is accurate with regards to his diagnosis/prognosis, respectively?
a. Choroidal nevus; observation.
b. Small choroidal melanoma; plaque radiotherapy.
c. Medium choroidal melanoma; enucleation.
d. Large choroidal melanoma; transpupillary thermotherapy.

For answers, see page 122.

Diagnosis

Based on the clinical findings and imaging studies, the lesion most likely represents a small choroidal melanoma, although we were not 100% certain. Clearly, the patient displayed risk factors that make



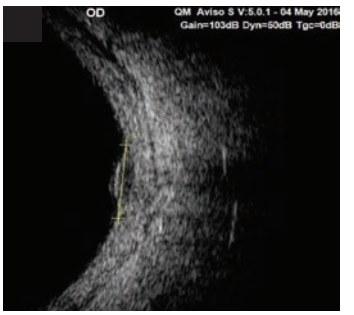


Fig. 3. Note the elevation in these B-scans of the lesion.

this lesion particularly worrisome, but because it is still quite small (5mm x 4mm x 1mm) and located near the fovea the ocular oncologist felt it was appropriate to observe it with close follow up to determine if it grows.

Discussion

Choroidal melanoma is the most common primary intraocular malignant tumor in adults, and the second most common site for malignant melanoma in the body.[1] They can either arise *de novo* or from a preexisting choroidal nevus.[2] Melanomas in the eye occur in the uveal tract, which includes the iris, ciliary body and choroid.[3] Only four to six primary choroidal melanomas cases are reported per one million cases a year in the United States, totaling about 1,400 annual cases, with a slightly higher rate in Scandinavian countries.[1,4,5,7] They are more common in middle-aged Caucasian men of Northern European descent with fair complexion and blue or green irises, typically with a mean age of 55 years.[1,7] Hispanic and Asian patients have a small risk, while the incidence in black patients is extremely low.[4] There is a possible association among arc welders, due to excessive UV light exposure, while an increased risk from a history of chronic sunlight exposure remains inconclusive.[1,2,4]

Patients with choroidal melanomas are typically asymptomatic, unless the tumor involves the macula, there is subretinal fluid, retinal detachment or even a vitreous hemorrhage. The most common symptoms include decreased visual acuity, as well as visual field deficits, flashes, floaters and, in rare cases, pain.[2,7] When present, pain can come from inflammation, tumor necrosis, neovascular glaucoma, massive extraocular extension or impingement of the long posterior ciliary nerves.[1,7] If large enough, the classic presentation of these lesions is a pigmented dome-shaped or

collar button-shaped tumor with an associated exudative retinal detachment and orange pigment at the level of the RPE.[1] Orange pigment, or lipofuscin, is composed of components from the incomplete metabolism of photoreceptor outer segments.[7]

Uveal melanomas can be quite variable in shape, size and presentation. They are typically elevated either like a mushroom or a dome but can be yellowish (amelanotic), dark-brown, green or grey in color.[7] Commonly, subretinal fluid is present, and with the progressive breakdown of the retinal pigment epithelium (RPE), an exudative retinal detachment can occur.[7] If the melanoma is large enough or involves the ciliary body, there can be the presence of prominent episcleral vessels, referred to as sentinel vessels.[1]

Treatment

The biggest challenge is trying to determine if a lesion is a suspicious nevus or a small choroidal melanoma, and the best way to differentiate between the two is to closely observe for growth over time. However, there are features that are more suggestive the lesion is a small melanoma such as orange pigment (lipofuscin), the presence of subretinal fluid, thickness greater than 2mm, largest base diameter greater than 7mm, visual symptoms, less than 3mm from the disc margin, ultrasound hollowness, and the absence of drusen and a halo.[1,4,7] The presence of one of the above risk factors increases the chance of growth from 3% to 38% over the course of five years, while the presence of two or more risk factors increases the risk of growth to more than 50%.[7] Along with low-medium internal reflectivity on the A-scan, our patient's lesion also has lipofuscin and subretinal fluid, suggesting it is likely a small choroidal melanoma. Choroidal nevi on the other hand are usually less than 2mm thickness and less than 6mm in diameter.[7] Drusen are often present in a suspicious nevus, indicating chronicity.

Besides recognizing the clinical features that suggest a choroidal melanoma, A-scan ultrasonography is perhaps the most sensitive and diagnostic test. Choroidal melanomas will show low-to-medium internal reflectivity while B-scans can show acoustically silent zones. The B-scan can also show a bright anterior aspect of the lesion, biconvexity, choroidal excavation and a solid mass shadowing of the orbit.[1,7] Together, A- and B-scans have a more than 95% sensitivity in diagnosing choroidal melanomas.[1] The value of the OCT is that it can show the presence of serous fluid as well as a choroidal lesion pushing up on the RPE.

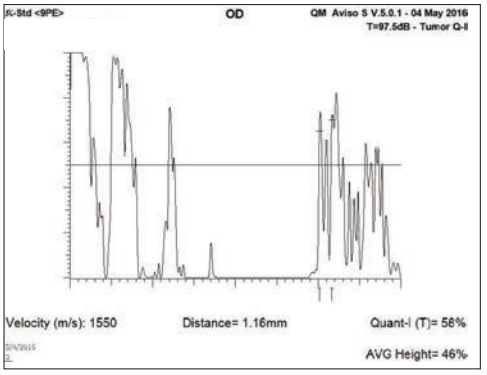


Fig. 4. What does this A-scan of the patient's right eye reveal?

The importance of determining if the lesion is malignant is to reduce the risk of metastasis and possible death. There is ongoing research exploring the molecular and genetic markers that can predict metastatic potential, and researchers believe that Monosomy 3 (loss of chromosome 3) is indicative of a more aggressive tumor.[1,2,6,9] Once a lesion is determined to be choroidal melanoma, treatment is initiated based on upon COMS (The Collaborative Ocular Melanoma Study) protocol.

Tumor treatment usually depends on the size of the lesion. Small melanomas, which are typically 2mm to 3mm in thickness and 6mm to 7mm in basal diameter, can be observed until evidence of growth. Once a decision is made to treat small melanomas, I-125 plaque radiotherapy is commonly used. With plaque therapy, a radioactive plaque is sutured on the surface of the globe exterior to the tumor for three to seven days depending on tumor size, with provides excellent control in about 98% of patients.[1,2,7] Other treatment options include external beam charged particle radiation, and transpupillary thermotherapy (TTT), among others.[1,7] TTT is done with a low-power infrared laser for a long duration, which is effective for small tumors about 3DD away from the ONH or center of vision.[2,7] It can be done in conjunction with plaque brachytherapy. Despite the diagnosis of a small choroidal melanoma, the estimated five-year mortality rate related to the tumor is only about 1% according to COMS.[8]

Medium size tumors, usually 3mm to 8mm thick and 7mm to 14mm in diameter, are usually treated with globe-sparing treatments such as I-125 (iodine) plaque brachytherapy, which showed about the same benefit in terms of mortality compared with enucleation.[1,6] From the medium arm of the COMS trial, patients had a 90% five-year survival in both the enucleated group and the plaque brachytherapy group.[8] Large tumors, which are greater than 8mm in thickness and greater than 14mm in diameter, are still generally enucleated although plaque therapy may be an option if a large enough plaque will fit over the tumor.[8] Unfortunately, however, according to COMS, the tumor-related mortality rate of patients with large choroidal melanomas is over 25% within five years, while the all-cause mortality is upwards of about 40%.[8]

Patients with uveal melanomas are always at risk for developing metastatic disease. In most of the studies, the larger the tumor, the greater the risk of metastasis.[9] Fortunately, about 98% of patients do not have metastatic disease at the time of diagnosis, but if a metastasis occurs, it is most often within the first year of diagnosis, although it can metastasize even more than 10 years after treatment.[4,7] Therefore, extensive testing is needed at the time of diagnosis such as CBC, liver enzymes, abdominal CT scan (or MRI or ultrasound) and a chest x-ray.[7] If, however, a distant metastasis is found at the time of diagnosis, treatment of ocular melanomas is simply palliative; the patient needs systemic chemotherapy.[1]

Unfortunately, the most common site of metastasis is to the liver, which has an estimated survival rate of about six months, compared with 19 months to 28 months in extrahepatic metastases.[1]

The choroidal lesion in our patient was relatively small, measuring 1mm in thickness and 5mm x 4mm in diameter. The ultrasound showed medium reflectivity as well as orange pigment and subretinal fluid involving his macula, all suggesting the lesion is a small choroidal melanoma. Because it was small, the ocular oncologist elected to follow the patient to determine growth. The patient was asked to return in two months for revaluation. ■

Dr. Brafman is an optometric resident at the Bascom Palmer Eye Institute in Miami.

1. Singh P, Singh A. Choroidal melanoma. J. Ophthalmol. 2012 Jan-April;5(1):3-9.
2. Choroidal Melanoma. WillsEye Hospital. 2011 Oct
5. Available www.willseye.org/health-library/choroidal-melanoma.
3. Finger P. Choroidal Melanoma. New York Eye Cancer Center. Available at: https://eyecancer.com/eye-cancer/conditions/choroidal-tumors/choroidal-melanoma/.
4. Komatsu T, Sowa T, Takahashi K, Fujinaga T. A case of solitary pulmonary metastasis of choroidal melanoma with an exceptionally long disease-free period. International Journal of Surgery Case Reports. 2013;4(10):849-51.
5. Tran E, Ma R, Paton K, et al. Outcomes of proton radiation therapy for peripapillary choroidal melanoma at the BC cancer agency. International Journal of Radiation Oncology, Biology, Physics. 2012 Aug;83(5):1425-31.
6. Marathe O, Jeffrey W, Lee S, et al. International Journal of Radiation Oncology, Biology, Physics. 2011 Nov 15;81(4):1046-8.
7. Weidmayer, S. Choroidal Melanoma is a Life Sentence. Rev Optom. 2012 Jan 15;149(1):48-55.
8. Margo C. The collaborative ocular melanoma study: an overview. Cancer Control. 2004 Sept-Oct;11(5): 304-9.
9. Finger P. Small Choroidal Melanoma: To Treat or Not To Treat, That is The Question! New York Eye Cancer Center. 1995. Available https://eyecancer.com/eye-cancer/research-articles/small-choroidal-melanoma-to-treat-or-not-to-treat-that-is-the-question/.

Up to 20 CE Credits*

ANNUAL

WINTER OPHTHALMIC CONFERENCE

A *REVIEW OF OPTOMETRY*® MEETING OF CLINICAL EXCELLENCE

CE AT ITS PEAK! WORLD CLASS EDUCATION BY LEADING OPTOMETRIC EDUCATORS

Early Bird Special: $75 OFF REGISTRATION Until Nov. 30, 2016

THE LONGEST RUNNING WINTER CE MEETING IN EYE CARE!

February 17-21, 2017

Aspen, Colorado

LOCATION:

WESTIN SNOWMASS

ALPINE CONFERENCE ROOM

100 Elbert Lane
Snowmass Village, CO 81615
Phone: (970) 923-8200

CONTINUING EDUCATION:

- Earn up to **20** hours of **COPE** CE* Credits
- **Registration Cost - $575**
- See website for meeting agenda

SPEAKERS:

Andrew Archila, OD
Jack Cioffi, MD
Fred Edmunds, OD
Murray Fingeret, OD
John Flanagan, OD, PhD, FCOptom
Ben Gaddie, OD
Howard Purcell, OD
Jack Schaeffer, OD
Leo Semes, OD

3 WAYS TO REGISTER

E-MAIL: REVIEWMEETINGS@JOBSON.COM **PHONE:** (866) 730-9257
WEBSITE: WWW.SKIVISION.COM

Discounted room rates: $199-$389/night
See website for all accommodations and rates

Also available on website: non-educational information including discounted lift tickets and Nastar racing opportunities.

Administered by
Review of Optometry®

cope® *Approval Pending


The *Other* Other Viral Infection

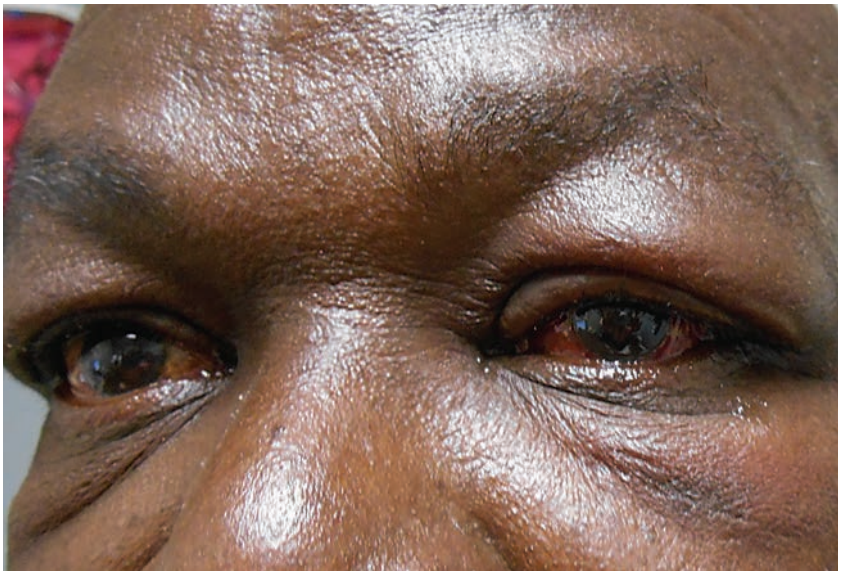
Just because a condition is a "zebra" doesn't mean you'll never see it.

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

A 59-year-old man presented acutely with a red, swollen, painful left eye that had been steadily worsening for several days. He reported crusty lids upon awakening and a brownish discharge. Gross evaluation revealed periorbital edema, extreme photophobia and chronic tearing, which was worse in the left eye than the right. Palpable, but not tender, preauricular nodes were present bilaterally. He denied any recent illness or known exposure to anybody with similarly affected eyes.

Examination

His visual acuities were 20/20 OD and 20/40 OS. Biomicroscopy demonstrated extensive subconjunctival hemorrhaging the left eye, involving nearly all of the visible bulbar surface. The inferior palpebral conjunctiva displayed a notable follicular reaction, with a dense, whitish pseudomembrane in the lower cul-de-sac. The left cornea displayed mild haze, but did not stain with fluorescein, and no infiltrates were observed. The right eye also displayed a mild inferior palpebral follicular reaction and bulbar hyperemia, but no subconjunctival hemorrhage; the right cornea was unremarkable. Bilaterally, his anterior chambers were deep and quiet. His intraocular pressure was measured at 11mm Hg OD and OS. The patient was diagnosed with acute hemorrhagic conjunctivitis (AHC).



This 59-year-old male patient's symptoms began in the left eye and spread to the right eye after several days.

Viral Rarities

When we discuss viral pathogens within the sphere of ocular disease, the most common entities that come to mind are likely members of the *Herpesviridae* family, most specifically herpes simplex and herpes zoster. Beyond these, physicians recognize that the adenoviruses—members of the *Adenoviridae* family—are also common pathogens, implicated in a variety of conjunctival infections such as pharyngoconjunctival fever and epidemic keratoconjunctivitis. However, other viral entities may impact the eye, particularly in cases of ocular surface infection.

AHC is a comparatively rare condition when contrasted with adenoviral conjunctivitis or herpes simplex keratitis. It is attributable to specific members of the Picornaviridae family, most commonly Coxsackievirus and enterovirus strains.[1-8] AHC can affect individuals of any age, race or gender, although it does appear to have a predilection for younger individuals, particularly those between ages 11 and 15 years.[7] The infection is more commonly noted to occur in developing nations, or in communities of comparatively lower socioeconomic status.[1-6] Epidemics have occurred in regions throughout the world since the early 1970s.[1-7] Although AHC is considered uncommon in the United States, outbreaks have been noted in warm, tropical areas such as Florida.[9,10] Investigators believe the viral pathogens—especially enterovirus—can survive and replicate

better at higher temperatures and humidity levels.[11]

The course of AHC typically begins innocuously enough, with itching and redness of the involved eye. Over the course of several days, the lids become increasingly swollen and serous discharge develops, causing lid crusting or matting upon awakening.[7] The fellow eye often becomes involved at this point. Pain intensifies, photophobia ensues and a low-grade fever is possible.[7] Viral-induced inflammation causes rapid dilation of small conjunctival blood vessels, which invariably rupture, resulting in petechial hemorrhages; these soon coalesce, resulting in conspicuous subconjunctival hemorrhaging that is characteristic of the disease.[7] The severely red eyes, increasing discomfort, discharge and compromised vision (from ocular discharge or corneal involvement) usually prompts patients to seek medical attention within a week. Examination will reveal the aforementioned findings, as well as a moderate-to-severe follicular response, variable punctate keratopathy and, in extreme cases, subepithelial corneal infiltrates. Preauricular lymphadenopathy is also possible, though it appears to occur in less than 10% of affected individuals.[7]

Making the Diagnosis

Detection of AHC is often empirical and based upon the clinical presentation, history and systematic exclusion of other, similar conjunctivitides, such as bacterial conjunctivitis, epidemic keratoconjunctivitis, adult inclusion conjunctivitis and atopic keratoconjunctivitis. Laboratory testing is considered impractical in the typical clinical setting, given the rapid course and non–sight threatening nature of the infection. Collecting conjunctival swab samples and subjecting them to reverse transcription polymerase chain reaction can detect the offending pathogen.[7]

Patients with AHC are extremely contagious, so employ proper disinfection protocols upon examination. Patients should likewise be warned about the potential to spread the disease. As with many viral pathogens, a specific antimicrobial therapy for picornavirus infection is not known to exist, and hence there is no particular treatment of choice for AHC. Most physicians endorse palliative therapy with cool compresses and ophthalmic lubricants as needed.

Although some discourage using topical steroids (due to a fear of potential corneal superinfection), we have found that corticosteroids significantly improve symptoms in patients with both adenoviral and acute hemorrhagic conjunctivitis and when used in conjunction with a broad-spectrum antibiotic the risk of superinfection is practically nil. Hence, we often prescribe a combination antibiotic/corticosteroid four times daily for these individuals. In all cases, AHC is self-limited and the infection resolves within a week after inoculation, although the subconjunctival hemorrhage and related periorbital edema may persist for up to 14 days.[8]

The presence of pseudomembrane in this case was atypical, as this feature is not commonly associated with AHC. Nonetheless, practitioners should be aware of the potential for membrane formation in all cases of ocular surface infection, and must always address them appropriately. Ideally, membranes should be removed from the conjunctiva thoroughly so as to prevent contracture, scarring, foreshortening of the fornix and potential symblepharon formation. This can sometimes be facilitated by simply using a cotton-tipped applicator under topical anesthesia. In our case, however, it required the use of small tissue forceps to gently peel away the pseudomembrane; this resulted in little discomfort and no further ocular insult.

Zebra Spotting

There's a popular metaphor in medicine that applies to diagnosis: When you hear hoof beats, think horses before zebras.

We find ourselves echoing this mantra over and over in our clinical teaching roles. Yet every once in a while, the zebra is bound to make an appearance. Recognizing key elements of rare conditions such as AHC and dealing with them appropriately can help to prevent anxiety, inappropriate therapy and poor outcomes. ■

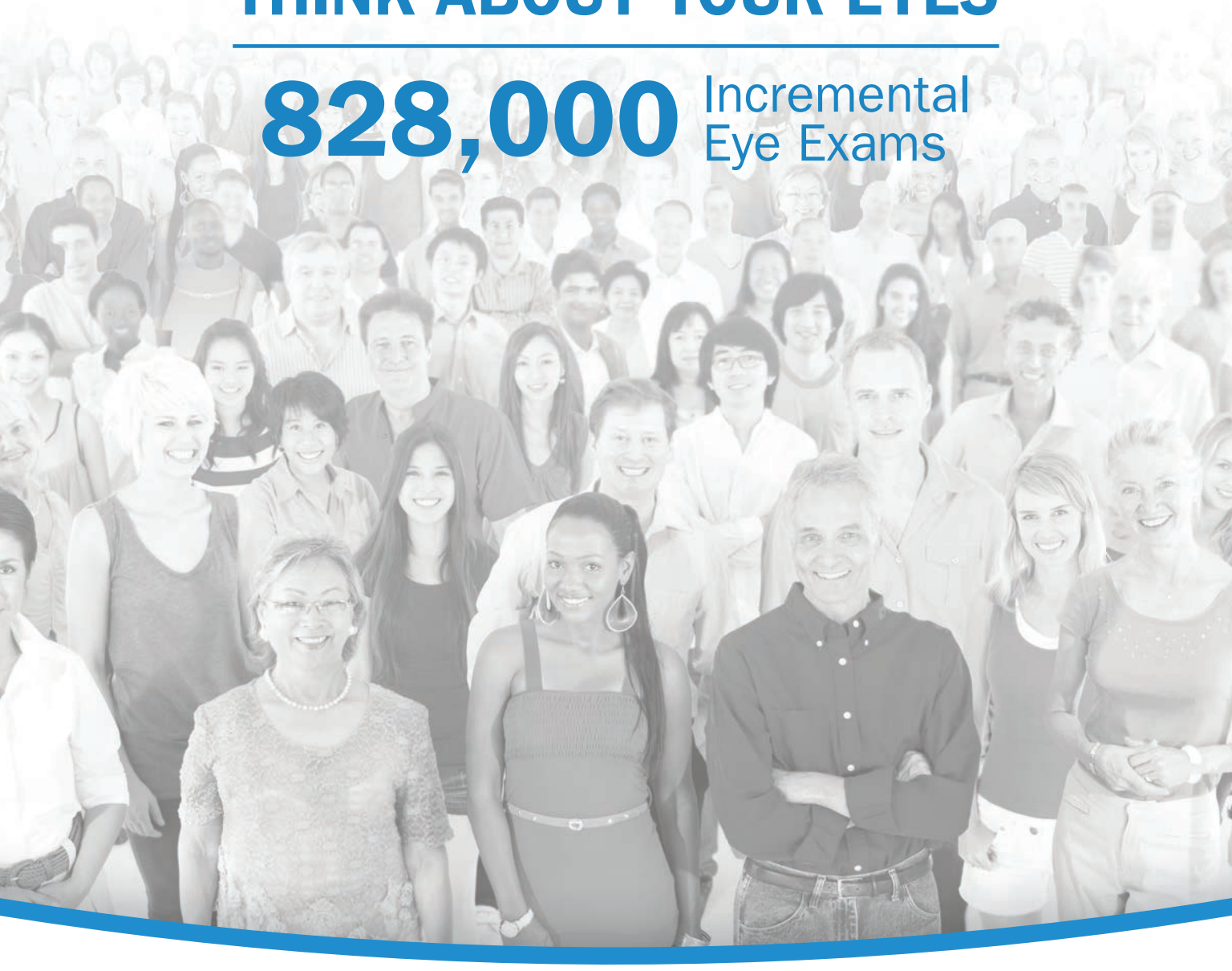
Special thanks to Gregory Wolfe, OD, for allowing us to share this case with our readers.

1. Mirkovic RR, Kono R, Yin-Murphy Met al. Enterovirus type 70: the etiologic agent of pandemic acute haemorrhagic conjunctivitis. Bull World Health Organ. 1973;49(4):341-6.
2. Moura FE, Ribeiro DC, Gurgel N, et al. Acute haemorrhagic conjunctivitis outbreak in the city of Fortaleza, northeast Brazil. Br J Ophthalmol. 2006 Sep;90(9):1091-3.
3. Gopalkrishna V, Patil PR, Kolhapure RM, et al. Outbreak of acute hemorrhagic conjunctivitis in Maharashtra and Gujarat states of India, caused by Coxsackie virus A-24 variant. J Med Virol. 2007 Jun;79(6):748-53.
4. Khan A, Sharif S, Shaukat S, et al. An outbreak of acute hemorrhagic conjunctivitis (AHC) caused by coxsackievirus A24 variant in Pakistan. Virus Res. 2008 Oct;137(1):150-2.
5. Centers for Disease Control and Prevention (CDC). Notes from the field: acute hemorrhagic conjunctivitis outbreaks caused by coxsackievirus A24v — Uganda and southern Sudan, 2010. MMWR Morb Mortal Wkly Rep. 2010 Aug 20;59(32):1024.
6. Aubry C, Gautret P, Nougairede A, et al. 2012 outbreak of acute haemorrhagic conjunctivitis in Indian Ocean Islands: identification of Coxsackievirus A24 in a returned traveller. Euro Surveill. 2012 May 31;17(22). pii: 20185.
7. Harada K, Fujimoto T, Asato Y, Uchio E. Virological and epidemiological analysis of coxsackievirus A24 variant epidemic of acute hemorrhagic conjunctivitis in Okinawa, Japan, in 2011. Clin Ophthalmol. 2015 Jun 15;9:1085-92.
8. Langford MP, Anders EA, Burch MA. Acute hemorrhagic conjunctivitis: anti-coxsackievirus A24 variant secretory immunoglobulin A in acute and convalescent tear. Clin Ophthalmol. 2015 Sep 10;9:1665-73.
9. Sklar VE, Patriarca PA, Onorato IM, et al. Clinical findings and results of treatment in an outbreak of acute hemorrhagic conjunctivitis in southern Florida. Am J Ophthalmol. 1983 Jan;95(1):45-54.
10. Hatch MH, Malison MD, Palmer EL. Isolation of enterovirus 70 from patients with acute hemorrhagic conjunctivitis in Key West, Florida. N Engl J Med. 1981 Dec 31;305(27):1648-8.
11. Mimura T, Usui T, Yamagami S, et al. Recent causes of subconjunctival hemorrhage. Ophthalmologica. 2010;224(3):133-7.

Strength in Numbers

THINK ABOUT YOUR EYES

828,000 Incremental Eye Exams



Think About Your Eyes (TAYE) has been instrumental in driving more eye exams in America. In 2015, there was a dramatic increase in incremental eye exams - an average of 23 more exams per practice! More eye exams mean better eye health and more patients in your practice.

thinkaboutyoureyes.com®

Brought to you by the AOA
AMERICAN OPTOMETRIC ASSOCIATION

Is your company supporting this important campaign?

If you are interested in joining, contact jtorrey@thinkaboutyoureyes.com

Product Review

Diagnostic Technology

Orbscan 3

Practitioners can now experience an improved Orbscan model by Bausch + Lomb. The new Orbscan 3 features multiple enhancements to serve as an indispensable screening tool for refractive procedures, according to the company.

The device offers the ability to examine anterior and posterior astigmatism and optical pachymetry, providing corneal biomechanic data and stability to inform surgical choices and identify appropriate candidates, according to B+L.

Orbscan 3 provides a contact-free analysis of the anterior segment using a slit scan. It also features user-friendly touch screen technology and new software, according to B+L.

Visit www.bausch.com.

Nidek Microperimeter

Nidek's microperimeter has enhanced functionality in the form of a new model, the MP-3. It now includes a wider range of stimulus intensity, from 0 to 34 dB, than the MP-1, according to Nidek. The MP-3 measures perimetric threshold values, even for normal eyes, while a maximum stimulus luminance of 10,000 asb allows evaluation of low-sensitivity, according to Nidek. The MP-3 comes with auto tracking and auto alignment functions, reducing variations between examiners for well-aligned follow-up exams, according to Nidek.

The device allows better evaluation of retinal morphology with the 12-megapixel fundus camera's high-res images and easy image acquisition, according to Nidek.

Visit Nidek.com.

Corneal Shape Analyzer Upgrades

Cassini's Corneal Shape Analyzer has gotten new upgrades that improve pre- and post-op surgical assessment. Its posterior cornea measurement capability received an algorithm update that provides customized total cornea data for refractive cataract surgery, according to Cassini. Stability tracking detects excessive eye movement and displays a warning signal to reduce error. And faster acquisition times provide a better user and patient experience, as this helps ensure the quality of data and reduces re-scans, according to Cassini.

Visit i-optics.com. ■

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.

Meetings + Conferences

November 2016

■ **18-20.** *Monterey Symposium 2016*. Monterey Marriott and Conference Center, Monterey, CA. Hosted by: California Optometric Association. CE hours: 50 total, 20 per OD. To register, email Sarah Harbin at sharbin@coavision.org, call (916) 266-5022 or go to www.coavision.org.

■ **19.** *Southwestern Optometric Glaucoma Symposium.* JW Marriott Houston, Houston, Texas. Hosted by: *Review of Optometry*. CE hours: 6. To register, go to www.reviewofoptometry.com/swogs2016.

December 2016

■ **1.** *Evening of Education*. UAB School of Optometry, Birmingham, AL. Hosted by: UAB School of Optometry. CE hours: 2. To register, email Amanda Kachler at uabsoce@uab.edu, call (205) 934-5701 or go to www.uab.edu/optometry/ce.

■ **2-3.** *A Terrific Tulsa Winter Weekend*. Hard Rock Hotel and Casino, Catoosa, OK. Hosted by: Northeastern State University, Oklahoma College of Optometry. CE hours: 8. To register, email Callie McAtee at mcateec@nsuok.edu.

■ **2-4.** *Retina Update 2016*. The Westin Kierland, Scottsdale, AZ. Hosted by: Optometric Retina Society and *Review of Optometry*. CE hours: 14. To register, go to: http://jobson.cvent.com/d/4fqyyn/1Q.

■ **3-4.** *Cornea, Contact Lens & Contemporary Vision Care Symposium*. Westin Memorial City, Houston. Hosted by: University of Houston College of Optometry. Key faculty: Jan Bergmanson. CE hours: 16. To register, email optce@central.uh.edu, call (713) 743-1900 or go to http://ce.opt.uh.edu.

■ **4.** *Clinical Topics in Optometry.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: Marshall B. Ketchum University. CE hours: 8. To register, email Antoinette Smith at asmith@ketchum.edu, call (714) 872-5684 or go to www.ketchum.edu/ce.

■ **5-6.** *Malinovsky Ocular Disease Seminar*. IU School of Optometry, Bloomington, IN. Hosted by: Indiana University School of Optometry. Key faculty: S.P. Srinivas, Don Lyon, Kimberly Kohne, Jeffrey Perotti, Todd Peabody, Patricia Henderson. CE hours: 14. To register, email Cheryl Oldfield at coldfiel@indiana.edu, call (812) 856-3502 or go to www.opt.indiana.edu/ce/seminars.htm.

■ **9-10.** *West Coast Optometric Glaucoma Symposium.* Hilton Waterfront Beach Resort, Huntington Beach, CA. Hosted by: *Review of Optometry*. CE hours: 12. To register, go to www.reviewofoptometry.com/wcogs2016.

■ **17.** *OD Excellence Information Meeting.* Office of Steve Chander, OD, Chicago, Ill. Hosted by: OD Excellence. Key faculty: Steve Chander. CE hours: 2. To register, email Anthony Senender at asenander@odexcellence.com, call (707) 433-5542 or go to odexcellence.com.

■ **24-31.** *A Day in My Retina Clinic*. Norwegian Cruise Line's Norwegian Escape, Eastern Caribbean Cruise from Miami. Hosted by: Dr. Travel Seminars. Key faculty: Jay Haynie. CE hours: 16. To register, email Robert Pascal at DrTravel@aol.com, call (800) 436-1028 or go to www.DrTravel.com.

■ **18.** *AHI Winter Optometry Seminar*. Hyatt Place Orlando Airport, Orlando, FL. Hosted by: American Health Innovations. CE hours: 10 total, 4 per OD. To register, email Waseem Khan at waseem@americanhealthinnovations.com, call (561) 398-5609 or go to www.americahealthinnovations.com/orlando2016/.

January 2017

■ **5-8.** *January Advanced Procedures Course.* Oklahoma College of Optometry Academic Wing, Tahlequah, OK. Hosted by: Oklahoma College of Optometry. Key faculty: Nathan Lighthizer, Richard Castillo, Doug Penisten, Joseph Shetler. CE hours: 32. To register, email Callie McAtee at nsuoco_ce@nsuok.edu or go to optometry.nsuok.edu/ContinuingEducation.aspx.

■ **7-11.** *Art & Science of Optometric Care—A Behavioral Perspective.* Vision Sense, Nova Scotia, Canada. Hosted by: Optometric Extension Program Foundation. Key faculty: Steen Aalberg. CE hours: 35. To register, email Karen Ruder at Karen.ruder@oep.org, call (410) 561-3791 or go to www.oepf.org.

■ **13.** *PRK Certification Course.* Lesley L. Walls Vision Center, Broken Arrow, OK. Hosted by Oklahoma College of Optometry. Key faculty: Jo'el Sturm, Dawn Holsted. CE hours: 10. To register, email Callie McAtee at nsuoco_ce@nsuok.edu or go to optometry.nsuok.edu/ContinuingEducation.aspx.

■ **13-15.** *AZOA 2017 Bronstein Contact Lens & Cornea Seminar*. Hilton Scottsdale Resort Villas, Scottsdale, AZ. Hosted by: Arizona Optometric Association. CE hours: 19. To register, email Kate Diedrickson at kate@azoa.org or go to www.azoa.org/Connect.

■ **14.** *Arkansas Optometric Association 2017 Coding Update.* Embassy Suites, Little Rock, AR. Hosted by: Arkansas Optometric Association. Key faculty: John McGreal. CE hours: 4. To register, email Vicki Farmer at aroa@arkansasoptometric.org, call (501) 661-7675 or go to www.arkansasoptometric.org.

■ **14.** *Glaucoma Symposium 2017*. Willows Lodge, Woodinville, WA. Hosted by: Pacific University College of Optometry. Key faculty: Howard Barnebey, Murray Fingeret. CE hours: 7. To register, email Martina Fredericks at frederim@pacificu.edu, call (503) 352-2207 or go to www.pacificu.edu. ■

To list your meeting, please send the details to:

Rebecca Hepp, Managing Editor

Email: rhepp@jobson.com

Phone: (610) 492-1005

Up to 12 CE Credits*

2016 WEST COAST OPTOMETRIC GLAUCOMA SYMPOSIUM

DECEMBER 9 - 10

Register Today!

Join our faculty of renowned ODs, MDs and PhDs for a highly interactive meeting covering the most up-to-date information in glaucoma care.
Up to 12 CE credits for only $275.

HILTON HOTEL HUNTINGTON BEACH

Waterfront Beach Resort
21100 Pacific Coast Highway
Huntington Beach, CA 92648

Discounted room rate $219/night plus tax. Please call the Hilton Waterfront Resort directly at 800-822-7873 and mention WCOGS for group rate. Rooms limited.

MEETING CO-CHAIRS

Murray Fingeret, OD

Robert N. Weinreb, MD

Speakers

Ben Gaddie, OD • Richard Madonna, MA, OD
Anthony Realini, MD • Derek Welsbie, MD, PhD

3 WAYS TO REGISTER:

Online: www.reviewofoptometry.com/WCOGS2016
E-mail: reviewmeetings@jobson.com
Call Lois DiDomenico: 877-451-6514

Administered by
Review of Optometry

cope®
*Approval pending

Partially supported by an unrestricted educational grant from
Alcon

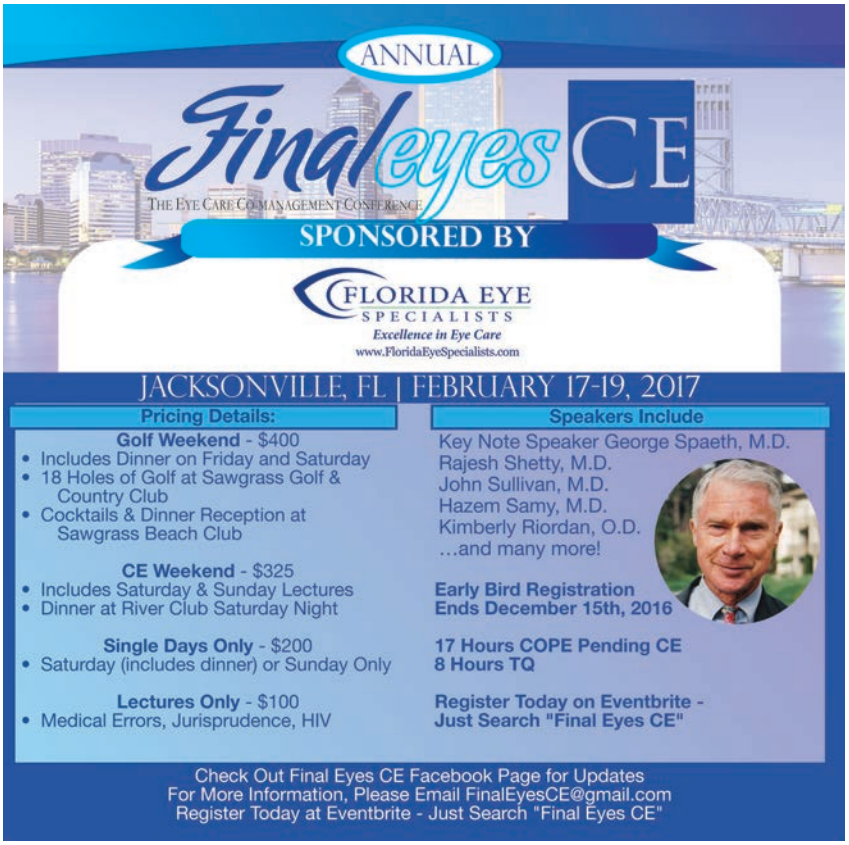
Merchandise Offered

Contact Lenses

Merchandise Offered

Merchandise Offered

Continuing Education



Practice For Sale

PRACTICE FOR SALE: ABERDEEN, WA

Ophthalmology/optometry office and practice for sale (including Broadway Optical shop) in Aberdeen,WA. Appraised value for practice and equipment $300,000. Office building separately appraised $358,000. Both together are offered $499,000. Practice and equipment can be purchased separately, with rental lease on the building. Aberdeen is the western gateway to Olympic NP, a paradise for the outdoorsman/sportsman. Address: Aberdeen Cataract & Laser, 118 W. 1st St., Aberdeen, WA 98520.

Contact owner Dr. William Hoot (817) 925-6918 (wrhoot@att.net) or office manager Joanne Watters at (360) 590-4636, (jkwttrs@comcast.net) or at office phone (360) 533-5800 Monday or Tuesday

PRACTICE SALES & APPRAISAL

Expert Services for:

- ☑ **Buying or Selling a Practice**
- ☑ **Practice Appraisal**
- ☑ **Practice Financing**
- ☑ **Partner Buy-in or Buy-out**

Call for a Free Consultation
(800) 416-2055
www.TransitionConsultants.com

ARIZONA PRACTICE FOR SALE

Thriving 25-year-old, full-time low vision rehabilitation practice for sale.

TWO LOCATIONS, Sun City, AZ and Mesa, AZ. Practice is still growing, current gross $2 million. Owner is retiring but willing to stay through the transition.

For more information, please inquire at info@ViewFinderLowVision.com

Career Opportunities

Anchorage, AK.

Full time Optometrist

opening with Alaska's premier eye care practice – **Alaska Eye Care Centers, APC.** A privately owned, two location practice with 7 doctors is seeking a new graduate or experienced O.D. for employment opportunity.

Each location has 4-5 full exam rooms and the appropriate equipment, special testing equipment, optometric pre-testers, and full administrative support. The main office offers full lens manufacturing for both locations. Each office has optical dispensaries consisting of over 2,500 frames and is serviced by licensed opticians. In addition, each office has full contact lens services, including licensed CL technicians.

This opening is for our Wasilla location which is a semi-rural setting approximately 40 miles from Anchorage with rotation into our Anchorage office once a week, more when other providers are on vacation. The population of the surrounding area is 50,000+ and most services are offered. Housing and utilities are comparable to West Coast levels. Entertainment, fine dining and the arts are easily available in Anchorage, a city of more than 298,000 residents.

Alaska is one of the most beautiful places to live and practice. **For further information about this GREAT opportunity, please contact:**

Alaska EyeCare CENTERS

Alaska Eye Care Centers
1345 W. 9th Avenue, Anchorage, AK. 99501
Contact: Deb Foster, Business Administrator
deb@alaskaeyecare.com
(907) 272-2557 X 1604 • (907) 274-4932 (Fax)
www.alaskaeyecare.com

STAFF OPTOMETRIST

Bard Optical is a leading Midwest vision care organization in business for over 70 years and we are still growing. The company is based in Peoria, IL with 20 retail offices throughout the central Illinois area, as far north as Sterling and as far south as Jacksonville. Once again this year we were named to the Top 50 Optical Retailers in the United States by Vision Monday – currently ranking 37th. A progressive optometric staff is vital to the continued growth of our organization whose foundation is based on one-on-one patient service. We are currently accepting CV/resumes for optometrists focused on full scope primary medical patient care. The candidate must have an Illinois license with therapeutics. The practice includes (but is not limited to) general optometry, contact lenses, and geriatric care. Salaried, full-time positions are available with excellent growth programs and benefits. Some part-time opportunities may be available also. Please email your information to **hr@bardoptical.com** or fax to **309-693-9754**. Mailing address if more convenient is **Bard Optical, Attn: HR, 8309 N Knoxville Avenue, Peoria, IL 61615**. Ask about opportunities within Bard Optical. *We have openings in several existing and new offices opening soon in central Illinois.*

Bard Optical is a proud Associate Member of the Illinois Optometric Association.

BARD OPTICAL

www.bardoptical.com

IT STILL EXISTS!

Delaware private practice with like new 3,500 square foot **free standing building**, custom designed and built to be an eye care facility. Immediate opening for an Associate or Partnership leading to purchase in a unique state with **no corporate practice** (OD's can't practice in Pearl Vision or LensCrafters per state law). Large concentration of Glaucoma and Ocular Disease. **Grossing $700K plus** with high net and phenomenal growth. **Will sell or lease building**. Extensive website with e-commerce included. Only 1/2 hour to Philadelphia or Baltimore, and 1 hour to the beach.

Call 302-994-2299 or e-mail **confidentially**
privpracticeopportunity@gmail.com

OPTOMETRIST

Busy Ophthalmology Practice in Bronx, NY seeking highly-skilled, seasoned Optometrist, part-time. Experience in post-operative care, dry eye, allergies and pediatrics preferred.

Contact:
gscalera@nyeye.com
or fax (718) 405-3808

Continuing Education

Medical Facility Optometrists

The American Board of Certification in Medical Optometry is utilized at accredited medical facilities to verify specialist-level competence in medical optometry. ABCMO certifies specialized competence of a higher level than that required for licensure and general practice.

Visit **www.abcmo.org** to learn why medical facilities require board certification of specialists and why only ABCMO board certification in the specialty of medical optometry is accepted. Questions? **myers.kenj@gmail.com**.

NOTICE: After August 1, 2003, all applicants for ABCMO board certification must have completed an ACOE residency in medical optometry and passed the Advanced Competence in Medical Optometry examination of the NBEO.

www.abcmo.org

Scientia est Potentia

Equipment and Supplies

Motorized Pretesting Tables FOR LESS!

Save hundreds for just shopping on line.

Go to EBAY and search the words PRETESTING TABLES and start saving on all your pretesting needs or

EMail: eyecareauctions@gmail.com

Products and Services

OD ONLINE

COMPLETE PM AND CERTIFIED EHR SOFTWARE
- 2014 Edition

All inclusive solution to enrich patient care and increase profitability for unlimited devices and users.

GET A FREE DEMO AT WWW.ODONLINE.NET

FIND MORE PRACTICES FOR SALE ON

opticlassifieds

CONTACT US TODAY to list your practice: 888-498-1460
opticlassifieds@kerhgroup.com

www.opticlassifieds.com

REVIEW® OF OPTOMETRY

Looking to increase sales?

Place Your Ad here.

Contact us today for classified advertising:
888-498-1460
E-mail: **sales@kerhgroup.com**

Up to 14 CE Credits*

REGISTER TODAY!

THE OPTOMETRIC RETINA SOCIETY AND *REVIEW OF OPTOMETRY* PRESENT:

RETINA UPDATE 2016

DECEMBER 2-4, 2016
SCOTTSDALE, ARIZONA

The Westin Kierland Resort

6902 E. Greenway Pkwy.
Scottsdale, Arizona 85254
Room rate: $189/night
Reservations: (480) 624-1000 or (800) 354-5892
Alaska and Hawaii: (800) 228-1212

Course Topics

- Posterior Segment Technology **Workshop**
- Dr. Larry Alexander Memorial Lecture
- Technology for the Posterior Segment
- Angiography With and Without the Dye
- Hospital-based Retina Grand Rounds
- Retinal Diseases You Don't Want to Miss
- Inherited Retinal Disease
- Co-Management in the ICD-10 Era
- Medical Coding and Compliance Issues

Registration Cost:
ORS Members $445
Non-members $495

3 Ways to Register

Online:
www.reviewofoptometry.com/ORSRETUPDATE2016

Email Lois DiDomenico:
ReviewMeetings@Jobson.com

Phone:
(800) 999-0975

ORS Mission Statement

"The mission of the Optometric Retina Society (ORS) is to promote the advancement of vitreoretinal knowledge for clinicians, ophthalmic educators, residents, and students. The ORS is dedicated to posterior segment disease prevention, diagnosis, management and co-management."

Co-Chairs

Brad Sutton, OD

Joseph Pizzimenti, OD

Faculty

Pravin Dugel, MD

Steven Ferrucci, OD

Len Koh, OD, PhD

Blair Lonsberry, OD

Mohammad Rafieetary, OD

John Rumpakis, OD

Leo Semes, OD

Chris Suhr, OD

Gay Tokumaru, OD

Administered by
Review of Optometry®

cope®
*Approval pending

The Optometric Retina Society and *Review of Optometry*® present:

RETINA UPDATE 2016
DECEMBER 2-4

Up to 14 CE Credits*

THE WESTIN KIERLAND RESORT
6902 E. GREENWAY PKWY. - SCOTTSDALE, ARIZONA 85254

Discounted room rate: $189 per night, plus applicable taxes and fees.
For reservations call the hotel directly and identify yourself as a participant of "Retina Update 2016" for group rate. Discounted room rates available 3 days pre- and post- conference, based on hotel availability.

Registration Information

Name | NPI # (NPI numbers will only be used for Sunshine reporting purposes) | OE Tracker #

Practice Affiliation

Mailing Address | City | State | Zip Code

Telephone | Fax | Email

Name Badge Information (please print clearly)

My Name | My Guest(s)

Payment Information

	Rate per person		No. in party		Subtotal
OD Registration - $495 (includes up to 14 hours of CE, breakfasts, lunches, reception) Call for daily and student rates.	$495	x	_____	=	$_____
Additional Guest(s) - $45 (12 years and older, reception only)	$45	x	_____	=	$_____

Complimentary Workshop (Friday, December 2, 5:45pm - 7:45pm)

❑ Posterior Segment Technology Workshop - (Earn 2 additional CE Credits)

❑ Check enclosed (make checks payable to Review of Optometry)

Charge my: ❑ American Express ❑ Mastercard ❑ Visa

Credit Card Number | Exp Date

Cardholder (print name)

Signature

CONFERENCE CANCELLATION POLICY

Full refund on registration fee until October 14, 2016

50% refund on registration fee until November 4, 2016

No refund past November 4, 2016

For more information or to register, contact Lois DiDomenico at 800-999-0975 or at ReviewMeetings@Jobson.com.

Administered by: *Review of Optometry*®

MAIL FORM : Review Group Meetings c/o Jobson
11 Campus Blvd, Ste. 100
Newtown Square, PA 19073

FAX FORM: Review Meetings Group
610-492-1039

cope®
*Approval pending

Images courtesy of The Westin Kierland Resort

Diagnostic **Quiz**

More Than Just Foggy Glasses

By Andrew S. Gurwood, OD

History

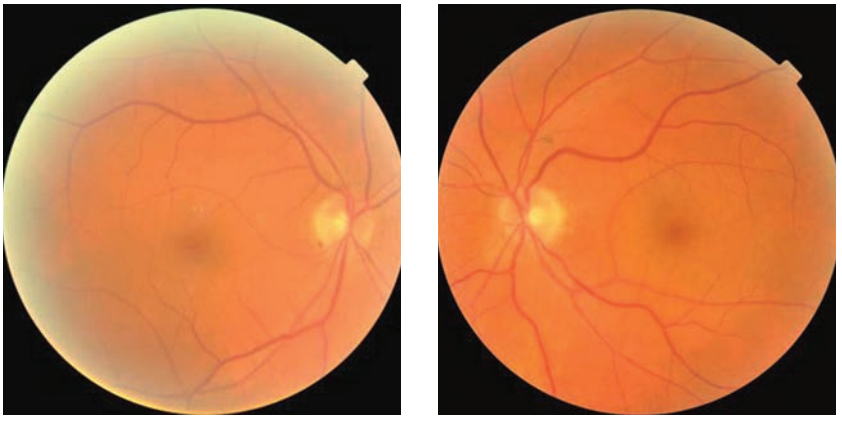
A 56-year-old man presented with a chief complaint of blurred vision for the previous six months. He explained that his spectacles were old and that he wanted new ones with an updated prescription.

He had no known previous ocular or systemic history and reported no allergies.

Diagnostic Data

His best corrected visual acuities were 20/30 OD and 20/30 OS at distance and 20/40 OU at near. External examination was normal and we saw no evidence of afferent pupillary defect.

Refraction revealed symmetrical, spherical myopia with a +0.50D change in the add yielding 20/25 acuity at near. No improvement could be achieved at distance.



These fundus photos show the eyes of a 56-year-old male patient who has suffered from blurred vision for six months. Can the images help you identify the cause of his condition?

The anterior segments of both eyes were in good health and applanation intraocular pressures measured 19mm Hg OU. The pertinent dilated fundus finding OU is demonstrated in the photographs.

Your Diagnosis

Does this case require additional tests? How would you manage this patient? What is the likely prognosis? To find out, please visit www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 113):* 1) d; 2) d; 3) b; 4) a; 5) b.

Next Month in the Mag

In December, *Review of Optometry* will present its 22nd annual surgery report.

Topics include:

- *Femtosecond Cataract Surgery and New Premium IOLs: Options and Optimism*
- *Postoperative Cataract Complication Management Strategies for the Optometrist*
- *Corneal Surgery Basics for Comanaging ODs*

Also in this issue:

- *Telemedicine for Diabetic Retinopathy Screening: The VA Experience*
- *The Protocols of Contemporary Dry Eye Diagnosis*
- *Annual Income Surgery: How Did ODs Fare This Year?*
- *Taking on Ophthalmic Migraines*

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.

BAUSCH + LOMB

Biotrue® ONEday lenses

For patients who want to **start and end the day with more moisture** than leading daily disposables[1]

Surface Active Technology™

With its unique dehydration barrier, Biotrue® ONEday maintains **98% of its moisture for up to 16 hours[2]**

Lens Cross Section

- PVP (hydrophilic polyvinylpyrrolidone) allows for a high water lens that is **78% water—the same as the cornea.[3]**
- Poloxamer 407, a Surface Active Macromer, forms a dehydration barrier that helps Biotrue® ONEday **start and end the day with more moisture.[1]**
- Like the lipid layer, Biotrue® ONEday provides a smooth optical surface and **resists dehydration.**

Prescribe Biotrue® ONEday daily disposable contact lenses

For more information, call 1-800-828-9030, contact your Sales Representative or log on to Bausch.com/ecp

Also available for Presbyopia

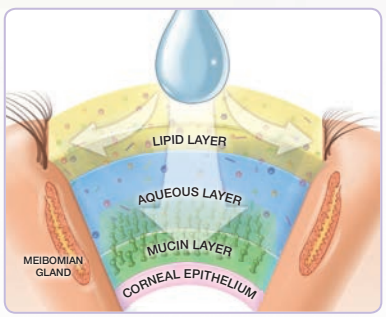
REFERENCES: 1. Data on file. Bausch & Lomb Incorporated. Rochester NY; 2015. **2.** Data on file. Bausch & Lomb Incorporated. Rochester NY; 2012. **3.** Bergmanson J. Clinical Ocular Anatomy and Physiology, 14th edition, 2007.

BAUSCH + LOMB
See better. Live better.

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

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]

Alcon®
a Novartis company

 05/14 SYS14005JAD-B

Systane® Family of Products

Relief that lasts