Motivate Your Patients with Meibomian Gland Imaging Techniques, **PAGE 54**

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OCTOBER 15, 2017

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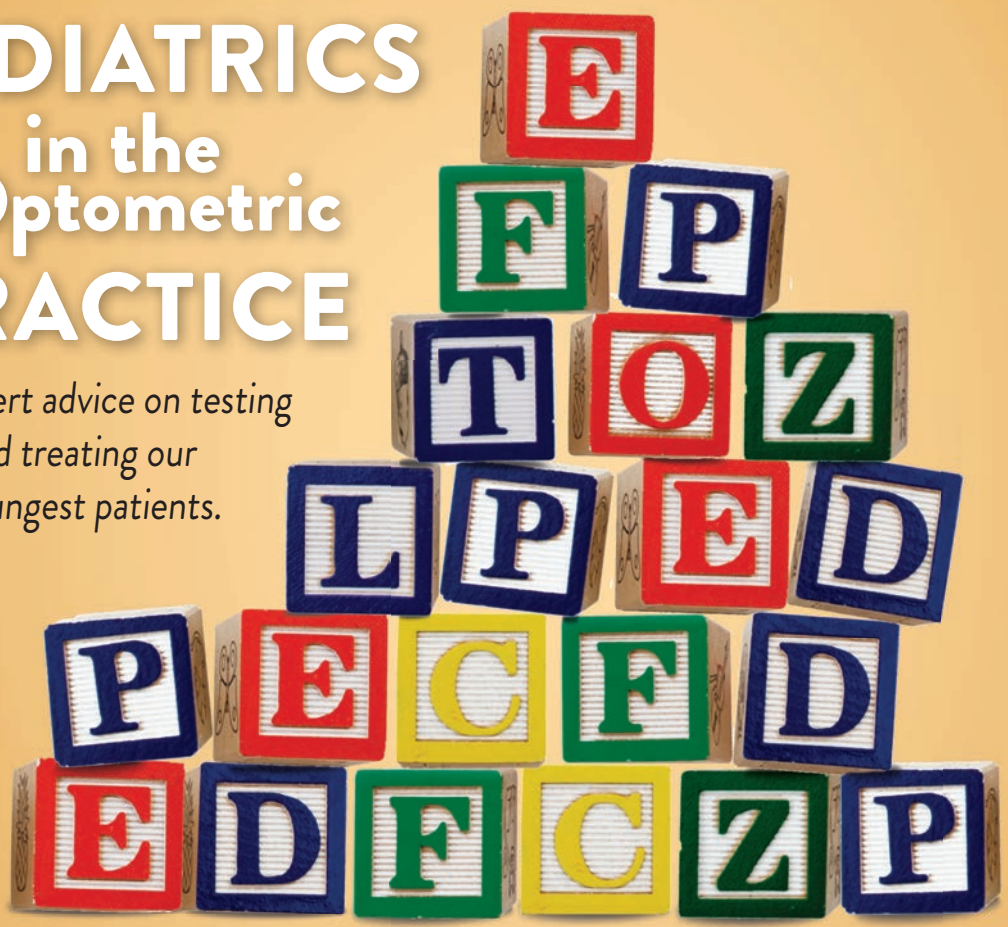

Testing Children for Accommodative and Convergence Disorders, **PAGE 62**

Managing Amblyopia: Can Vision Therapy Cut It?, **PAGE 74**

Kids and Pharmaceuticals: How to Tailor Your Therapy, **PAGE 81** – EARN 2 CE CREDITS

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xiidra® (lifitegrast ophthalmic solution)5%

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OVER

what Dry Eye patients have been waiting for

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 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.

US Patents: 8367701; 9353088; 7314938; 7745460; 7790743; 7928122; 9216174; 8168655; 8084047; 8592450; 9085553; 8927574; 9447077; 9353088 and pending patent applications.
Last Modified: 12/2016 S26218

VOL. 154 NO. 10 ■ OCTOBER 15, 2017

IN THE NEWS

The enzyme **leukotriene B_4 may be to blame for end-of-day contact lens (CL) discomfort**, according to new research. Investigators collected evening tears from 22 symptomatic and 23 asymptomatic CL wearers, then measured the prostaglandins, leukotriene B_4 and cysteinyl leukotrienes. They found no significant different in most enzyme levels, with the exception of leukotriene B_4, which may play a role in lower comfort scores reported by symptomatic CL wearers.

Masoudi S, Stapleton FJ, Willcox MD. Differences in tear film biochemistry of symptomatic and asymptomatic lens wearers. Optom Vis Sci. 2017;94(9):914-8.

British researchers found prescribing **a supplement that contains antioxidants, zinc and copper may improve the long-term outcomes of patients with wet age-related macular degeneration (AMD)**. After analyzing data from AREDS, UK treatment costs and AMD prevalence figures for patients older than 55, the researchers calculated that those who take a supplement would need **eight fewer injections of anti-VEGF** over the course of their lifetime.

Lee AY, Butt T, Chew E, et al. Cost-effectiveness of age-related macular degeneration study supplements in the UK: combined trial and real-world outcomes data. British J Ophthalmol. 2017. [Epub ahead of print].

After studying 11 professional and 31 amateur musicians, investigators discovered **intraocular pressure (IOP) often rises after playing wind instruments**. Although they found similar or even higher IOP levels occurred during other daily activities or at night (as revealed by 24-hour IOP monitoring), they concluded the IOP rise due to playing a wind instrument may play a role in managing glaucoma progression in this subset of patients.

de Crom R, Webers C, van Kooten-Noordzij M, et al. Intraocular pressure fluctuations and 24-hour continuous monitoring for glaucoma risk in wind instrument players. J Glaucoma. 2017. [Epub ahead of print].

Model Shows Promise for Macular Disease

Human stem cells may open the door for better research—and treatment targets.

By Rebecca Hepp, Managing Editor

Without an effective, patient-derived model to work with, researchers have struggled to study macular dystrophies in the laboratory setting. But that might change with a new study in which researchers re-programmed skin cells from patients with three forms of macular dystrophy into stem cells, from which they created retinal pigment epithelium (RPE) cells.[1] When aged in a dish, these cells mimicked several characteristics of the disease—effectively giving the researchers a working human stem cell model.[1]

"Now we can actually identify and test a rational drug therapy in patients' own cells," Ruchira Singh, PhD, lead author of the study, said in a press release. "So far, this has not been possible, but now we can actually study macular diseases in parallel and identify what might be the central defect across macular diseases."[2]

"This study reveals some interesting observations about the role of the RPE in specific similar maculopathies," says Sara Weidmayer, OD, a clinical assistant professor in the Department of Ophthalmology and Visual Sciences at the University of Michigan.

Already, the model has helped researchers discover that: (1) dysfunctional RPE cells can cause specific aspects of degeneration without the help of other cells or the retina, and (2) a group of molecules within the RPE cells could be targeted by new pharmaceuticals.[2]

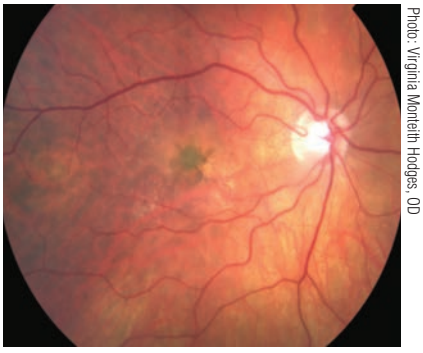

Photo: Virginia Monteith Hodges, OD

The new model may lead to treatment discoveries for macular dystrophies, such as vitelliform maculopathy.

"These discoveries are exciting and may open the door for continued research to further the understanding of the pathophysiologic mechanisms of macular dystrophies and degeneration," Dr. Weidmayer says. "Better understanding each retinal cell layer's role in the development of disease should spur advances that can directly improve patient outcomes in the future."

1. Galloway CA, Dalvi S, Hung SSC, et al. Drusen in patient-derived hiPSC-RPE models of macular dystrophies. Proceedings of the National Academy of Sciences. 2017:201710430.
2. University of Rochester Medical Center. New model for hard-to-study form of blindness paves way for future research. ScienceDaily. September 6, 2017. www.sciencedaily.com/releases/2017/09/170906170129.htm. Accessed September 13, 2017.

Study: Retina Sheds Light on Alzheimer's

A new imaging modality in development may help clinicians detect early signs of Alzheimer's disease (AD). It is well established that the accumulation of amyloid β-protein (Aβ) deposits in the brain may be a biomarker for AD. Currently, brain Aβ is measured with PET scans and cerebrospinal fluid analysis.[1]

The new imaging technique, relying on fundus autofluorescence (FAF) photography, suggests corresponding deposits of Aβ can be found in the retina. The researchers studied donated eyes and brains of Alzheimer's patients and controls, as well as high-resolution FAF imaging of Aβ in the eyes of AD patients—the first time technology shows potential to scan living patients, according to the researchers.[1,2]

They found a 4.7-fold increase in retinal plaque burden in 16 patients with AD, compared with controls, and discovered unique geometric distribution and layer location of amyloid pathology in the retina.[1,2] Researchers observed deposits of Aβ in the peripheral regions of the superior quadrant and innermost retinal layers, findings characterized as 'hot spots'.[2] In the future, such high-resolution retinal imaging could be used to identify those at risk for the disease and monitor those who already have a confirmed diagnosis.

Clinical Significance

"On the whole, I am quite encouraged," says Peter Snyder, PhD, professor of neurology and surgery at Brown University's Alpert Medical School. It's still an evolving field, and little consensus exists on the best acquisition techniques and metrics, he says. "But, even if this specific technique is not exactly what we want, in terms of a widely available and easy-to-use screening approach, this shows us that we are on the right track biologically."

Not everyone agrees with an Aβ-centric approach. "This publication is an example of analytics—using the retina as a 'potential surrogate marker' of AD," says Stuart Richer, OD, PhD, president of the Ocular Wellness and Nutrition Society. "However, tau protein accumulation is likely more important than amyloid, as amyloid is not sufficient itself to bring about AD." In some cases, centenarians have abundant Aβ on autopsy without documented loss of cognitive function, he says.

The real power of such technologies, he says, is in the future of predictive analytics. "This will involve combining structural data, genetics and environmental information encompassing lifestyle and nutritional factors." He adds that ODs, who examine 120 million people per year, are key stakeholders in this research. "Optometrists will surely be involved in this emerging worthwhile quest, hopefully by providing counseling, with the goal of educating patients concerning lifestyle choices and behaviors to protect both their eyes and brain."

1. Businesswire. Clinical study shows that retinal imaging may detect signs of Alzheimer's disease. www.businesswire.com/news/home/20170822005468/en/Clinical-Study-Shows-Retinal-Imaging-Detect-Signs. Accessed September 7, 2017.
2. Koronyo Y, Biggs D, Barron E, et al. Retinal amyloid pathology and proof-of-concept imaging trial in Alzheimer's disease. JCI Insight. 2017;93621:1-29.

Apps Detect Disease, Injury Through the Eyes

Researchers are turning to the eyes to better screen for a number of systemic conditions such as concussion and pancreatic, gall bladder and liver dysfunction—with apps.

"Eye doctors are gatekeepers and look to the eye to tell us more about the body," says Carlo Pelino, OD, assistant professor at PCO at Salus University. While ODs usually catch systemic concerns with dilation, these apps capitalize on ocular effects to the anterior segment.

The apps currently use a box to control light exposure, but the goal is to launch them without the need for accessories.

Cancer Screening

An app called BiliScreen uses a photo to detect increased levels of bilirubin in the sclera. When used with a 3D-printed box that controls light exposure, the app successfully identified cases of concern 89.7% of the time, according to the preliminary study.[1] Its developers hope the app may one day help improve screening methods for patients at risk for systemic diseases associated with increased bilirubin levels, such as pancreatic cancer.

"Sometimes it can be difficult to detect pancreatic, gall bladder

(continued on page 9)

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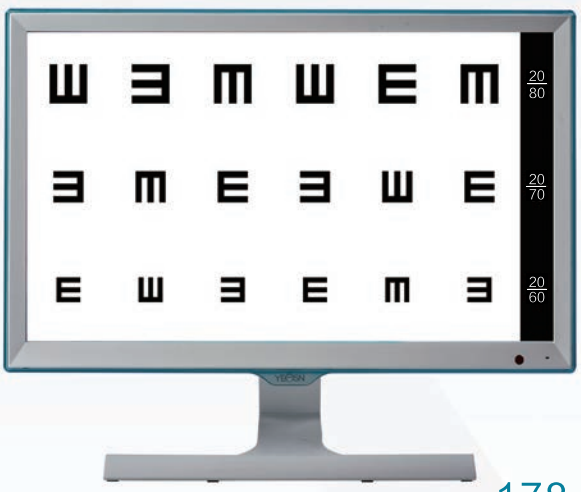

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FDA to Rein in Stem Cell Clinics

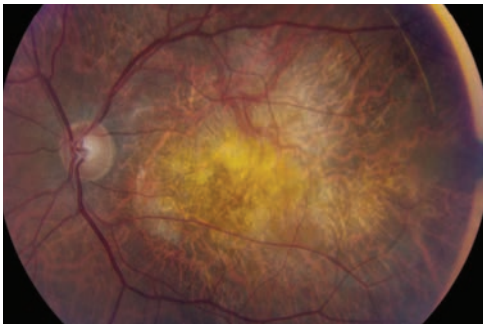

Photo: Mark T. Dunbar

The FDA's recent stance on stem cell treatment practices is aimed at protecting AMD patients seeking novel treatments.

In the wake of several reported incidents of stem cell treatments gone wrong, Food and Drug Administration (FDA) Commissioner Scott Gottlieb, MD, announced August 28, 2017, that the agency is increasing oversight of companies involved in regenerative medicine—an evolving industry often under no obligation to adhere to pharmaceutical regulations.[1] While the FDA regulates medical products, many emerging therapies hover between what is regulated and a therapy individualized by a surgeon in such a way that it is no longer subject to FDA regulation.[1]

"There are a small number of unscrupulous actors who have seized on the clinical promise of regenerative medicine, while exploiting the uncertainty, in order to make deceptive, and sometimes corrupt, assurances to patients based on unproven and, in some cases, dangerously dubious products," Dr. Gottlieb said in a press release.[1]

To combat this problem, the agency plans to revamp the regulations and ensure they are properly defined, as well as better enforce them to stop clinics from abusing cell-based regenerative medicine and risking patients' health.

After the agency investigated stem cell clinics in Florida and California, Dr. Gottleib "directed the FDA to launch a new working group to pursue unscrupulous clinics through whatever legally enforceable means are necessary to protect the public health."[1-3]

The agency also plans to more clearly define what is and is not regulated and provide manufacturers with a better framework for understanding if approval is necessary for each new product.

Lastly, the FDA will provide a less burdensome path to approval for responsible product developers, which includes individual providers using their own products for regenerative medicine procedures.[1]

"We can't let a small number of unscrupulous actors poison the well for the good science that holds the promise of changing the contours of human illness and altering the trajectory of medicine and science," Dr. Gottlieb emphasized.[1]

The FDA plans to release documents specifically outlining which new regenerative medical treatments and products are subject to FDA regulation, as well as details on an expedited path for legitimate companies applying for market approval, according to Dr. Gottlieb.[2]

1. US Food and Drug Administration. Statement from FDA Commissioner Scott Gottlieb, MD, on the FDA's new policy steps and enforcement efforts to ensure proper oversight of stem cell therapies and regenerative medicine. August 28, 2017. www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm573443.htm. Accessed September 6, 2017.
2. Grady D. Patients lose sight after stem cells are injected into their eyes. NYTimes. March 15, 2017. www.nytimes.com/2017/03/15/health/eyes-stem-cells-injections.html. Accessed September 6, 2017.
3. Kaplan S, Grady D. F.D.A. cracks down on 'unscrupulous' stem cell clinics. NYTimes. August 28, 2017. www.nytimes.com/2017/08/28/health/fda-stem-cell.html?smid=fb-nytimes&smtyp=cur. Accessed September 6, 2017.

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Screening Apps

(continued from page 6)

and liver problems until the jaundice of the skin and sclera becomes prominent, which is often too late," says Dr. Pelino. "But if we can pick that up earlier, that's huge. That's an extremely useful tool."

Currently, bilirubin levels are monitored with a blood test, which can be invasive and inconvenient. The researchers hope the app's algorithms can detect changes before they are visible to the naked eye, possibly leading to earlier diagnoses and initiation of crucial treatment.

The next step is to test the app on a much larger scale and iron out usability issues such as removing the need for accessories, according to the researchers.

Identifying Concussion

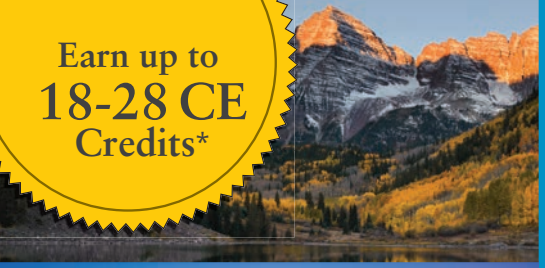
An app known as PupilScreen may help sports professionals quickly identify concussion symptoms on the sidelines.[2] The new app is designed to detect changes in pupillary response to light using a smartphone's video camera and deep learning tools.[2] Because traumatic brain injury (TBI) can negatively affect pupillary response, the researchers hope the app will function as an objective screening tool for concussion, mainly for medical personnel on the sidelines.[2,3]

Also using a 3D-printed box, the app uses the smartphone's camera and flash to stimulate the patient's eyes and record a three-second video. The algorithm assesses a patient's pupillary light reflex, according to the researchers.[2] During a pilot study of 48 patients, clinicians identified all six patients within the group who had suffered a TBI.[2]

"We need something like this to detect concussion quickly, so coaches know a player can't go back onto the field," says Dr. Pelino. "Several tools exist, such as the King-Devick test, but this is another piece of the puzzle. We need as many tools as possible on the field to help protect players from the long-term effects of continued injury."

Dr. Pelino notes the app must be accepted by the neurology community. In addition, he hopes medical personnel are properly trained to use the app and refer to neurology and an eye care professional after positive results. ■

1. Mariakakis A, Banks MA, Phillipi L, et al. BiliScreen: Smartphone-based scleral jaundice monitoring for liver and pancreatic disorders. PACM Interact Mob Wearable Ubiquitous Technol. 2017;1(2):Article 20.
2. Mariakakis A, Baudin J, Whitmire E, et al. PupilScreen: using smartphones to assess traumatic brain injury. PACM Interact Mob Wearable Ubiquitous Technol. 2017. https://ubicomplab.cs.washington.edu/pdfs/pupilscreen.pdf. Accessed September 7, 2017.



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- **CONTAINS 2 KNOWN MOISTURIZERS**[3]
  - Glycerin and propylene glycol

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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.

References: 1. Rajpal RK, Fong R, Comstock TL. Loteprednol etabonate ophthalmic gel 0.5% following cataract surgery: integrated analysis of two clinical studies. *Adv Ther.* 2013;30:907-923. **2.** Coffey MJ, Decory HH, Lane SS. Development of non-settling gel formulation of 0.5% loteprednol etabonate for anti-inflammatory use as an ophthalmic drop. *Clin Ophthalmol.* 2013;7:299-312. **3.** LOTEMAX GEL [package insert]. Tampa, FL: Bausch & Lomb Incorporated. **4.** Apt L, Henrick A, Silverman LM. Patient compliance with use of topical ophthalmic corticosteroid suspensions. *Am J Ophthalmol.* 1979;87(2):210-214. **5.** LOTEMAX SUSPENSION [package insert]. Tampa, FL: Bausch & Lomb Incorporated.

* Fingertip Formulary data 2017

LOTEMAX® GEL
loteprednol etabonate ophthalmic gel 0.5%

BAUSCH + LOMB

Visit www.LOTEMAXGEL.com

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

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, a division of Valeant Pharmaceuticals North America LLC
Bridgewater, NJ 08807 USA

US Patent No. 5,800,807

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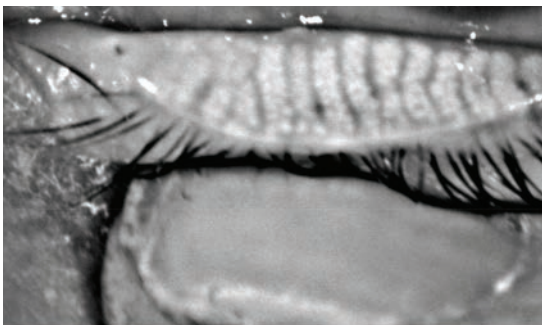

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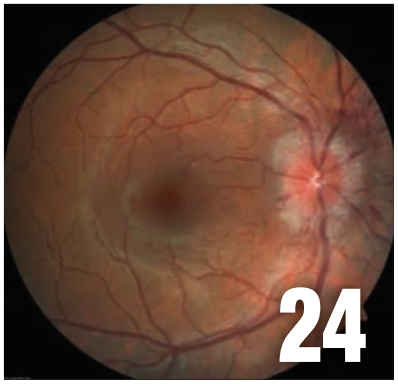

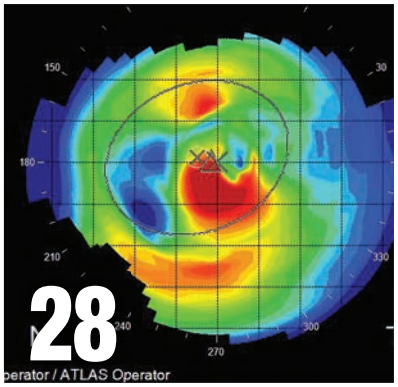

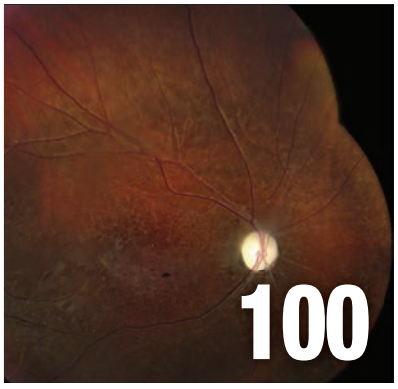

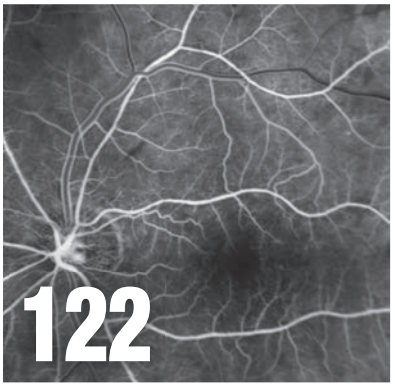

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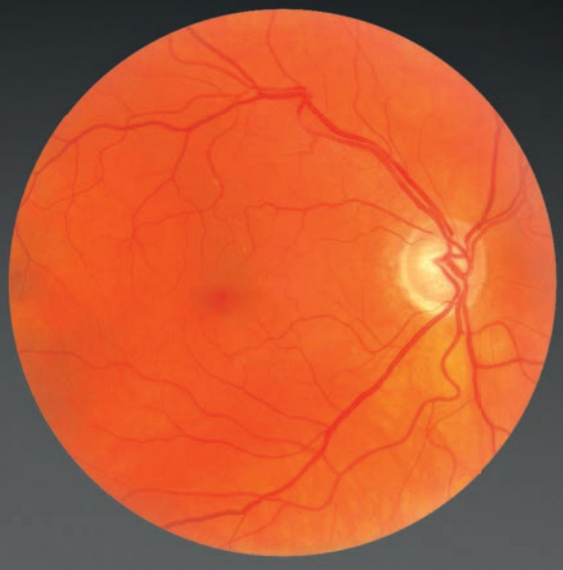

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Outlook

By Jack Persico, Editor-in-Chief

Status Update

Developments in neurology could radically change the daily life of the optometrist.

As a college student, Mark Zuckerberg likely never imagined that the simple website he set up to send messages to his classmates would have a global impact on our social interactions and, if the speculation about electoral shenanigans is true, political ones as well. He now finds himself swimming in uncharted waters. Such is the chaotic, ever-evolving life of an innovator. Optometry parallels the tech world in that regard. The profession has been both the source and subject of radical changes wrought by innovation, not all of them welcome.

A few years ago, when studies began to show that Alzheimer's disease could be detected by an eye exam, optometrists were intrigued, excited and apprehensive. Did this mean they might one day be responsible for detecting this insidious disease? The prospect of bearing that burden gave many ODs pause.

Will anxious family members of your elderly patients be looking to you for an early diagnosis that could be life-changing? In a word: maybe.

As we report in this month's news section (*see page 6*), the research and clinical tools to screen for Alzheimer's through the eye are developing with alacrity. A recent study found that fundus autofluorescence scans can pick up amyloid plaques in the retina that could be a surrogate of corresponding plaques in the brain. Other research efforts have linked Parkinson's disease to ocular changes as well. Should these correlations be validated by larger studies, optometrists will soon find themselves in the neurology business, like it or not.

It's important to recognize that the optic nerve is the "front of the brain," says American Academy of Optometry President Joseph Shovlin, OD. "What we image there is going to give us valuable information—at least someday—about how the brain is doing." Some feel it already does, Dr. Shovlin notes, as OCT scans can already detect multiple sclerosis. "In addition, keep in mind all primary retinal disease has secondary optic nerve insult, and the converse is true: all primary optic nerve disease has secondary retinal findings." Efforts to untangle the connections among the retina, optic nerve and central nervous system are at the forefront of today's ophthalmic research.

Those of you attending the Academy meeting will have the chance to hear about such cutting-edge work from Robert Sergott, MD, director of the neuro-ophthalmology service at Wills Eye Hospital in the meeting's plenary session. It should prove both fascinating and a bit intimidating.

Either way, expect a glimpse of optometry's future. Alzheimer's "is becoming so prevalent that it can't be ignored," Dr. Shovlin says. "Fortunately for us, the information gleaned is easily accessed through the eye." That easy accessibility seems destined to give ODs an important new responsibility, but also the peace of mind to take it on. "No other part of the body can be easily examined like the eye without certain advanced, even invasive techniques," he says.

Facebook quickly outgrew its original mandate. Optometry continually does the same. As Mr. Zuckerberg would say, time for a status update. ■

Through **My Eyes**

By Paul M. Karpecki, OD, Chief Clinical Editor

Flex Your Core Muscles

Although the medical model is critical to the profession, we shouldn't forget our elemental knowledge of vision testing—it's a real life-saver for patients.

Recently, I saw a patient who was on three topical anti-inflammatory medications (lifitegrast, cyclosporine and an ester-based steroid), two oral treatments (antibiotic and omega fatty acid), an artificial tear and gel, lid scrubs, a Bruder mask and he had Lipiflow done two months ago. He also walked into the lane carrying a small plastic bag that contained a set of scleral lenses. Talk about a shotgun approach to therapy! This poor gentleman could get no relief despite toting around nearly every remedy his doctors could provide.

He informed me he'd had dry eye symptoms since his LASIK surgery one year ago. He was being managed by the dry eye center at the surgical practice and he didn't want them to know he was seeking a second opinion before purchasing the sclerals. He estimated he had spent over $10,000 between the surgery, procedures, treatment options and now the cost of the scleral lenses.

A Fresh Start

Where do you even start with this patient? The first test I ran was osmolarity (TearLab). It measured 291mOsmol/L and 290mOsmol/L. When it's between 280mOsmol/L and 295mOsmol/L and both eyes are within 5mOsmol/L of each other, I estimate that there is only about a 2% to 3% chance a patient has dry eye disease (DED)—provided they didn't instill drops within the last hour or the tech caused reflex tearing when taking the measurement.

Next, I expressed the meibomian glands, which were mildly turbid but had good expressibility and at least five glands yielding liquid secretion in the lower nasal to central lower lid. Meibography showed minor truncation of about three glands in each eye, and he had trace inferior corneal staining with NaFl dye.

Based on these findings, it was time to go back to the basics. New theories suggest that patients with subtle binocular fusion issues can overstimulate the trigeminal nerve, and the resultant saccadic and pursuit eye movements can, over time, cause headaches and even dry eye symptoms.[1-5]

So, I asked him about headaches, and he mentioned they had begun at about the same time as the eye dryness after LASIK. They were frontal in location and occurred as the day progressed or when using a computer. He also noticed his eyes were much dryer when using a computer than he recalled prior to LASIK, and he spent most of his eight-hour workday on digital devices. He said he had worn glasses for about 4.50D of myopia prior to surgery.

Next, a cover test showed exophoria at distance and slight left hypertropia. Von Graefe confirmed the findings, including convergence insufficiency.

I prescribed prism to a new pair of glasses, and his headaches went away almost immediately, as did his dry eye symptoms. He also set up an appointment with the local vision therapy expert and has reported no further headaches, eye pain or dryness.

Optometry at its Core

What does this case tell us? While advances in medical eye care and new diagnostic technologies were crucial, the most important step was remembering the core strengths that set optometrists apart from other professions; in this case, looking for and measuring subtle eye misalignment issues changed the diagnosis—and the patient's life.

Many articles in this month's issue get back to the basics of optometry, including testing for accommodative and convergence disorders, when to use prism vs. vision therapy in amblyopia treatment, a better understanding of meibography and its application to DED management, nutrition and the eye and, harkening back to the example above, differentiating diseases that mimic dry eye.

It's important that we advance our knowledge in medical eye care, invest in new diagnostic technologies and focus on patient education, but never lose sight of the core strengths we have in this profession—that's what truly differentiates us. ■

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Gettin' Old Has Its Perks

Dreams do come true if you work hard enough—or, in my case, wait long enough.

By Montgomery Vickers, OD

My whole career I have felt like my true calling was to become the "old guy in the clinic." I don't know of any optometrist who made it through school without having a dozen stories about the old guy in the clinic. My classmates (PCO 1979) would love to spend a couple hours regaling you with tales about Dr. Hooten, or "Rybie," and many, many others. We loved, and still love, those fine gentlemen who, through experience, knew more than anyone about the most important thing in optometry—the patients.

The funny thing is, they were probably younger than I am right now when I was calling them the "old guys."

Here's a good one: After a particularly disastrous outcome on a test, Rybie called me into his office and asked, "Vickers, what are you going to do when you leave PCO?" My proud answer? "Be an eye doctor in West Virginia." He looked right at me and said, "No, really." Maybe I needed a little less Monty Python and a little more studying?

But here I am, carrying forth the old guy tradition as the "old junior associate in the practice." Cool.

Take the Good with the Bad

There are pros and cons to being the old guy in the clinic:

Pro: The younger doctors ask you for your advice on complex cases.

Con: You have to Google corneal hysteresis.

Pro: Staff think you're wonderful.

Con: That's why they ask, "Do you feel OK?" everyday. Right?

Pro: You can handle the angriest patients with the greatest of ease.

Con: They schedule you all the angry patients.

Pro: You have a great work ethic.

Con: They want you to work every Saturday.

Pro: You look awesome all dressed up in your doctor clothes.

Con: They want you to wear a fluorescent golf shirt.

Pro: Staff understand when you take four bathroom breaks a day.

Con: Each break is 15 minutes.

Pro: You get to work with young, energetic people.

Con: You have to work with young, energetic people.

Pro: You can help young partners make good equipment choices.

Con: You just assume they want a Shiotz tonometer.

Pro: You have *in vivo* experience with multifocal contact lenses.

Con: You can't drive in them either.

Pro: Your children don't want you to work so hard.

Con: Your children don't want to pay your bills either.

Pro: You confidently refer cataract patients to the best surgeons.

Con: While you're at it, you also refer yourself.

Pro: You enjoy a long, productive marriage.

Con: You kind of thought she'd catch on and hit the trail by now.

Pro: You can prescribe the most advanced meds to help patients.

Con: You still prescribe chloramphenicol and pilocarpine.

Pro: You have time to attend advanced CE courses.

Con: What the hell are they talking about?

Pro: You remember a lifetime of ophthalmic information.

Con: You forget to zip your fly.

I love being the old guy in the clinic, and I plan to stay at it. Remember, my wife never caught on. The young practice owners probably won't either! ■

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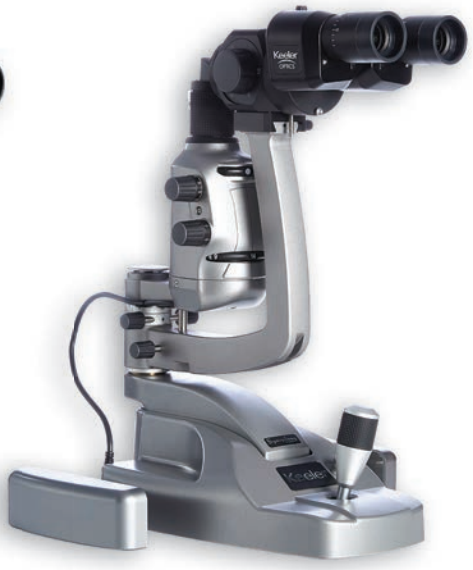

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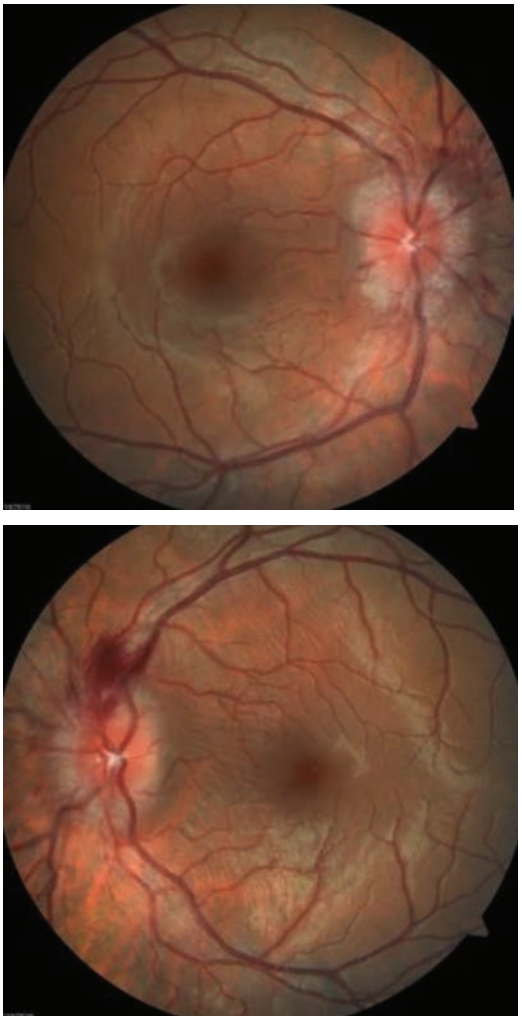
A Nerve-racking Experience

The need to distinguish true papilledema from a benign anomaly puts our diagnostic skills to the test—while the clock is ticking. **By Paige Thompson, OD, and Richard Mangan, OD**

Imagine this scenario: A new patient presents to your office with a chief complaint of headaches. She reports they occur multiple times per week and occasionally wake her from sleep. You perform a comprehensive ocular health examination and a dilated fundus exam reveals the findings pictured.

This patient portrays the telltale signs of papilledema. This neuro-ophthalmic condition is characterized by optic disc edema in the presence of increased intracranial pressure (ICP), which occurs secondary to a disruption in the balance of blood, cerebrospinal fluid (CSF) and brain tissue in the cranium. An increase in volume in any of these cranial components can raise ICP. This can subsequently cause a disruption of prelaminar axoplasmic flow into the optic nerves and result in optic disc edema.[1]

This article reviews the differential diagnosis, necessary work-up and treatment options for cases of papilledema



The optic disc edema seen in these fundus images is a telltale sign of the neuro-ophthlamic condition papilledema.

Presentation

Papilledema may be accompanied by visual and neurologic symptoms. One of the most commonly reported symptoms among papilledema patients is headache. These headaches are typically variable and may be severe enough to wake the patient from sleep.[1] In addition to headaches, papilledema patients may report symptoms of nausea and vomiting. Increased ICP can cause blurred vision and transient visual disturbances, which worsen with postural change.[2] Patients may also report a pulsatile tinnitus or hearing a "whooshing" sound, specifically when lying down.[2] Furthermore, papilledema may cause diplopia, which is often due to compression and subsequent paresis of the abducens nerve. Often, however, papilledema patients present for evaluation without any symptoms at all.[3]

Ophthalmoscopy

On dilated examination, patients with increased ICP will commonly present with optic disc edema. This is usually a bilateral and symmetric finding; however, asymmetric or unilateral optic disc edema occurs in roughly 10% of papilledema cases.[1] Optic disc edema can be graded based upon its severity, which includes factors such as extent of clock-hour involvement and vessel obscuration by the edematous nerve fiber layer.

Patients with disc edema secondary to increased intracranial pressure will not exhibit a spontaneous venous pulsation. While roughly 10% of the healthy population does not have a spontaneous venous pulsation, the presence of one is an important finding and eliminates papilledema from the differential diagnosis.[3] Additional clinical findings with papilledema may include a visual field defect, most commonly in the form of an enlarged blind spot.[3] These patients may also report photophobia or eye pain.[3]

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- DuraSite® increases ocular surface retention time, resulting in increased bromfenac absorption[2-5]
- Provides 24-hour coverage with BID dosing[1]
- Available in 5 mL bottle

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

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.

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.

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite®, 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®. 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®, 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 Effects:** 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. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite®, and should be closely monitored for corneal health. 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:** 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.

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.** 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 March 2, 2017. **4.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66. **5.** 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.

SUN OPHTHALMICS

BromSite® (bromfenac ophthalmic solution) 0.075%
Brief Summary

INDICATIONS AND USAGE
BromSite® (bromfenac ophthalmic solution) 0.075% is indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

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
The following serious adverse reactions are described elsewhere in the Brief Summary:
- Slow or Delayed Healing
- Potential for Cross-Sensitivity
- Increased Bleeding Time of Ocular Tissue
- Keratitis and Corneal Reactions
- Contact Lens Wear

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).

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SUN OPHTHALMICS

It's the clinician's job to differentiate true optic disc edema from optic disc anomalies, such as tilted disc syndrome and optic disc drusen, to avoid unnecessary costly work-up. This can be accomplished using diagnostic tools, including optical coherence tomography and fluorescein angiography, to rule out true swelling of the optic nerve. Also, A- and B-scan ultrasonography can be used to distinguish optic disc edema from optic disc anomalies. More specifically, a 30-degree test can be performed in which the optic nerve width is measured in primary gaze and in lateral gaze. If the optic nerve width is reduced by more than 10% with 30 degrees of lateral gaze, it is indicative of true optic disc edema.[4] The lateral gaze position causes stretching of the optic nerve and increases the distribution area for the fluid surrounding the nerve, which results in a decrease in optic nerve width.[4]

Diagnosis

Bilateral optic disc edema should always be treated as an emergent situation. Clinicians must rule out urgent causes of increased ICP, including mass and hemorrhage. Comprehensive work-up should begin with same-day magnetic resonance imaging (MRI) of the brain and orbits, with and without contrast material. If MRI is not readily available, then a computer tomography (CT) scan with contrast dye should be obtained.

If MRI findings are normal, one should consider a magnetic resonance venography (MRV) to rule out venous sinus thrombosis. With unremarkable findings, a lumbar puncture should be the next step in diagnosis.[3,5] This should always be performed after an MRI or CT scan, due to the risk of herniation, specifically with Chiari I malformation.[4] A lumbar puncture is typically performed while the patient is lying on their side.[2] An opening pressure of greater than 250mm CSF in adults is diagnostic for increased ICP. Additionally, the CSF contents should be examined to rule out infectious or inflammatory causes of disc edema.[3]

If neuroimaging and lumbar puncture results are normal, pursue a laboratory work-up to rule out other potential causes of bilateral optic disc edema. This laboratory evaluation should include a complete blood count with differential, platelet count, HbA1C, ESR, CRP, ANA, RF, ACE, Lyme titer, serum folate and serum B12.[6] Also, measure blood pressure in all cases of bilateral disc edema, to exclude malignant hypertension as the underlying cause.[3]

If increased ICP is noted without any other abnormalities following comprehensive investigation, a diagnosis of idiopathic intracranial hypertension can be made. Be aware that several drugs are associated with idiopathic intracranial hypertension, including tetracyclines, oral contraceptives and vitamin A analogs.[2]

Treatment Options

Papilledema should be managed by treating the primary source of the increase in ICP. If the elevation in pressure is found to be idiopathic, or is not fully resolved with treatment of the causative mass or vascular abnormality, additional management options should be considered. First, in the case of idiopathic intracranial hypertension, or pseudotumor cerebri, weight loss should be an initial therapeutic consideration. Research shows a 6% reduction in body weight may contribute to resolution of papilledema. Additionally, acetazolamide, an oral carbonic anhydrase inhibitor, may be used to treat increased ICP. Standard dosage of acetazolamide in the treatment of elevated intracranial pressure is 500mg BID, with the option of increasing to a total dosage of 4g daily, if necessary.[3]

Further, options that may be considered for patients who are intolerant or unresponsive to acetazolamide treatment include loop diuretics, serial lumbar puncture, optic nerve sheath fenestration and shunting procedures.[1,3] For headache management, topiramate may be added to the therapeutic regimen. Topiramate has been shown to induce mild weight loss, which may contribute to improvement in papilledema as well.[1]

A key responsibility in addressing these patients is to differentiate true optic disc edema from other, less emergent optic disc abnormalities. When papilledema is suspected, immediate referral should be made for neuroimaging and potential lumbar puncture, to rule out emergent causes of bilateral optic disc edema. Rapid diagnosis and evaluation is essential in cases of papilledema to save the patient's vision and, potentially, their life. ■

Dr. Thompson is a consultative optometrist at SouthEast Eye Specialists in Chattanooga, Tenn.

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2. Mollan S, Markey K, Benzimra J, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. Practical Neurology. 2014;14(6):380-90.
3. Friedman DI. Papilledema and Idiopathic Intracranial Hypertension. CONTINUUM: Lifelong Learning in Neurology. 2014;20:857-76.
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Getting the Lay of the Land

Before cataract surgery, detect problems and ensure proper calculations with corneal topography. **Edited by Paul C. Ajamian, OD**

Q I have a 64-year-old female patient with irregular astigmatism due to map-dot-fingerprint dystrophy who needs cataract surgery. Is she a candidate for a toric lens?

A Advising a cataract patient on the best intraocular lens (IOL) options depends on several factors, many of which are revealed with corneal topography, according to Steven Sorkin, OD, of Corneal Associates of New Jersey. "Corneal topography is a must-do test prior to referring patients for cataract surgery" to detect patients who may be eligible for a toric lens vs. those with irregular cylinder and various corneal conditions that would rule these options out, Dr. Sorkin says.

Toric IOLs

These lenses play a huge role in cataract correction, and higher cylinder corrections are readily available to treat one to four diopters of regular corneal astigmatism, says Dr. Sorkin. Regular astigmatism can also now be corrected, up to about two diopters, in patients who want to explore a multifocal or presbyopic lens option.

However, multifocal lenses (with or without astigmatism correction) are not good options for patients whose corneal cylinder is irregular from ocular surface disease, dystrophies or degenerations.

Distortion's Causes

Anything from contact lens overwear to Salzmann's nodular degeneration and even map-dot-fingerprint dystrophy can cause corneal distortion and irregular astigmatism—a major concern that must be addressed before cataract surgery, Dr. Sorkin explains. "Just as important, ocular surface disease can induce topographical changes, as the measurements are affected by dry spots on the cornea." Also, superficial punctate keratitis, pterygia and lid abnormalities such as chalazia can manifest as distortion on corneal topography, he adds.

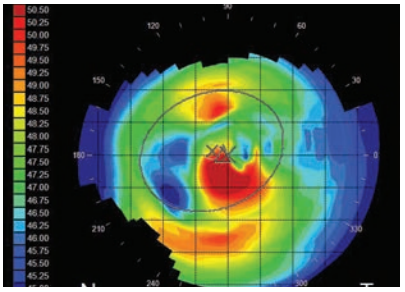

Fig. 1. Irregular astigmatism caused by map-dot-fingerprint dystrophy.

He explains that contact lenses such as thick toric soft lenses and long-term gas permeable lens wear can cause corneal warpage due to hypoxic stress. "Corneal warpage will show up on topography as irregular astigmatism and distortion," says Dr. Sorkin. Contact lens wear must be discontinued for a period of time to allow the cornea to stabilize prior to cataract surgery. "Soft lenses should be discontinued a minimum of two weeks. Gas permeable contact lenses may need to be discontinued for a month or more until keratometry and topography is stabilized over multiple visits," says Dr. Sorkin.

Optimize the Surface

Before referring the patient, it is imperative to "clean up" the lids and corneal surface, as accurate measurements must be obtained to prevent refractive surprises and suboptimal post-op acuity, Dr. Sorkin explains. Aggressive ocular surface treatment with an appropriate combination of options that may include lubrication, Restasis (cyclosporin A, Allergan) or Xiidra (lifitegrast 5%, Shire), punctal plugs, eyelid hygiene, essential fatty acid supplementation, hypochlorous lid scrubs and Lipiflow (Tearscience) "can help optimize the ocular surface prior to surgery," says Dr. Sorkin.

This Patient's Options

The 64-year-old patient in question expressed a desire to get a toric IOL for clear unaided distance vision. However, she would not be a good candidate for this lens due to her map-dot-fingerprint dystrophy. This is something best explained in advance by you, her primary care eye optometrist, according to Dr. Sorkin. The highly irregular topography would prevent accurate readings, and the outcome would be less than optimal *(Figure 1)*.

If the optometrist didn't perform corneal topography, it would be impossible to counsel the patient on the best lens choice for their particular situation. The patient might get their hopes up for a premium lens only to be turned down by the surgeon, a frustrating and embarrassing moment for all. ■

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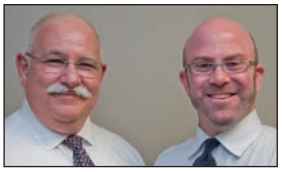


Discretion is the Better Part of Valor

A decompressive surgery patient makes strides in vision that come short of binocularity. Sometimes, good is good enough. **By Paul Harris, OD, and Marc B. Taub, OD, MS**

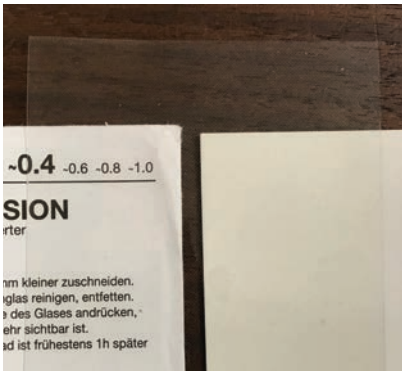
This month's column title quotes Falstaff from William Shakespeare's *Henry IV, Part I*, and it's apt for the topic at hand: to adhere to the Hippocratic oath and do no harm, sometimes practitioners must ignore a sacred cow of our training. Optometric education has drilled into us the notion that the goal is clear, single binocular vision. However, in this patient's case, meeting this goal could have caused more harm than good.

Ultimately, making the patient happy and enabling them to function again are paramount and take precedence over other clinical goals.

Self-described 'Bobble Head'

Nadine is the 70-year-old mother of a colleague. We first saw her during the holidays in 2015. She has Graves' disease and had orbital decompression of the left eye in July 2014. In rapid succession, she had three extraocular muscle surgeries, two on the left eye and one on the right, in an attempt to achieve alignment.

Balance issues. For six months prior to the decompression operation, she wore an opaque patch over the left eye in an attempt to correct the double vision, which resulted in her experiencing balance problems. She reported that if she looked quickly to the left, she experienced an increase in double vision and problems with balance. When watching her approach the examination chair, it was obvious that she moved her head and body, rather than her eyes, to see. She tipped her head regularly and described herself as a 'bobble head.' At the end of most days, she had neck and upper back pain and discomfort. In addition, she also had bilateral ptosis and was scheduled for surgery in several weeks.



Here is the Bangerter filter used to treat this patient.

Ocular motility testing was quite revealing. She was unable to raise her eyes greater than 15 degrees below the horizon. After physically lifting her upper eyelids, we discovered the limitation was in her eye movements. To look at a target and see single in what should be her primary gaze, she had to tip her head back about 10 to 15 degrees. In her current primary gaze—downward about 15 degrees to the midline—she showed a low esophoria of about the same amount at distance and near.

Non-concomitancy. When she moved her eyes 30 degrees to her left, she began to experience vertical double vision. When she moved her eyes 45 degrees to her right, she once again experienced vertical double vision. All testing was performed in downgaze because she had no upgaze.

Her near point of convergence was eight inches; her left eye would go out and she experienced double vision. Visual acuities with her current glasses at distance were 20/40 OD and 20/18 OS. At near, her visual acuities were 20/50 OD and 20/25 OS.

We chose to do a monocular refraction, which yielded the following findings:

-0.25 -1.00 x 20 20/18 OD
+0.50 -0.50 x 10 20/15 OS

Back to Mono

We could have tried a prescription that fully corrected the refractive condition, or even explored yoked prism to move objects down to the patient's field of view, allowing her to keep looking in a downward angle. The yoked prisms might have enabled her to view straight ahead without having to tilt her head back. However, her high level of non-concomitancy was greatly concerning. Had we tried the full correction in a standard multifocal (progressive or trifocal), her non-concomitancy would have made it so that she saw single vision in one and only one position of gaze. To see in other gazes she would have experienced double vision of varying degrees, regardless of whether she moved her eyes to look away from her one location for seeing single.

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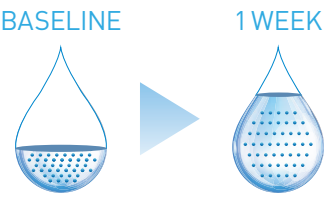

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Reference: 1. Ng L, Nguyen A, Karpecki P, Houtman D. Evaluation of Tear Osmolarity Over Time with Sustained Use of TheraTears® Lubricant Eye Drops. Poster presented at: The American Academy of Optometry Annual Meeting; November 9-12, 2016; Anaheim, CA.

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With her other alternative, had we gone the standard prescriptive route, she would have had to move her head and eyes as a unit, often involving her upper body as well, to maintain single vision. So we went down another path: monovision in glasses.

The patient's dominant right eye moved more freely over a wider range of movements compared with the left, so we assigned the right and left eyes to see at distance and near, respectively. The final Rx was:

OD -0.25 -0.75 x 20
OS +3.00 -0.50 x 10

When we put this prescription into a trial frame and gave it to the patient, a big smile spread over her face. She looked out at the distance chart and read it easily. To her husband's surprise, she looked down at the reading material in front of her and started to read out loud. They both remarked that she hadn't read anything in months and were quite happy with these new results. We suggested trying this prescription for a few weeks and following up to gauge if we needed to make any adjustments.

As often happens, we didn't see her back until a year later. On September 15, 2016, the patient underwent a second decompression operation, which was scheduled several months prior, but delayed when the patient fell in the waiting area and sustained a blowout fracture of the orbit. So, the second surgery was delayed a few months to give her time to heal.

In general, she reported improvements following surgery. She has decreased pressure and soreness in her face, but the middle branch of the facial nerve seemed to be damaged, most likely from the blowout fracture. She has no feeling in the middle part of her face on the right side and in eight upper teeth on the right side of her mouth. Unfortunately, the monovision glasses broke in the fall and she didn't have them from July until we saw her again in December. She stated that the glasses dealt with most things, but she still has some diplopia.

The world with and without the 0.4 Bangerter filter.

Reaffirmation

At this point, the patient says she's free of double vision early in the day. She notices that it begins at about 5pm each day and gets worse until bedtime. Once diplopia starts, she transforms back into a 'bobble head.'

We tried, more courageously this time, five-prism diopter yoked base up prism, which raised the area of binocular vision. While it didn't seem to be significant for her, it relieved some tension in her neck and upper back. Phorias were:

- Six eso at distance with six left infra.
- One to two eso at near, with five left infra to get alignment.

Vergences were done in the phoropter. We needed six diopters of base up prism in the left eye to do the testing at distance and got ranges of X/8/5 base out and X/-2/-3 base in, meaning her clear, single binocular vision ranged from two to eight prism diopters base out with six diopters of vertical prism. This seemed like too much work for such little gain, reaffirming that monovision in glasses might continue to be the best option. Had we put this on her, she would have had to make moment-to-moment choices to either move her eyes and see double or move her head and upper body to attempt to keep the world single. Her binocular system is simply too fragile.

We ordered monovision glasses again; this time, however, we placed a 0.4 Bangerter filter over the upper portion of the left lens, which degraded the image to help eliminate the diplopia at distance. This did the trick—she has been happily using these glasses full time.

There are cases when a practitioner feels they are giving up on fully solving the patient's visual problems. However, using discretion can help patients achieve greater well-being and become more efficient with their visual processes rather than stretching for clear, single binocular vision. Achieving the best vision possible for your patients while doing no harm is the ultimate end. ■

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Trendy Diets: Do They Put Your Patients at Risk?

Some prioritize a single goal over all others. Here's how to ensure your patients are caring for their ocular and systemic health. **By Cecelia Koetting, OD**

Your patients' food choices and dieting patterns can sometimes be overwhelming. Today, new dietary trends, such as paleo or gluten-free, and specific labeling, such as hormone-free and free range, can be confusing to patients simply looking for the healthiest diet—and to practitioners managing patient health. By understanding the nutritional makeup of food and its impact on systemic and ocular health, practitioners can better educate patients on their dietary choices and how to protect their ocular health.

Patients need to maintain the proper levels of macronutrients, vitamins and minerals through their diets.

Here is a look at what the body needs, nutritionally, and what many of today's common diets do to the body—and the eye.

Dietary Basics

Most diets can be healthy, as long as they are done properly by ensuring sufficient intake of necessary vitamins and nutrients in meals or, if needed, supplements. The three main components of a proper diet are: protein, carbohydrates and good fats. Proteins help the body maintain muscle mass, speed up metabolism and feel full longer. Carbs provide quick energy when the body breaks them down into single glucose molecules, which are then stored for later use. Lastly, fats keep you warm and provide you with essential fatty acids, which help to control inflammation and aid in blood clotting and brain development.[1] Fats also help the body absorb fat-soluble vitamins A, D, E and K.[1]

Vitamins and minerals. The United States government provides recommended daily values for all vitamins and nutrients. Each one has a job in helping our bodies and eyes grow and function properly.

From recent studies such as the AREDS I and II, we know that zeaxanthin and lutein help to reduce chronic eye disease, filter harmful blue light and act as natural macular antioxidants to help decrease progression of severe age-related macular degeneration (AMD).[2] The body uses alpha lipoic acid to prevent certain types of cell damage, restore vitamin levels and break down carbohydrates to improve insulin sensitivity.[3] Zinc is important for fetal development, wound healing, DNA synthesis and the immune system.[3] It is found in high amounts within the retina, and studies show zinc deficiency causes dark adaptation defects.[4,5] Vitamin A, which is critical to the immune

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system's function, also aids in retinal melanin production.[6] Deficiency can lead to night blindness, which is often accompanied by Bitot's spots on the conjunctiva.[6] In severe forms of deficiency, corneal ulceration can occur along with keratomalacia.[6,7,3]

Vitamin C is associated with a lower risk of cataract formation by protecting the proteins of the crystalline lens from oxidation.[8] Research also suggests it acts as an antioxidant, inhibiting retinal cell damage from free radicals liberated by UV exposure.[5] It also helps to develop connective tissue and biosynthesize collagen, both of which aid corneal wound healing.[8] Vitamin C keeps neurotransmitters functioning and, of course, boosts immune function.[8] Vitamin E also helps with immune function and protects cells from free radicals by acting as an antioxidant, which helps to delay or prevent chronic systemic and retinal diseases.[5,8] Deficiency of this vitamin has been identified as part of the pathogenesis of AMD, though supplementation of vitamin E alone was not found to decrease incidence.[2,5,8]

As with other vitamins and minerals, vitamin K bears clinical significance, as blood clotting factors are affected by vitamin K absorption, which can cause concern for retinal bleeding.[3]

Several B vitamins exist, and each plays an important role in the body and the eye:

• B1 (thiamine) helps in energy metabolism but also with the function of the central nervous system.[3] Deficiency can lead to Wernicke's encephalopathy with nystagmus, rectus muscle fatigue or paralysis, optic atrophy and loss of visual acuity.[3,9]

• B2 (riboflavin) also aids in metabolism by facilitating amino acid production and acting as a cofactor in antioxidant production.[3] Lack of riboflavin can lead to corneal epithelial dystrophy, vascularization of the cornea, angular blepharoconjunctivitis and decreased vision.[3,9]

B vitamins play a large role in maintaining ocular and systemic health.

• B6 (pyridoxine) influences red blood cell production, brain function, immune function and decreased homocysteine.[3,9] A deficiency in this vitamin can lead to optic neuritis and angular blepharoconjunctivitis.[9]

• B7 (biotin) helps to mediate cell growth, fatty acid production, fat metabolism and steady blood sugar levels.[3,9] This is found in concentrated quantities within the retina, aiding in the maintenance of retinal health.[5]

• B9 (folic acid), critical for fetal development, also combats anemia and aids in red blood cell production.[3] Low levels can lead to nutritional optic neuropathy.[5,9]

• B12 (cobalamin) is used for the essential task of myelin synthesis and repair and DNA/RNA production.[3] Research shows some diabetes patients taking metformin are at increased risk of B12 deficiency as a side effect of the medication.[10,11] Studies show abnormal B12 metabolism in patients with peripheral neuropathy, diabetic retinopathy and optic neuropathy.[4,9,10,11]

Essential fatty acids (EFAs). Multiple studies, including the Dream Study, show the importance of essential fatty acids in treatment of dry eye disease (DED), which inhibit T-lymphocyte production associated with the disease and pro-inflammatory cytokines, IL-1, IL-2 and TNF-alpha.[12,13,14] EFAs such as omega-3s, which are components of cell membranes, aid visual development and maintain retinal function, increase meibomian gland secretions, boost the immune system, decrease inflammation, maintain the nervous system's integrity and decrease cholesterol levels.[2,12,14-17] One study observed a correlation between low dietary intake of omega-3 EFAs and DED in women, and a 30% decrease in DED with each additional gram of omega-3 EFAs consumed daily.[18]

The Risks

Popular diets as well as dietary lifestyles come with both risks and benefits. Here's a breakdown of the most popular diets and dietary habits, how much (or little) of their popularity is based on myth or fact, and how to best make dietary changes without hurting ocular health:

Paleo. This diet's rather simplistic mantra is, "if a caveman couldn't eat it, neither can you."[18,19] Thus, all processed foods are avoided, including grains, legumes, added salt, dairy and alcohol. Although the diet hasn't been around long, the idea of eating clean unprocessed foods is not new. The mainstays of the diet include fresh fruits and vegetables, meats, seafood and nuts. According to one study, the right balance should be approximately 35% of calories coming from fat, 35% from

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carbohydrates and 30% from protein.[21] Maintaining this diet may help the body decrease its glycemic load through a healthy ratio of saturated to unsaturated fatty acids, an increase in vitamin and nutrient consumption, and an optimal balance of protein, fat and carbs.[20] Other studies have found the paleo diet improved blood sugar control over 12 weeks compared with a Mediterranean diet, and overall better glucose tolerance; long-term, this may reduce risk for type 2 diabetes.[19,20]

The popular paleo diet may alleviate a common issue with the American diet: pre-diabetes and diabetes. However, this dietary trend isn't without its risks.

Concerns with the diet include the risk of consuming too much protein; a 1:1 ratio of calories from meat to that of produce can lead to kidney damage.[22] Consuming too much saturated fat from red meat can also lead to heart disease.[23] Since dairy is not a part of paleo, patients on the diet could be low on calcium and vitamin D, which can lead a higher risk for osteoporosis, hypertension and diabetes.[24] Some research shows a lower risk of MS in Caucasians with high levels of vitamin D, with increasing incidence rates in correlation to distance from the equator. Therefore, researchers hypothesize that vitamin D may mediate an anti-inflammatory immune response, specifically enhanced regulatory T-cell function.[24]

Ocular involvement: Research shows a high amount of meat protein, a common concern with paleo, is linked to a higher risk of early cataract development.[25] Vitamin D deficiencies associated with this diet may also lead to an increased risk for of AMD, which is thought to result from the increased inflammation and angiogenic effects that occur in the absence of the vitamin.[26] These then contribute to early retinal vascular damage.[26] One study also suggests the impaired immunity caused by vitamin D deficiency increases risks for corneal diseases such as herpes simplex zoster and contact lens-associated acanthomoeba.[24,26]

Vegetarian/vegan. While vegetarian diets can vary significantly, their defining characteristic is abstention from the consumption of meat, including red meat, poultry, seafood and flesh of any animal. *Ovo-lacto* vegetarians eat eggs and dairy, *ovo-vegetarians* eat eggs but no dairy and *lacto-vegetarians* eat dairy but no eggs. *Vegans* abstain from eating animals or any animal byproducts (as well as refraining from use of any products made from animals, including wool and leather).

Vegetarian and vegan diets, when done properly, tend to be lower in fat and cholesterol with higher levels of fiber and antioxidants. This has been associated with a lower body mass, lower rate of death from heart disease and lower levels of cholesterol.[27] Research also suggests there is a lower rate of hypertension, type 2 diabetes mellitus and prostate and colon cancer in vegetarians.[27]

Ocular involvement: In a 2011 study, researchers found a correlation between a lower risk of cataract development and a lower consumption of meat.[28] However, as reported by the Academy of Nutrition and Dietetics, the lifestyle is, at times, also associated with low dietary levels of protein, iron, zinc, calcium, vitamin D, riboflavin, vitamin B12, vitamin A, omega-3 and iodine.[9] Such shortages may contribute to ocular issues; night blindness or corneal ulcerations arise from vitamin A deficiency, low EFA intake worsens DED and MGD and optic neuropathy can arise with vitamin B12 deficiencies, for example.[7,16,17,28]

Gluten-free. While some patients eliminate gluten from their diet due to rumors that it's the root of all modern maladies, in several cases, valid medical reasons exist for avoiding gluten: wheat allergy, non-celiac gluten sensitivity (NCGS) and celiac disease. Wheat allergy is an immune reaction to a wheat protein, mediated by immunoglobulin E (IgE) antibodies that are sent out to attack the wheat.[29] NCGS, as its name suggests, is a sensitivity to gluten, which causes intestinal and extraintestinal distress that is not IgE mediated or an autoimmune reaction.[30-32] It is currently a diagnosis made by exclusion.[31,32]

Celiac disease is an autoimmune disorder triggered by ingestion of gluten. Affected individuals must inherit the genetic predisposition, be consuming gluten and have the disease activated. Activation usually occurs secondary to stress, trauma or surgery, and sometimes viral infections.[33-35] When a person with activated celiac consumes gluten, the cells lining the small intestine malabsorb nutrients, which may cause deficiencies.

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Ocular involvement: Wheat is sometimes found in cosmetics and lotions, causing a wheat allergy to manifest as allergic conjunctivitis, eye irritation and eczema both of the lids and the periorbital skin.[36,37] Most commonly, malabsorption associated with celiac disease occurs with vitamin A, vitamin D and calcium, leading to retinopathy, night blindness, an increase in cataracts, DED and, occasionally, idiopathic intracranial hypertension.[8] The autoimmune disorder-related ocular effects include orbital myositis, uveitis, thyroiditis with orbitopathy and brain occipital calcifications.[33] While ocular issues associated with the disease itself may not be avoidable, making sure patients increase their dietary or supplementary vitamins A, D, B and calcium can help them avoid some of the other gluten-free complications.

Wheat, a staple of the American diet, is intolerable to some due to gluten allergy, non-autoimmune gluten sensitivity and celiac disease.

Health Mantra

When counseling a patient regarding a specialty diet, as with all things, the key word is moderation. The mantra for any of your patients on a special diet should be: read your labels and supplement any missing vital vitamins and nutrients.

Labels. Patients have to wade through many new food labels with a wide range of implications. Here is a quick look at what some of these new labels actually mean.

Cage-free indicates that the animal has never been contained in a cage, while *free range* animals have open access to food and water, with allowance to roam in certain defined spaces.[38]

USDA-labeled *organic foods* have a checklist of criteria that differentiate them from natural, including:[1]

- no toxic persistent pesticides
- no genetically modified organisms (GMOs) in the feed
- no antibiotics
- no sludge or irradiation
- meets specific animal welfare requirements (e.g., cows are required to be on pasture for pasture season)
- contains low levels of environmental pollution

All of these are closely monitored with audit trails as well as inspection certifications.

Hormone-free and claims of *no antibiotic use* are voluntary FDA labeling commonly used with milk and other dairy products to indicate that the cows were not treated with growth hormones. Three levels within these categories exist: (1) raised without antibiotics, (2) not fed antibiotics and (3) no detectable antibiotic residue.

GMO is a voluntary FDA label that indicates the organism (plant, animal or microorganism) has had genetic material altered in a way that did not happen naturally through mating or natural recombination. Studies show these foods can contain increased amounts of dietary vitamin A and carotenoids, which decrease the risk of certain diseases, including retinal degenerations.[39]

Supplementation shortcomings. The body always absorbs nutrients and vitamins more readily from food, so the best source is the diet. Foods lose nutrient and vitamin content during processing, storage and milling, so fresh is better.[40] While a deficiency of vitamins and minerals exposes the individual to potential systemic and ocular problems, the opposite can be true as well. For instance, excessive amounts of vitamin A can cause increased cerebrospinal fluid pressure, papilledema, double vision and dry mucous membranes.[1,3,7] While taking excessive amounts of water-soluble nutrients may cause some uncomfortable side effects, such as nausea and diarrhea with vitamin C, they most likely pose little ill effects. On the other hand, fat-soluble vitamins—A,D,E and K—pose real risks for toxicity.[41] Therefore, instruct patients to adhere to the proper dosages listed on their supplements, assuming their supplements are up to par with the standards for quality and efficacy.

Additionally, while dietary supplements can help make up for what's lacking in the diet, the body's absorption of these items varies based on composition. And, while all supplements contain labels, the actual contents vary.

To ensure patients are getting the best value, clinicians should recommend specific supplements by looking for independent certification labels from the Natural Products Association (NPA), US Pharmacopeial Convention (USP) and NSF. Independent chemical analysis websites such as labdoor.com and consumerlab.com break down the contents of many brands to assess the accuracy of labels, product

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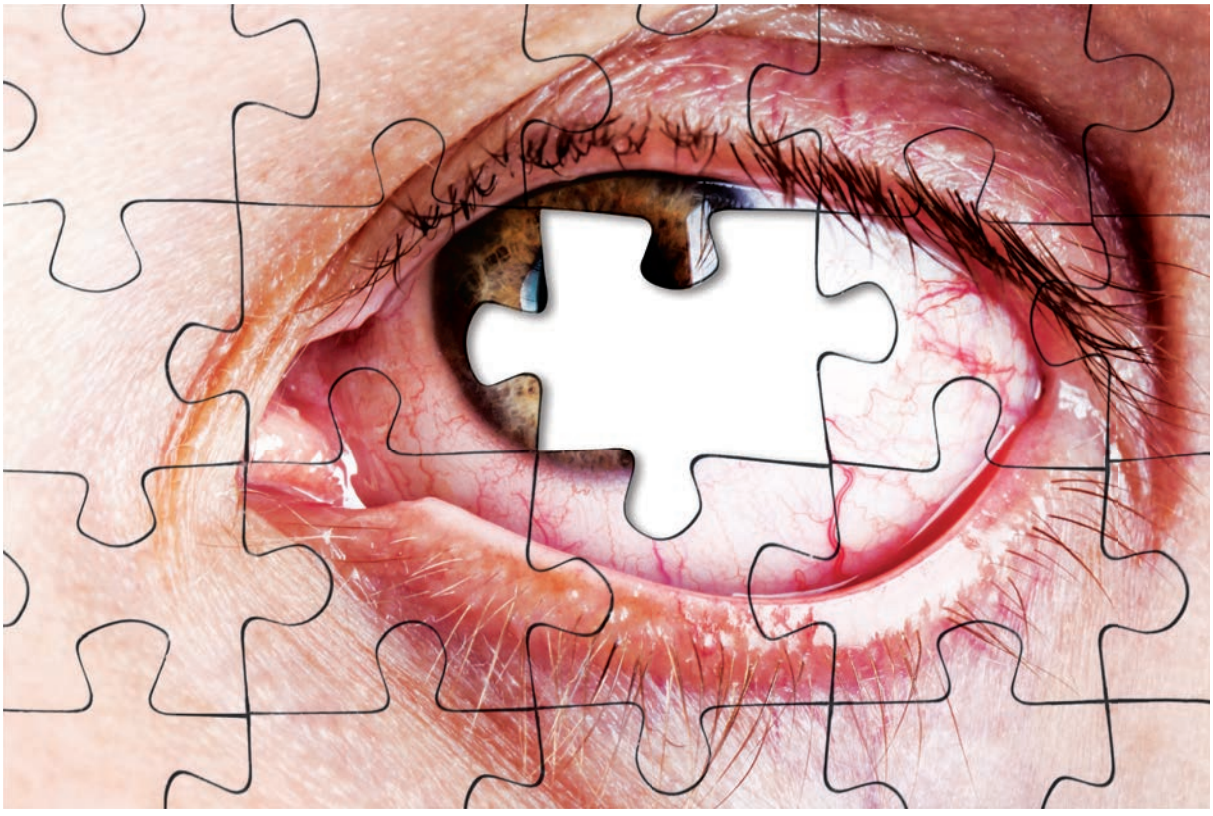

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purity, ingredient safety and potential efficacy. For example, labdoor.com found the average tested values of vitamin label claims were off by 22.6% and minerals by 29.2%. The most overstated amounts were vitamins A and C. Gummy and other chewable multivitamins had 54% less vitamin content and 70% less minerals than their standard counterparts.[42]

For EFA supplements in particular, the key is the level of docosahexaenoic acid (DHA) and its precursor, eicosapentaenoic (EPA). AREDS2 used the dosage of 350mg DHA and 650mg EPA per day, while the Dream Study used 1,000mg DHA 2000mg EPA per day.[14] Gamma-Linolenic acid, a healthy omega-6, shows benefit in dry eye disease, including in Sjögren's syndrome.[43]

Although no consensus on dosage exists, most studies support 1,000mg of DHA and 1,000mg of EPA daily as adequate to support a healthy ocular system.[16,17,44]

Krill oil is another alternative for omega-3 supplementation. It is more concentrated and a smaller pill size, which is often more palatable for patients. A recent study shows that after three months of supplementation with either krill oil at a dosage of 945mg EPA and 510mg DHA per day or fish oil at 1,000mg EPA and 500mg DHA, study participants had improved tear osmolarity and increased tear stability compared with placebo.[45]

Popular diets and dietary habits will come and go, but the body's nutritional requirements will not change. When clinicians look out for patients' dietary deficiencies and educate on moderation, we help ensure their ocular health. ■

Dr. Koetting is the referral optometric care & externship program coordinator at Virginia Eye Consultants in Norfolk, VA. She is a fellow of the American Academy of Optometry and a trustee of the Virginia Optometric Association.

Though chewable supplements improve compliance, studies show they contain lower levels of vitamins and minerals.

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When Dry Eye Disease is a Secondary Condition

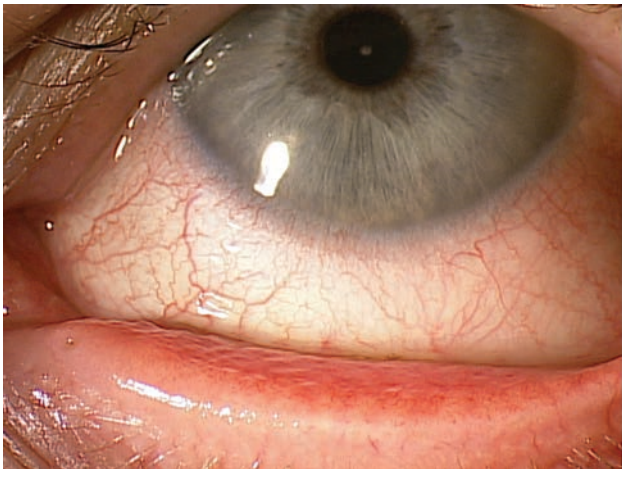
Identify the source of ocular surface dryness by considering these common instigators. **By Paul Karpecki, OD, and Jeff Krall, OD**

As far as dry eye disease (DED) treatment goes, the times are a-changing. Not long ago, an optometrist would have said a patient with no symptoms of DED had no DED. But as research continues to show that DED signs and symptoms don't necessarily correlate, clinicians must turn to other markers for a diagnosis.[1,2]

Here, we examine several of the underlying conditions that could be causing patients' itchy, stinging, dry eyes.

Case Example

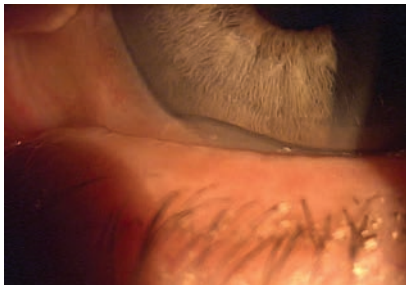
A patient presents reporting symptoms of dryness, such as grittiness and irritated red eyes, that typically occur late in the day or after computer use. That seems like a pretty straightforward diagnosis for DED, especially since meibomian gland expression shows mild turbid secretions. The patient also displays mild inferior corneal staining. He was previously placed on topical anti-inflammatory medications and a lipid-based tear. After three months, omega fatty acids were added and, at six months, punctal plugs. In frustration, the patient discontinued all medications and scheduled an appointment. Upon examination, the patient mentioned the exact same symptoms: dry, gritty, red and irritated eyes late in the day and especially while on the computer. At this examination, he was tested for osmolarity with the TearLab Osmometer (TearLab) and measured 293 OD and 291 OS. The expression was similar, as was inferior corneal staining. There was no lissamine green dye staining of the conjunctiva, and the tear meniscus height was normal. Noninvasive tear break-up time (TBUT) took longer than 10 seconds.



This patient's red, irritated eye is a classic sign of allergic conjunctivitis.

We relied on the combination of these more advanced tests and point-of-care diagnostics and were quickly able to see that this was not DED at all. Cover testing revealed exophoria at distance and convergence insufficiency at near. The patient also had a positive tight lid seal test in both eyes, indicating that his eyelids do not completely seal, especially at night.

He was told to discontinue using a ceiling fan at night and to use lid hygiene products and a hydrating compress for mild blepharitis. He was given the option of vision therapy and prescribed a new pair of progressive lenses with base in prism for his exophoria/convergence insufficiency. Within a week, the patient's symptoms were gone and he was able to work on the



Conjunctivochalasis, seen here, can look like or, in some cases, lead to, dry eye.

computer with comfort; he even commented that, not only did his dry eye symptoms disappear, but he rarely experiences headaches anymore.

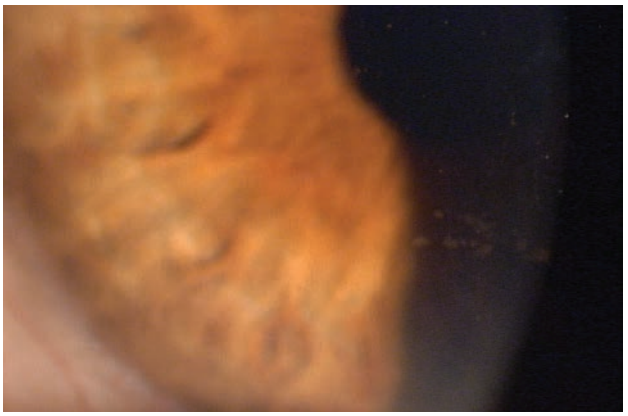
This case shows the importance of using advanced, sensitive testing and realizing there are many other conditions that have symptoms similar to dry eye that are not dry eye.

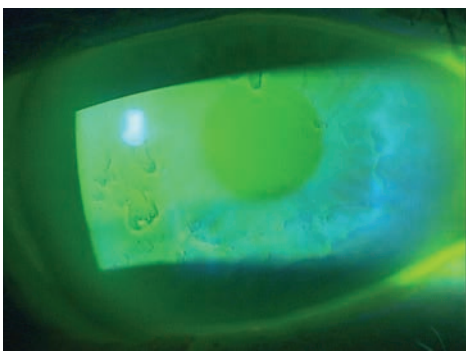
Conjunctivochalasis

This condition can eventually lead to DED, but in most stages it is unrelated and typically causes symptoms of grittiness, foreign body sensation, epiphora and red, irritated eyes. Conjunctivochalasis (CCH) usually tests positive for corneal and conjunctival staining and a rapid TBUT, but in mild-to-moderate cases, osmolarity is normal while MMP-9 testing can be elevated.[3-5]

The presence of more frequent subconjunctival hemorrhages may point to CCH, but the overlap with DED represents yet another reason to initiate advanced OSD testing.[6] The symptoms are quite similar to dry eye; however, in CCH the hallmark symptom is pain or foreign body sensation in a localized area. Patients have the ability to pinpoint it, like when they have an eyelash in their eye. Most commonly, they feel it when they blink or with eye movement.[7] In fact, a clinician can reproduce pain with local pressure by pressing on the lids over the area of conjunctival chalasis while the patient looks in the opposite direction. This condition is sometimes confused with DED. Further complicating matters, many CCH patients experience epiphora secondary to the redundant conjunctival tissue, which prevents tear flow to the punctum. Also, DED can worsen in the presence of CCH, making the distinction more difficult.[8] This is important because in advanced cases of CCH there is likely a comorbidity of DED. Advanced cases of CCH also can result in exposure problems.[8] Another characteristic of conjunctivochalasis is that almost all cases occur in the temporal and sometimes central conjunctiva.[7]

The actual condition is characterized by loose or redundant conjunctival tissue and may be associated with absence or stretching of subconjunctival tenons fascia. Histology slides of CCH show the presence of





Gritty, dry eyes is a symptom of epithelial basement membrane dystrophy, a common anterior segment issue.

lymphangiectasia. The chronic prolonged mechanical obstruction of lymphatic flow may result in lymphatic dilation and eventually give rise to CCH. Inflammation plays an important role in the pathogenesis of CCH, and pooling of inflammatory cytokines in tears of patients with chalasis has been shown to induce distinct adverse effects that compromise the patient's ocular surface health.[5]

Risk factors for CCH include age (older than 50), a history of DED, prior ocular surgery or a history of conjunctival chemosis. Conditions that may cause conjunctival chemosis include long-standing allergic conjunctivitis, dry eye, trauma or inflammatory conditions such as episcleritis.[9]

Research also suggests an association between conjunctivochalasis and immune thyroid disease.[10] A prospective study found that the prevalence of CCH in patients with autoimmune thyroid eye diseases was as high as 88%.[10] It is imperative for optometrists to rule out an association with thyroid disease and to have systemic management be initiated by an endocrinologist.

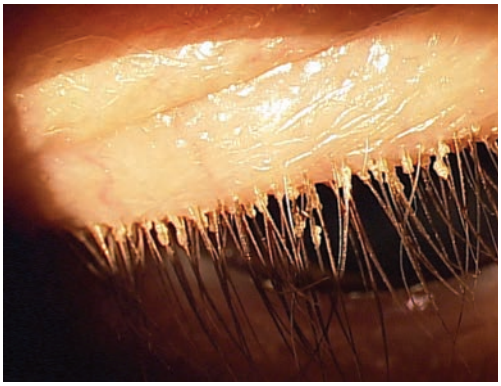
Salzmann's Nodular Degeneration

Research shows this can mimic dry eye.[11] Salzmann's nodular degeneration (SND) histopathology reveals that the epithelium over the nodules is considerably thinned, resulting in symptoms of dryness, foreign body sensation, gritty eyes and irritation.[11] The most common symptom is blurred vision.[2] SND is bilateral in 63% of cases.[12] Studies show that approximately 40% of patients with SND also have meibomian gland disease (MGD).[12,13] SND has also been linked to inflammation and even inflammatory systemic conditions, such as Crohn's disease.[13] Treatment typically involves surgical removal, as these tend to exist in the peripheral cornea.

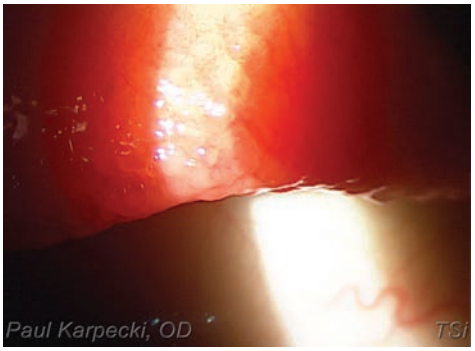
Blepharitis

Infections, including staphylococcal and *Demodex*, can cause itching, irritation, dryness and blurred vision.[14]

Eventually, this inflammation will lead to MGD, resulting in potential DED; thus, mechanical treatment will often stave off the progression caused by the biofilm.[15] But blepharitis can also exist on its own, without inducing dry eye disease in many cases—especially early cases. One poster presented at the TFOS conference in Montpellier, France in September 2016 shows the three most common conditions in patients with symptoms of DED but normal osmolarity are allergic conjunctivitis, blepharitis and epithelial basement membrane dystrophy (EBMD). Patients with blepharitis—especially as a result of *Demodex*—will complain of itchy, gritty eyes, although the itching is typically localized to the lid margin/lash area.[16]



A close examination of the lashes reveals the cause of this patient's blepharitis—*Demodex*.



This patient's irritation may have felt similar to dry eye, but MMP-9 testing shows an underlying giant papillary conjunctivitis.

Epithelial Basement Membrane Dystrophy

This is one of the most common anterior segment conditions an eye care physician will observe in clinical practice.[17] Although many patients with EBMD may be asymptomatic, patient symptoms range from blurred visual acuity (VA) and gritty, dry eyes to severe pain and recurrent corneal erosion (RCE). EBMD affects nearly 42% of individuals across all age groups, and up to 33% of patients with EBMD experience RCE during their lifetime.[18,19]

The clinical signs of EBMD typically include a bilateral presentation of epithelial microcysts and whirling superficial defects, such as corneal ridges and opacities. EBMD is not considered an inherited condition, but several reports show otherwise.[20,21] In one study, researchers identified two different point mutations in the genes associated with other corneal dystrophies.[20] EBMD is associated with a faulty basement membrane, which is thickened, multilaminar or redundant, and misdirected into the epithelium. The basal epithelial cells manufacture either unconventional redundancies or finger-like projections that protrude from an abnormally thickened basement membrane.[21]

Dryness is one of the more common symptoms of EBMD, and patients may report fluctuating vision, grittiness or photophobia.[22] A slit lamp examination can reveal the presence of microcysts, which are epithelial cells trapped in intercellular debris in the redundant basement membrane formation. These microcysts can be observed best with fluorescein staining and often appear as inverse or negative staining areas. A slit lamp examination may reveal the classic identifying maps, dots, lines and even fingerprint-type formations.

A Question of Comfort

Contact lens discomfort typically results in symptoms of dry eye disease, but dissipates when the contact lens is not worn. The cause could range from subclinical DED and MGD to issues with the contact lens itself, solutions or compliance. Daily disposable lenses and higher quality materials that retain moisture are often the best options for these patients, as well as contact lens solutions containing hyaluronic acid or other lubricating agents.

Patients should be observed for partial blinking habits and MGD and should be tested with more sensitive testing, such as noninvasive TBUT, osmolarity and lissamine green dye staining.

Giant Papillary Conjunctivitis

Other contact lens complications, such as giant papillary conjunctivitis (GPC), can result in symptoms that can

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range from decreased contact lens wearing time to foreign body sensation to irritated, dry eyes with ropy, clear discharge.[23] Making the differential even more difficult, one study shows that patients with GPC have greater levels of meibomian gland dropout in both lower eyelids and that the meibum expressed from patients with GPC is more viscous than patients without GPC.[24]

Testing for GPC typically shows normal osmolarity, but increased MMP-9 levels.[25] These patients often have a history of contact lens wear, but lack compliance, usually wearing lenses beyond the two weeks or one month time frame indicated. Everting their eyelids will show large papillae and hyperemia without corneal or bulbar conjunctival involvement.

Conjunctivitis

Even conjunctivitis itself can seem like DED. Studies show that patients with DED also suffer from symptoms such as itching and dryness, especially in the case of allergic conjunctivitis patients.[26] Differentiating these two conditions requires an astute clinician. Investigators have found that the more common oral pharmaceutical agents for allergy treatment have a significant drying effect on the ocular surface and can actually induce dry eye.[27] That dry eye can allow allergens to remain on the surface longer and may exacerbate the allergic conjunctivitis. Compounding this is the realization that symptoms of DED may be quite similar to those of allergic conjunctivitis.

In one study, clinically significant itching was found in 28.2% of allergic conjunctivitis cases, dry eye as a symptom found in 35.8% and redness in 28.2%.[26] An ideal way of making a differentiation is to look to the more classic allergic conjunctivitis clinical findings, such as conjunctival papillae, conjunctival chemosis or eyelid edema or a combination or all three.[28] A key systemic differentiator is rhinitis, which is present in more than 80% of cases of allergic conjunctivitis, but is not a symptom known to be associated with dry eye disease. Other findings found in patients with allergies include as strong family history, atopic dermatitis or the presence of asthma.[28]

Floppy Eyelid Syndrome

Many patients with this diagnosis will also develop DED; however, DED will not resolve with standard treatment unless the loose eyelids are addressed. Floppy eyelid syndrome (FES) is characterized by chronic superficial keratitis, MGD, corneal abrasions, giant papillary conjunctivitis and mucous discharge.[29] Most tellingly, when the upper eyelid is everted, affected lids are rubbery and may even flop easily or have a history of spontaneous

eversion. Because FES is directly linked to sleep apnea and obesity, test for associated systemic conditions, such as diabetes and hypertension.[30]

Management for FES typically involves treating the components of the disease with anti-inflammatory medications, lubricants and lid hygiene and referring to an oculoplastic surgeon for eyelid repair or tightening.

Limbal Stem Cell Deficiency

These patients will often complain of blurred or fluctuating vision associated with photophobia and dry, irritated eyes.[31] The typical causes include chemical burns and severe ocular scarring secondary to conditions such as Stevens-Johnson syndrome.[32]

A diagnosis of limbal stem cell deficiency (LSCD) is made if corneal epithelium damage results in a dysfunctional stem cell population. The final outcome is an inability to produce healthy epithelium. The key signs of LSCD are corneal haze, conjunctivalization, epithelial irregularity and superficial neovascularization of the cornea.[33] Typically, these signs begin with the superior cornea and then move centrally.[34] In addition to chemical burns and Stevens-Johnson, other causes of LSCD include contact lens-induced keratitis, aniridia, multiple ocular surgeries, peripheral ulcerative disorders, chronic neurotrophic keratitis, pterygium, severe microbial keratitis, chronic bullous keratopathy and radiation therapy.[33] In cases with no pathology or history of trauma, question the patient about contact lens abuse.[35]

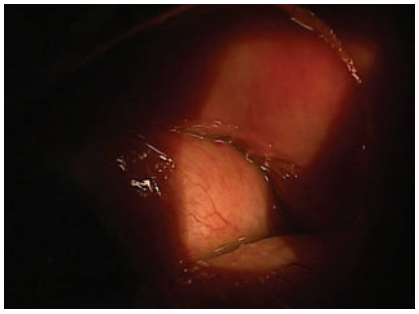
In addition to evaluating the clinical picture, a conclusive diagnosis of LSCD can be made via impression cytology. Although not a readily available test, the hallmark finding of conjunctivalization reflects the presence of goblet cells in that area of the cornea and cytokeratine-19 positive cells.[36] Furthermore, a statistically significant percentage of patients with LSCD also have decreased corneal sensitivity.[37]

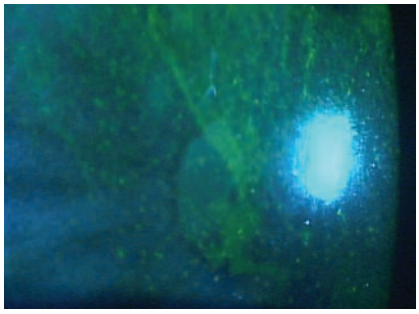
Mimickers Roundup

Besides the ocular conditions mentioned above, many others can mimic DED symptoms. These include conjunctival concretions that result in foreign body sensation and irritation. Removal of exposed concretions typically relieves the patient's symptoms. Less common causes include superior limbic keratoconjunctivitis, an allergy to a chronic medication such as brimonidine in glaucoma treatment or even reactions to preservatives in other chronic topical medications. Patients with raised pterygia or pinguecula can have dry, irritated, red eyes with burning and fluctuating vision; keep in mind that arid conditions and UV exposure can cause the formation of pinguecula and pterygia. Even exposure keratopathy in patients with lagophthalmos or previous eyelid cosmetic surgery procedures can develop inferior corneal staining, even though their tear film may not be affected at first. Eventually, it leads to MGD and DED.

As clinicians, it's imperative that we look at more technologies and use multiple tests in the diagnosis of DED. Relying on symptoms alone will likely frustrate both the patient and doctor. The advantage of multiple tests—especially more sensitive ones such as osmolarity, non-invasive tear break-up time measurements, meibography and even lissamine dye staining—is that they can help specify the precise type of DED, when performed in concert with slit lamp examinations and a comprehensive patient history. At the same time, running too many tests or performing too many procedures or treatments can make things difficult on patients and costly. Selecting the right tests to arrive at a confirmatory diagnosis is key and mild symptoms or subtle signs such as meibomian gland expression can point to the need for osmolarity, TBUT, staining and meibography. When things don't add up, start considering these differential diagnoses. This will allow you to address the patient's condition more accurately, quickly and, hopefully, lead to a resolution. ■

Dr. Karpecki is a consultant/advisor to: AMO, Alcon Labs, Allergan, Akorn, Bausch + Lomb/Valeant, BioTissue, Bruder Healthcare, Imprimis, Beaver-Visitec, Cambium Pharmaceuticals, Oculus, Rendia, Eyes4Lives, Focus Laboratories, Katena, Ocusoft, Shire Pharmaceuticals, BlephEx, Science Based Health, Sun Pharmaceuticals, TearLab, TearScience, Topcon, Paragon BioTeck, TelScreen and Visionmetrics.





At left, this patient's dry eye symptoms won't resolve until the underlying floppy eyelid syndrome is treated. At right, this patient's LSCD could be due to chemical burns and severe ocular scarring secondary to conditions such as Stevens-Johnson syndrome.

Dr. Krall is a consultant and advisor to eyeBrain medical.

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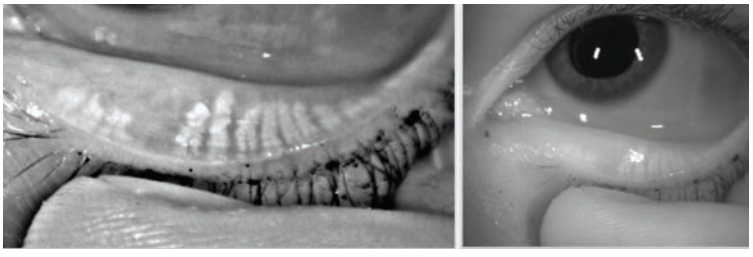
Motivate Your Patients with Meibomian Gland Imaging Techniques

Take a lesson from how this specialty practice uses imaging to help combat dry eye disease. By Kambiz Silani, OD

From automated refracting technology to autocapturing non-mydriatic fundus cameras, state-of-the-art diagnostic devices provide the finest and most updated eye care experience. Most recently, another device is taking the spotlight, aiding in the diagnosis of meibomian gland dysfunction (MGD): the meibographer.

In our boutique clinic in Beverly Hills, Calif., we've found the meibographer to be a tool that resonates with dry eye clients—particularly those middle-aged and younger. Its ability to put measurable results in the hands of our patients offers tremendous value, as it supports our patients' understanding of their condition and, hopefully, encourages their compliance with treatments.

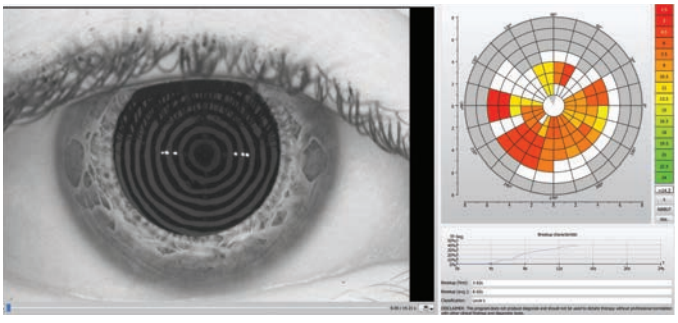
This article reviews how we use the meibographer, the benefits it's brought us and how you can incorporate one into your clinic—whether you run a boutique practice or a traditional family practice.



This 35-year-old female patient developed gland atrophy as a result of contact lens overwear.

Meibography Basics

Conventional meibography—also called "contact" meibography—used a transilluminating light probe and an infrared camera system to evaluate a small central region of the lid margin.[1] But the time-consuming and invasive nature of the test made it impractical. Approximately nine years ago, however, a non-contact method was introduced that used an infrared charge-coupled device attached to a slit lamp biomicroscope, without the need for a probe. Additionally, it expanded the area of observation to the upper and lower lids, providing the ability to perform extensive examination.[1] This is when the meibographer as we know it really took off. As manufacturers sought to distinguish their contributions, the market began to produce a variety of meibographers, each with unique capabilities (*Table 1*).



This patient, a 27-year-old female, displayed an abnormal tear break-up time.

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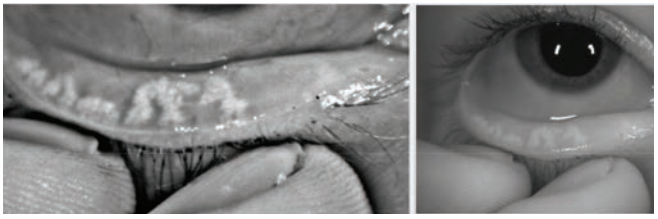
But for the most part, meibographers expose the meibomian glands, which, under healthy circumstances, have a piano-key-like appearance. Abnormal glands, on the other hand, will appear tortuous, dilated, congested or atrophied.



This 35-year-old patient was given a treatment plan of switching to daily, single-use contact lenses. We also advised her to avoid makeup near or around the eyelash margin, and started her on our dry eye kit.

Dysfunction Defined

The meibomian glands of the upper and lower eyelids play a valuable role in secreting the lipid layer of the tear film. Disturbances in their function may result in MGD. This can alter secretion and variations in tear composition, eventually leading to evaporative dry eye, which patients report as ocular discomfort.[2]

MGD can be classified as primary or secondary to systemic diseases; it can be focal or diffuse and may lead to symptoms of lid discomfort, neovascularization, ocular irritation, alteration of the tear film or inflammation of the meibomian gland orifices. Numerous structural changes can take place in MGD that will result in the functions of the glands being altered.[1,3] When the glands are blocked, there is not enough oil to coat the tear film, and the aqueous layer may evaporate rapidly. Without adequate lubrication between them, the eyelids and cornea can abrade each other, causing inflammation and cell damage.

As MGD progresses, structural changes within the eyelid margin may occur, such as thickening, rounding or distortion secondary to the destruction of the meibomian gland orifices. Even eyelash misdirection and weakness may occur as a side effect.

Other changes occur to the mucocutaneous junction of the lid, which may become irregular, displaced posteriorly or elevated. Additionally, meibomian gland orifices may change in number, representing a potentially permanent loss of these glands. Salvageable glands may be expressed (by applying digital pressure to the lids) and the secretions assessed; the secretions may be classified as clear, cloudy, granular or inspissated (toothpaste consistency). These changes can be seen in MGD, where the meibomian glands do not function efficiently or where a disturbance exists within the functional mechanisms of these glands.[4]

MGD Drivers

According to the prestigious MGD Workshop report, researchers suggest that MGD may be the leading cause of dry eye disease (DED) throughout the world.[5] With more than 50% of patients knowingly or unknowingly suffering from signs or symptoms of DED, eye care professionals must educate patients on the causes, symptoms and treatment of this highly prevalent condition.[5] Patients who could benefit from screening for MGD include, but are not limited to, contact lens wearers, patients living in dry climates and those with ocular allergies, blepharitis, marginal staphylococcal keratitis, ocular rosacea, dermatitis (eczema) or psoriasis. Even patients who are considering refractive surgery (i.e., LASIK) should consider testing for MGD to improve post-op outcomes.

Additionally, many common prescription and over-the-counter systemic medications may contribute to dry eye symptoms, including antihistamines, decongestants, hormone replacement therapy, contraceptives, antidepressants, diuretics, beta blockers, acne medicine and sleeping pills.[6] Often, patients may leave one or more of these off their list of current medications because they are not aware of the ocular side effects.[6] Be sure to specifically probe about these medications, as well as their frequency, dosage and duration of use.

Unfortunately, DED also affects young, healthy patients. In a national survey polling 1,000 optometrists, approximately 90% agree the use of modern technology contributes to dry eye symptoms. Furthermore, DED is becoming more common because of today's digital device use with an increase in patients between the ages of 18 and 34 with dry eye symptoms.[7]

Seeing is Believing

In my practice, I commonly diagnose MGD patients in their 20s, 30s and 40s. Some may present with no symptoms while others display vision fluctuation, contact lens intolerance, photosensitivity, ocular hyperemia, epiphoria or any combination of these.

Whatever their age or symptoms, the goal is the same: to address the signs and symptoms of MGD before they become irreversible. All patients should be educated on the importance of early detection and prevention to avoid chronic issues later in life. Here, meibography is

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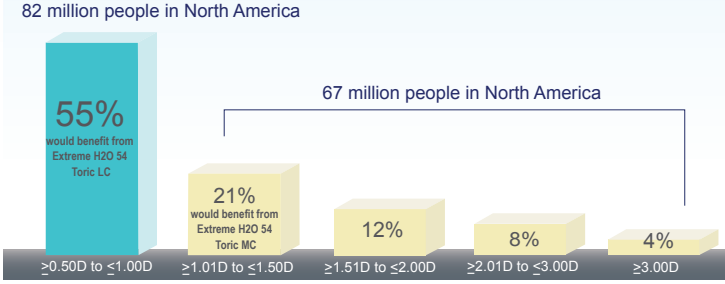

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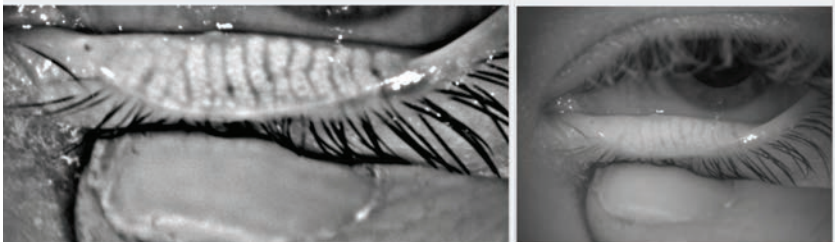
1 Morgan PB, Efron SE, Efron N, Hill EA, Inefficacy of aspheric soft contact lenses for the correction of low level of astigmatism. Optom Vis Sci 2005; 82: 823-82

Table 1. Meibographers on the Market

Keratograph 5M (Oculus)	Offers a relatively comprehensive DED analysis that shows the rate of tear evaporation, measures the tear meniscus height, scans for conjunctival hyperemia, analyzes lipid layer thickness and evaluates the structure of the glands.
Lipiview II (TearScience)	Provides a high-definition images for accurate visualization of the gland structures, measures lipid layer thickness and evaluates a patient's blink rate and pattern.
LipiScan (TearScience)	Offers rapid imaging of the glands using the company's dynamic meibomian imaging; the device is limited to just this feature.
Meibox (Box Medical Solutions)	Works as an efficient, lightweight option that still offers high-resolution image of the glands.



This is how the meibomian glands of a healthy, pre-LASIK patient should appear.

a powerful tool in MGD diagnosis. The key is to image the patient's meibomian glands (MG), then review any changes in anatomical structures. If changes exist, offer alternative lifestyle solutions as well as treatment options to increase the likelihood of MGD relief. When patients recognize the difference between their glands and healthy ones, they tend to be genuinely concerned and open to a custom treatment plan.

Our Workup

Properly diagnosing, educating and treating MGD patients is key to satisfaction. To start, ask the appropriate questions with the SPEED II questionnaire for ocular surface disease.[8] Next, assess the anatomy of the glands using infrared imaging with a meibographer, looking for destruction of glandular tissue vs. normal appearance. Non-invasive keratograph break-up time (NIK-BUT) or tear break-up time (TBUT) can be helpful in assessing the stability of the tear film and therefore the severity of MGD. Another test provides early presence of ocular surface disease for an inflammatory marker. The InflammaDry (Quidel) detects matrix metalloproteinase-9 (MMP-9), a cytokine produced by epithelial cells experiencing inflammation.[9] Knowing if a patient's MGD is exacerbated by inflammation (i.e., elevated MMP-9 levels) can guide the treatment plan.

When evaluating the anterior segment at the slit lamp, we use a Korb Meibomian Gland Evaluator (TearScience) to assess the function of the oil glands. The MGE exerts pressure on the outer eyelids consistent with a deliberate blink, allowing evaluation of gland secretion.[10] The glands are classified as one of the three following grades: no secretion, inspissated (or cloudy) secretions or normal secretions.

These diagnostics collectively tell a story that ultimately leads to a customized and favorable treatment protocol.

Happier Patients, Happier Doctors

For our patient base, we offer both in-office and at-home solutions. In the office, patients are presented with BlephEx (RySurg), LidPro (Mibo Medical), Lipiflow (TearScience), Mibo ThermoFlo (Mibo Medical) and meibomian gland expression. BlephEx and LidPro are handheld devices used in-office to gently exfoliate the eyelid at the lash line and remove the inflammatory biofilm that leads to chronic lid disease and discomfort.[6]

Another in-office procedure is thermal pulsation (Lipiflow), which provides controlled, safe heat (~108° F) for 12 minutes to the inner eyelid surface while pulsating pressure is simultaneously applied to the outer lids.[11] This intermittent pressure combined with the effective heating allows the meibomian gland oils to release without causing injury to the eye itself.[12] One of the few drawbacks of Lipiflow is its high out-of-pocket cost.

An alternative, economical approach incorporates the in-office heating device Mibo ThermoFlo, which is applied at the same constant 108° F but only externally to the eyelids. In my practice, many patients—even denizens of Beverly Hills—find the latter more cost effective, as well as quite soothing. This is immediately followed by manual massage with a meibomian gland expressor to release the stagnant oils, unclogging the glands.

To complement these in-office services, we offer our 60-day dry eye kit (or MGD kit) with premium eye care products. Three key items included are a high-quality warm compress with medical grade beads to maintain and deeply penetrate, such as Bruder's Medibeads mask; a gentle, anti-inflammatory lid wash from Ocusoft or Zocular;

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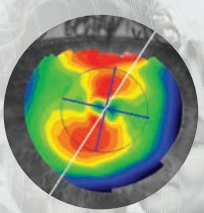

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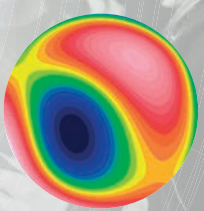

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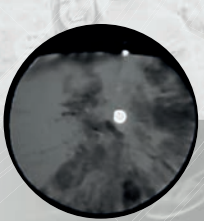
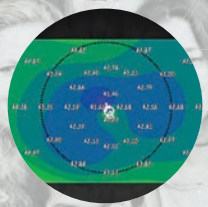


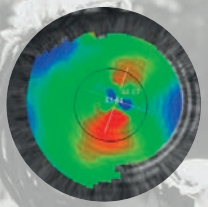
Lens Opacity

Cataracts



Posterior Corneal Elevation

Keratoconus risk



Topography

Coding Connection

By John Rumpakis, OD, MBA, Clinical Coding Editor

Get the Picture: Coding MGD Imaging

Is educating patients with ocular surface imaging billable to carriers?

The surge of ocular surface disease (OSD) management in optometric practice has driven technology to the forefront. Now, it not only helps to diagnose and treat this disease state but also creates the opportunity to educate and motivate patients to begin and maintain their treatment regimen.

The issue with imaging, of course, is who is paying for it—and that depends on why the image is taken and the role it plays in diagnosing, treating or educating and motivating the patient.

Necessary Imaging

New MGD imaging technologies provide images of the anterior surface of the eye for clinical evaluation of OSD. Much of the manufacturers' literature also touts the marketing and educational value of the images to help patients have a better understanding of their clinical status. That, however, cannot be the primary reason for capturing an image and billing to a third-party carrier, even if it is reimbursable with their clinical diagnosis.

This is where we have to revisit the concept of medical necessity. According to Medicare, it is defined as:[1]

> *Services or supplies that are proper and needed for the diagnosis or treatment of the patient's medical conditions, are provided for the diagnosis, direct care and treatment of the patient's medical condition, meet the standards of good medical practice in the local area and aren't mainly for the convenience of the patient or the physician.*

Just because you have a piece of cool tech that you want to use and a billable diagnosis doesn't mean you have the green light to capture the image and bill the carrier. Taking the image for educational purposes and not billing the patient isn't acceptable either because it demonstrates a discriminatory or biased approach to your billing decisions. If an image is clinically worthy to capture, it should be billed either to the carrier or the patient. If you have a philosophy of capturing images but only billing to the carrier, that wouldn't be appropriate, and if you capture images and don't bill the patient, that could be viewed as an inducement or biased decision-making.

Proper Coding

My advice is, if your patient has clinical signs and symptoms of OSD and you order imaging to assist in diagnosing, following a diagnosis, treating or following the treatment, you have established proper medical necessity. The CPT codes you will most likely encounter are:

- 92285: external ocular photography with interpretation and report for documentation of medical progress.
- 92025: computerized corneal topography, unilateral or bilateral, with interpretation and report.
- 0330t: tear film imaging, unilateral or bilateral, with interpretation and report.

All of these tests require an interpretation and report with:

Clinical findings: pertinent findings regarding the test results.

Interpretation of the findings.

Comparative data: comparison to previous test results, if applicable.

Clinical management: how the test results will affect management of the condition/disease going forward:

- Change, increase or stop medications.
- Recommendation for surgery.
- Recommendation for further diagnostic testing.
- Referral to a specialist/subspecialist for additional treatment.

Don't forget that the CPT clearly states, "interpretation and report by the physician is an integral part of the special ophthalmological services where indicated and that the technical procedures (which may or may not be performed personally by the physician) are often part of the service, but should not be mistaken to constitute the service itself." Technically, a diagnostic test is not complete until the physician's interpretation and report is finished.

New imaging technology provides a great opportunity for patients with this chronic disorder to receive a prompt diagnosis and proper treatment. But be sure to follow the basic rules of medical necessity and create a bullet-proof clinical record by including all elements required to meet the definition of the CPT code used. Don't fall into the trap of using this technology in a cavalier manner with inconsistent billing patterns.

Send your own coding questions and comments to rocodingconnection@gmail.com.

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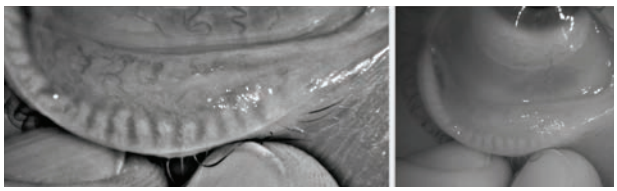

This patient displays significant gland atrophy.

and pharmaceutical grade omega-3 supplements (we prefer Nordic Naturals or PRN). If the patient has a positive MMP-9 result, we consider starting Restasis (cyclosporine 0.05%, Allergan), Xiidra (lifitegrast 5%, Shire) or a steroid drop prior to punctal occlusion. Inserting a punctal plug in the presence of active inflammation may cause an increase in ocular surface disease symptoms.

Another option that may prove valuable is a higher viscosity, preservative-free artificial tear such as Retaine MGD (Ocusoft) or Oasis Plus (Oasis) to provide instant as well as longer lasting relief. These drops may sooth the ocular surface both directly by providing lubrication and reducing friction between the lid and the eye, as well as indirectly by diluting inflammatory cells until they are eliminated.

Not every patient will want or need the somewhat indulgent, spa-like treatments often favored by those who live in the 90210 zip code. Cheaper, simpler alternatives can often suffice. But educating patients about the role of the meibomain glands through direct visualization does impress upon them the importance of good lid hygiene. I consider it a foundational step that sets the patient up for success. An educated patient will be more amenable to working with you to develop a treatment regimen that fits their lifestyle, budget and clinical picture. ■

Dr. Silani is the chief clinical director at Beverly Hills Optometry in Beverly Hills, Calif.

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Testing Children for Accommodative and Convergence Disorders

To obtain accurate test results for pediatric patients, consider these clinical strategies.

By Jasleen Jhajj, OD

Accommodative and binocular vision (BV) conditions are about nine times more common than ocular disease in patients six months to 18 years old.[1] Of these, approximately 20% have a binocular vision condition (*Table 1*).[1]

These bold statistics highlight how necessary it is for primary care optometrists to assess every pediatric patient's BV system. Many patients with BV conditions can be easily managed in the primary care setting with a few tips.

This article streamlines the primary care pediatric exam by providing practical strategies for successfully examining children as well as BV test selection, interpretation, and diagnostic techniques.

Fig. 1. This young patient is being trialed with overminus lenses. Constant exotropia improves to intermittent exotropia with -2.00 OU.

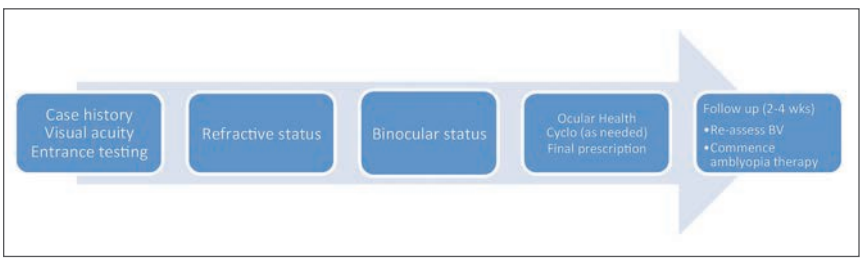

Fig 2. Follow this sequencing chart to perform a proper BV exam.

Exam Strategies

An attentive patient is essential to the binocular vision exam because it ensures fixation and accommodation.[2,3] Consider a patient with esophoria at near who has an accommodative convergence/accommodation ratio (AC/A) of 9/1. If this patient is distracted and under-accommodates by a diopter during cover test, they might appear orthophoric, or even exophoric (relaxing accommodation increases divergence).[2,3]

To ensure adequate fixation, continuously engage the patient by asking questions about the target, such as, "What color is Spiderman's face," or "Can you see Spiderman's two eyes?"

You can verify fixation by moving the target from side to side very slightly (1cm to 2cm), and watch for the expected pursuit movement. This is a highly sensitive technique because a pursuit movement not only requires accurate fixation but accommodation as well.

Pediatric patients with BV conditions can be more passive and

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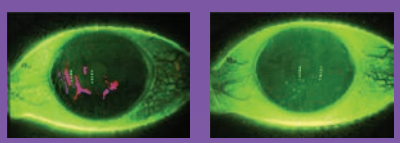

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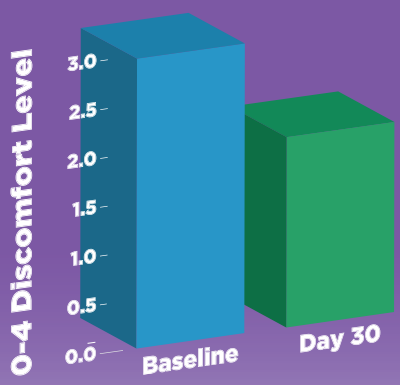

Case 1. Uncorrected Hyperopia with Esotropia

	Visual Acuity	Cover test
Uncorrected	OD 20/20 OS 20/20	Dist 15 IAET Near 18 IAET
Dry retinoscopy OD +5.00 DS OS +7.00 DS	OD 20/30 OS 20/70	Dist ortho Near 3 exo
Manifest OD +1.00 DS OS +1.00 DS	OD 20/20 OS 20/25	Dist 5 IAET Near 8 IAET

The patient has refractive esotropia that improves to resolution with full correction of refractive error. If the manifest refraction was prescribed without assessing the BV system, the patient would be left with a residual strabismus and anisometropia. Previously uncorrected high hyperopes will always present with reduced DVA through dry retinoscopy, because the lens is in accommodative spasm. An objective measure of refractive error is critical in the management of all pediatric patients. Final prescription is based upon binocular status, dynamic visual acuity (DVA) and age. Here are some tips to treat these patients:

- Older children require good DVA for school and are given a partial prescription (offering ~20/30 OU DVA). Preschool children rely less on their distance vision because their world is at arm's length. They can be given more of the full prescription initially.
- Cut the distance prescription and prescribe a near add to facilitate adaptation. Follow up every two to four weeks and incrementally increase the prescription, as accommodation relaxes and DVA improves. If binocularity is good, the near add can be removed entirely.
- In general, cut the hyperopia by the same amount in each eye from the cycloplegic refraction. However, you may not cut symmetrically initially if the prescription was, for example, +4.00 in one eye, and +0.25 in the other.[2,3]

inattentive during the exam because the task is difficult. Explaining that the testing is like "gym class for the eyes" can encourage participation by creating a relatable analogy. If the patient responds poorly, switch to objective testing (e.g., for vergence ranges, use prism bars and watch for the expected converging/diverging eye movements).

Controlling fixation and accommodation is not difficult if the optometrist is vigilant about target selection, room lighting and verifying fixation. Targets for fixation that are small and of high contrast most effectively stimulate accommodation and encourage good fixation.[2,3] Sticker targets are excellent for young children, and can be the deciding factor in the exam's success. Stickers should be small, popular among children and include fine detailing to guarantee accurate accommodation. We have successfully had infants fixate stickers with proper engagement.

For distance fixation, parents can load a video on a smartphone or tablet computer. Normal room lighting should be employed during BV testing. The only tests where dim room lighting is indicated are binocular fused cross cylinder test (rarely used in pediatrics), retinoscopy and manifest refraction.

Pediatric patients with BV conditions do not always complain to adults because they may have no concept of what "normal" even is. To guard against this possibility, optometrists must ask probing questions during case history, and always question the family about symptoms if a BV condition is suspected during the exam.[2,3]

Optometrists should ask about frontal headaches, visual blur, diplopia, words jumping or moving on the page and poor attention and scholastic performance.

Be Comprehensive

In a busy private practice setting, optometrists are constantly challenged to be efficient, yet thorough. However, to rule out binocular dysfunction, every primary care exam must evaluate eye alignment and fixation, eye movement skills, vergence skills and accommodative skills.[2,3]

To perform a BV test in a proper sequence and select sensitive tests that minimize redundancy, follow the exam sequencing succession chart provided (*Figure 2*). Refractive error that induces amblyopia or disrupts normal binocular functioning requires a prescription (*Table 3*).

Table 1. Most Common BV Conditions in Children[1]

	Accommodative (5%)	Non-strabismic (14%)	Strabismic (12%)
Most common	• Accommodative insufficiency • Accommodative excess	• Convergence excess • Convergence insufficiency	• Constant esotropia
Least common	• Accommodative infacility • Ill sustained accommodation	• Divergence excess • Divergence insufficiency	• Intermittent exotropia • Intermittent esotropia • Constant exotropia

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Table 2. Recommended Minimum Database Testing for the Primary Care BV Assessment[2,3]

Test	School-aged	Preschool	Infant
Visual acuity	Snellen	Lea matching, picture chart	Preferential looking Fix and follow
Stereoacuity	Randot	Randot	Lang
Extraocular Muscles (EOM)	Transilluminator	Transilluminator	Finger puppet
Cover test (dist/ near)	Letter target Dist: 1 exo +/-2 Near: 3 exo +/- 3	Sticker target Dist: 1 exo +/-2 Near: 3 exo +/- 3	Sticker target Hirshberg/Kappa <4 mo: phoria, <20 ET/XT >3mo: 3 exo +/- 3
Refractive status	Retinoscopy Manifest	Retinoscopy Manifest	Retinoscopy
Near point of convergence (NPC) *–Non-accommodative target*	Transilluminator 7cm/10cm	Transilluminator 7cm/10cm	Transilluminator 7cm/10cm
Near point of convergence (NPC) *–Accommodative target*	Letter target 5cm/7cm	Sticker target 5cm/7cm	Sticker target 5cm/7cm
Accommodative amplitude	Letter target 15 – ¼(age) +/-2	Letter target 15 – ¼(age) +/-2	
Vergence facility (12 BO/3 BI at near)	Letter target >10 yo: 15 cpm +/-2		
Binocular accommodative facility (+/- 2.00)	Letter target 8-12 yo: 5 cpm +/- 2.5		

In children older than three years, +2.00D of uncorrected bilateral refractive error can be disruptive in some cases.[2-4]

BV testing is performed with the intended refractive correction in place. If no significant refractive error is found, testing is performed uncorrected.

Retinoscopy vs. Autorefraction

In pediatric exams, autorefractors frequently give falsely myopic readings due to the effects of proximal accommodation.[2] The SPOT (Welch-Allyn) autorefractor is the exception and produces readings similar to actual refractive error.[5] Still, retinoscopy provides valuable data an autorefractor would miss.

Retinoscopy racks/sciascopy bars are useful for young children who are unable to sit behind the phoropter. Be careful to scope along the visual (foveal) axis, while maintaining working distance. Scoping off axis may result in more myopia or astigmatism. This becomes especially important when assessing patients with strabismus. A neutralizing prism should be placed over the fellow eye to help the practitioner scope on axis.[2,3] Another option is to occlude the turned eye during retinoscopy to ensure the eye is fixating the target.

Case 2. Near Esophoria and Convergence Excess (CE)

CT (dist)	Orthophoria
CT (near)	8 esophoria
Retinoscopy	Plano OU
Near BI	x/12/10
AC/A	4/1

This patient's CT is abnormal, and the compensating vergence range is near BI. This child might benefit from a near add. Binocular accommodative facility (BAF) and monocular estimation method (MEM) retinoscopy can both help determine whether the patient will be symptomatic. If symptoms persist at follow up, the child should be referred for vision therapy to increase BI reserves.[2,3]

CE is differentiated from near esophoria by a high AC/A. Clinically, this distinction helps the optometrist understand the utility of a near add (i.e., high AC/As respond best to adds). Newly corrected myopes often have near esophoria in the presence of accommodative insufficiency (AI) and normal BI reserves. The eso is secondary to poor accommodative skills (convergence is driving accommodation) and usually improves following spectacle adaptation.[2,3]

Eye Alignment

An uncorrected distance and near cover test (CT) is recommended for all patients. If spectacles are being prescribed, CT may be repeated with the correction in place to note changes in alignment (*Case 1*). Repeating the cover test depends on the refractive error found and is not always necessary.

The expected CT results are 0-3 exo for distance, and 0-6 exo at near. Esophoria is always abnormal. Patients who deviate from expected results need to have their compensating vergence ranges, and AC/A, measured (*Figures 4 and 5*).[2,3] Base

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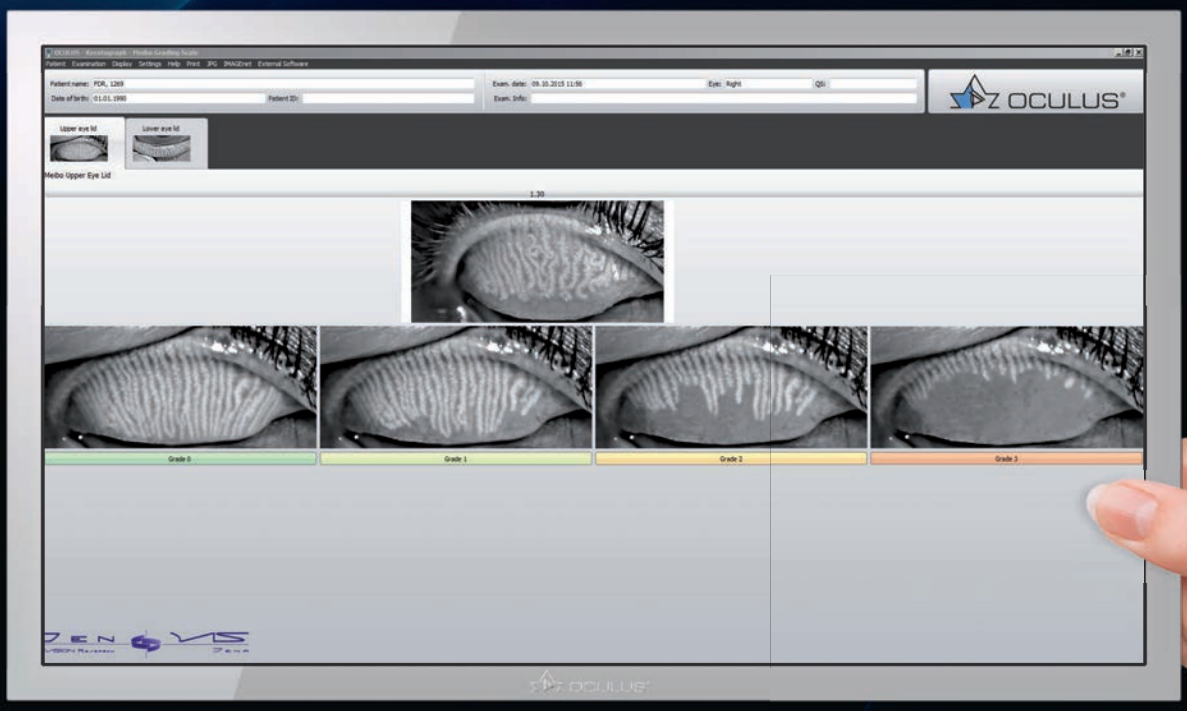

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Fig. 3. This young patient is using a polaroid bar reader and glasses as suppression checks for binocular accommodative facility testing.

in (BI) ranges compensate for eso deviations (norms near 11/19/10, dist x/5/3), and base out (BO) for exo (norms near 14/18/17, dist 7/15/8). Gradient AC/A is calculated by repeating CT with -1.00 OU at near. A high AC/A is defined as a change in phoria greater than 6pd, and a low AC/A is a change less than 2.[2,3]

CT is valuable because it is objective and can be confidently performed on most children. Always give the patient enough time to fixate and accommodate the target before moving the paddle and verify by moving the target side to side. Patients with strabismus have poor monocular skills and take longer to fixate.

Patients with symptomatic convergence excess (CE), near esophoria or accommodative esotropia frequently have poor binocular accommodative skills because they are continually relaxing accommodation to help with divergence. Hence, CT often underestimates the eso. The importance of a careful CT with a small, accommodative target cannot be understated.

Hirschberg/Kappa is a test of alignment for patients who are poorly attentive or can be used to confirm a CT finding. To perform this test, a transilluminator is held at 40cm. Typically, corneal reflections are displaced 0.5mm nasally from the center of the pupil. If the light is displaced nasally, the eye is turned out; if the light is displaced temporally, the eye is turned in. Alternately, occluding each eye verifies angle kappa (location of reflection relative to center of pupil). A displacement of the light 1mm equates to 22pd.[3]

Managing Strabismus

The first question with this condition tends to be, "Is the deviation constant or intermittent, and does it alternate?"

Establishing frequency and laterality gives valuable insight into the patient's sensory status and guides all management decisions.[3] Infants' constant deviations that do not improve with refractive correction require timely interventions to facilitate the development of stereopsis.

If correction of the refractive error does not improve the deviation, a Fresnel prism may be considered to achieve alignment.

Surgery should be considered for infants only as a last resort due to the risks. The Infantile Esotropia Observation Study showed infants with large angle deviations who had surgery at six months had better stereo outcomes at four and a half years than those who waited until seven to 15 months.[6,7]

Children with intermittent deviations do not require immediate referrals because development of stereopsis is not at risk.[2,3] Infants

Table 3. Amblyogenic Refractive Error[2-4]

Anisometropia		Bilateral
Hyperopia	>0.75 D	>4.00 D
Myopia	>3.00 D	>-8.00 D
Astigmatism	>1.50	>-2.50 D

Accommodative Insufficiency

This is the most common accommodative condition among children. Patients tend to be symptomatic if their amplitude of accommodation is less than two times the dioptric working distance.[2] In young children, MEM is useful for identifying symptomatic patients.[2]

Treating accommodative conditions. Most patients with accommodative conditions experience symptomatic relief with a near add because it reduces the need for patients to use the accommodative system *(Table 4)*.

Table 4. Accommodative Conditions[2]

Accommodative insufficiency (under accommodating)	Reduced AA Difficulty clearing (-) on BAF	TREATMENT Near add –refine with MEM Vision therapy
Accommodative excess/ spasm/pseudomyopia (over accommodating)	Fluctuating retinoscopy Difficulty clearing (+) on BAF Possible esophoria	
Accommodative infacility (slow accommodative response)	Reduced BAF Reduced MAF	
Ill-sustained accommodation (difficulty maintaining accommodation)	Reduced AA after repeated testing	

and toddlers with constant strabismus who do not alternate fixation almost always have amblyopia due to lack of sensory stimulus to the turning eye. With that in mind, in the absence of confident visual acuity (VA) measurements, commencing amblyopia therapy on this evidence alone is appropriate.[2,3]

Esotropia and Exotropia

Patients with esotropia at near should have CT re-performed with plus at near over the proposed distance prescription. If the magnitude or frequency improves, a near add maximizing alignment should be prescribed.[2,3] Sometimes, improvement does not occur right away, especially in partially accommodative cases. Prescribing an add is always recommended in these cases. (*Case 2*).

Patients with exotropia at distance and near may be re-evaluated with -2.00 overminus OU. In this case, the least amount of overminus that improves the frequency of the turn at distance is prescribed.[2,3,8,9] Overminus lenses are only indicated in strabismic cases and should not be used in treating convergence insufficiency (intermittent or constant exotropia at near only).

Retrospective case analyses demonstrate overminus to effectively improve eye alignment.[8,9] The final overminus is tapered over time.[8,9] Research into alternate patching (occluding the right and left eye for two hours every day) shows no therapeutic benefits over observation in children.[10,11]

Vergence and Accommodative Skills

Near point of convergence (NPC) should be performed with accommodative and non-accommodative targets (e.g., penlight). Patients with convergence insufficiency often leverage accommodation to drive convergence. Performing this test with a penlight is important because it can tease out a receded NPC.[2,3] Patients with "pseudo-CI" accommodative insufficiency (AI) will show an improved NPC with a non-accommodative target.[2,3]

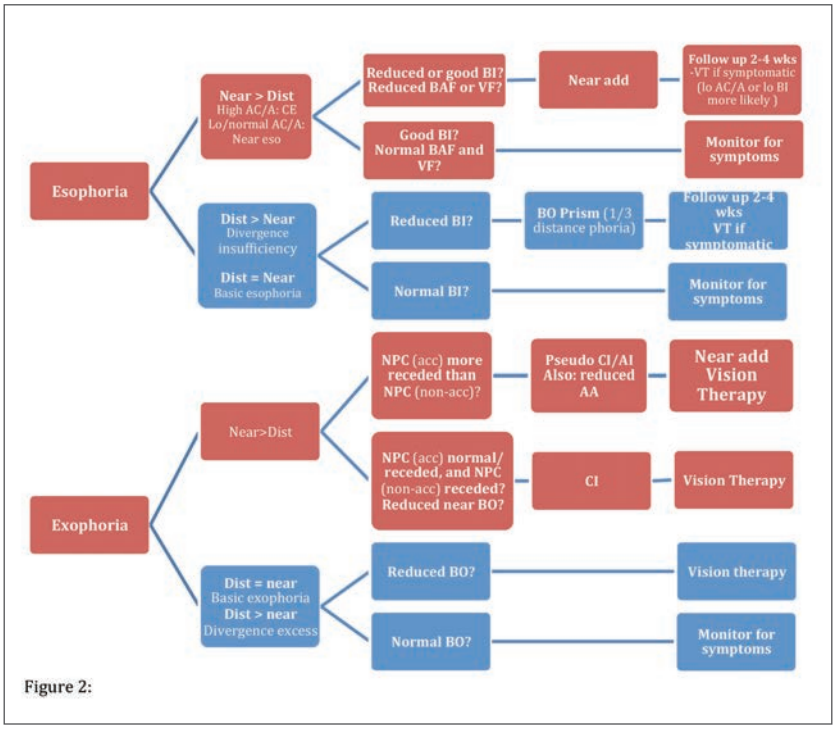

Fig. 4. Diagnosis and sequential management of heterophoric binocular vision conditions.

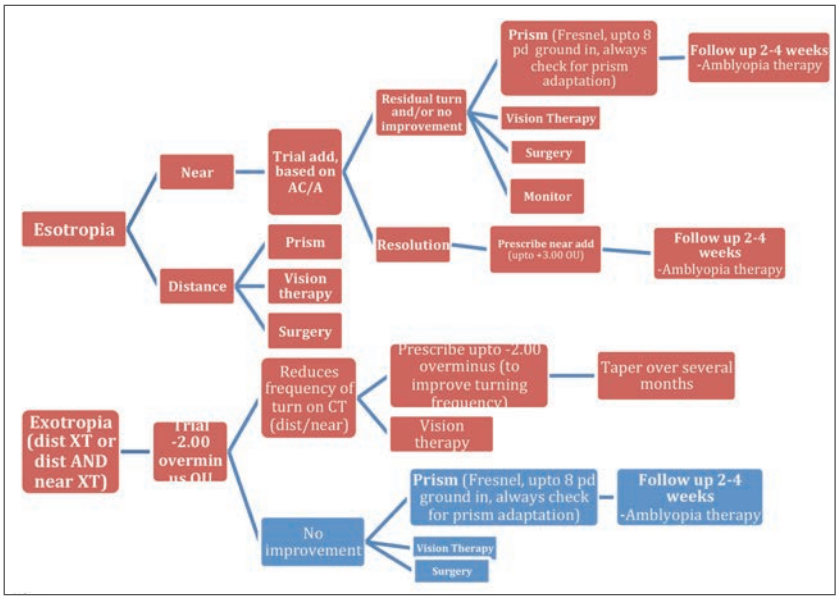

Fig 5. Diagnosis and sequential management of strabismic binocular vision conditions.

My Five Favorite Binocular Evaluation Tests

By Megan Sumrall Lott, OD

When examining children with complaints of blurred vision or strained eyes, a variety of tests can detect binocular deficit that spectacles alone may not correct. Today, more and more near point demands are being placed on children, and many are not able to meet them. These five tests can be easily administered without adding significant time to your examination:

1. COVD QOL Checklist

The first instrument is the College of Optometrists in Vision Development Quality of Life checklist. I include a 19-item checklist in my history. The information from this allows ODs to quickly determine if a visual problem is present. The checklist is divided into five areas: orientation, ocular motor, binocular, accommodative, visual perceptual.[1] The parents answer questions on a scale of zero to four and if the total score is 20 or more, suspect a functional visual problem. If you wish to pursue a diagnosis further, you can save time by identifying the area most marked. Don't forget: patients may have multiple, subclinical vision disorders.

2. Randot Stereo Testing

Stereopsis has been called the single best indicator of the overall function of both the sensory and motor portions of the visual system.[2] A Randot or near point stereopsis test of your choice will work well. By school age, a child should be able to appreciate 20 seconds of arc. If a decreased appreciation of depth perception is apparent, a binocular dysfunction may be present.

3. Vergence Facility Using a Prism Stick

This test also addresses binocular deficiency, but in a more dynamic way. The patient will be changing their viewing distance from distance to near (think copying notes from the chalkboard). Vergence facility is both subjective and objective, and yields useful information beyond the facility alone. To test vergence facility, you will need a 12BO/3BI prism stick. The patient is asked to fixate on a target, such as a Wolff Wand. As the BO is presented before the eye, this should immediately cause diplopia. Ask the patient to report if they see two images and to let you know when the ball returns to one. Once the patient is able to fuse the target, rotate the prism to present the BI. Again, this should elicit diplopia and the patient is to report when ball becomes one.

This is performed for 30 seconds, and each change of the prism is counted. The expected result for this test is 15 cycles/minute.[3] If the patient is lower than this, they may have a binocular problem. Keep in mind that some patients may suppress, so watch the eye movement to ensure that they are actually fusing two images. You should see the eyes move in the appropriate direction with each change of the prism. In particular, observe how a patient focuses while actively engaged in near tasks.

4. Spot Retinoscopy

To test the accommodative response, get out your retinoscope (preferably spot) and an age-appropriate reading card. The patient holds the card at 40cm and begins to read. If you notice movement, slowly move the retinoscope back while maintaining the card at the same distance, observing the reflex until a neutral reflex is seen. Measure the distance from the card to the retinoscope and record in diopters.

5. NSUCO Ocular Motility Testing

Poorly controlled eye movements are the biggest indicator of poor reading skills. When testing for ocular motor accuracy, I like the grading standards of the Nova Southeastern University College of Optometry Ocular Motility Test. These standards are straightforward and easy to retest. Pursuits and saccades are graded on a scale from one to five in the areas of ability, accuracy, head movement and body movement. This test is administered with the patient standing. Again, use a Wolff Wand as a target. If significant head or body movement is noted, suspect a significant ocular motor deficiency.[4] ■

Dr. Sumrall Lott is founder of Belle Vue Specialty Eye Care in Hattiesburg, MS.

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Accommodative amplitudes are measured using a push up, pull away or minus lens to blur. In young children, pull away can be efficiently performed by occluding an eye, presenting a 20/20 target close to the eye and asking the child to "guess the secret letter" as the fixation stick is slowly pulled away. The dioptric working distance is measured when the correct answer is given.

Vergence facility (VF) and binocular accommodative facility (BAF) are sensitive tests for uncovering symptomatic conditions.[2,12] These tests are efficiently performed without the phoropter.

For VF, the patient fixates a 20/30 target at 40cm and alternates between fusing 12BO/3BI. The cycles per minute (CPM) are recorded (less than 12cpm is indicative of a vergence disorder).[2,12]

BAF is administered with +/-2.00 flippers, polaroid glasses/bar reader and a near target. Research demonstrates the importance of suppression checks to ensure continued binocularity.[2] If the patient is suppressing, the target, as seen by the suppressed eye, appears black.

Failing (+) on BAF is indicative of a convergence disorder, or AI.

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Fig. 6. Constant alternating esotropia improves to 4 esophoria through +1.50 add.

Failing (-) suggests accommodative excess or a divergence disorder. Decreased overall CPM may be associated with any BV condition.

Prescribing

A near add is indicated in most accommodative and some vergence conditions. It can be quickly determined based on the results of monocular estimated method (MEM) testing *(Figure 7)*.

MEM retinoscopy measures binocular accommodative response. It is performed at Harmon's distance (between child's shoulder and elbow). MEM is performed over the subjective distance. Age appropriate targets are attached to the retinoscope and lenses are quickly dipped down to neutralize the reflex (*Figure 4*). Expected values are +0.25DS to +0.75DS. The add power is increased if a lag (extra plus) is noted, and decreased with a lead.[2]

All patients with esophoria, esotropia, moderate to high hyperopia, and accommodative spasm should undergo cycloplegic retinoscopy.[2] Patients with accommodative spasm demonstrate against motion on MEM retinoscopy. The reflex switches between neutral and against motion, which may frustrate the optometrist, but is strong evidence of this condition. These patients typically have a low minus prescription in distance (<-1.00D), which reduces to plano or hyperopia on cycloplegic retinoscopy. They may also be esophoric.[2,3]

Fig. 7. Monocular estimated method retinoscopy near target cards.

Patients with newly uncovered BV conditions should be followed up two to six weeks after they have been wearing their new spectacles to assess adaptation and BV status.[13]

Additional amblyopia therapy should not be started before the child has adapted to the spectacle prescription. Spectacle therapy is first-line intervention for amblyopia. Maximal improvement of VA occurs at 18 weeks, with 32% showing complete resolution at this time.[13]

The final decision to begin amblyopia therapy is based upon patient factors and clinician preference (e.g., if patching is burdensome, therapy may be deferred). Bangerter foils, patching and atropine therapy are all effective treatments. In-office vision therapy is a good option.[2,14-16]

An attentive child is critical for the BV exam. Children must be engaged and participating throughout to ensure accurate results. Significant refractive error must always be corrected prior to BV evaluation. For a primary care exam to be comprehensive, it must evaluate alignment, accommodation, vergence and eye movement skills. BV conditions are managed with near add lenses, prism, overminus, vision therapy or surgical referral. ■

Dr. Jhajj is an assistant professor at Nova Southeastern University where she teaches binocular vision, pediatric, vision therapy and primary care.

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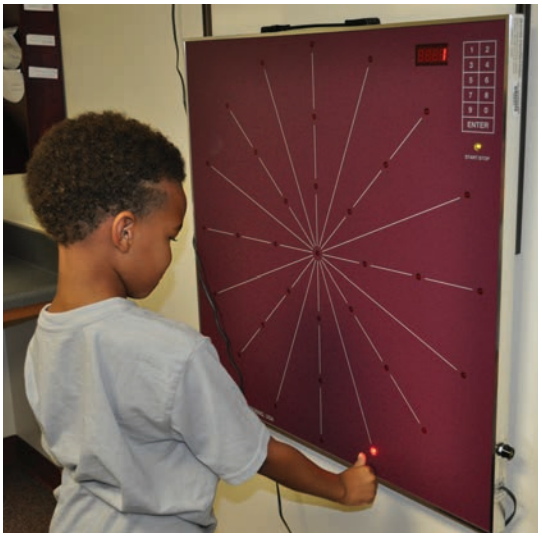
Managing Amblyopia: Can Vision Therapy Cut It?

New technologies are making this once-controversial treatment option more viable.

By Kara Tison, OD, and Amanda Nicklas, OD

Amblyopia, a disorder of visual development that occurs in childhood, can have lasting effects in adulthood if not detected and treated in a timely manner. It occurs frequently, with a prevalence between 1% and 5% of the general population.[1] The World Health Organization estimates 12 million children younger than age 15 are visually impaired due to uncorrected refractive errors and amblyopia.[1] Research also suggests functional amblyopia is the leading cause of monocular vision loss in adults older than age 20.[2] This vision loss can be prevented with timely diagnosis and treatment, and clinicians are capable of providing their amblyopic patients with good vision in childhood that can last throughout life.

This is not a rare condition, and eye care providers, particularly those caring for a pediatric population, must have the knowledge and tools to effectively treat amblyopia.[1] While refractive correction and occlusion are mainstays, vision therapy is making new inroads.



This oculomotor activity addresses saccadic dysfunction with a Wayne saccadic fixator and a timed exercise with multiple setting and testing options.

Ocular Effects

Although visual acuity is often the focus of management, amblyopia impacts many other elements of the visual system. Patients with amblyopia often have decreased oculomotor and fixation ability, accommodative skills, binocular summation, contrast sensitivity and stereoacuity.[3] These deficiencies are often paired with increased spatial distortion and crowding effects, as well as suppression under binocular viewing conditions.[2,3] Amblyopia can affect a child's ability to function in a classroom setting and increase their chances for a vision-based learning problem.

Early detection is key in preventing long-term effects of amblyopia. The primary development of visual pathways occurs from birth to six to eight years of age.[2] During this time, the visual system is most vulnerable to amblyogenic factors—the mechanisms that disrupt normal visual development. Such factors include optical defocus, strabismus, form deprivation or a combination of all three.[2] If any of these are present at birth up to age eight when visual development is susceptible, amblyopia can develop.

Accommodative flippers are used in many vision therapy activities.

Optical defocus. This is caused by uncorrected refractive error in one or both eyes. Anisometropic amblyopia is caused by a difference in refractive error between the two eyes, while isoametropic amblyopia is caused by high refractive error roughly equal between both eyes (*Tables 1 and 2*).[2]

Strabismus. Strabismic amblyopia is most commonly associated with an early-onset constant unilateral strabismus.[2] The deviation causes the eyes to be presented with two different images, causing double vision and visual confusion. Rather than trying to reconcile these different images, the image from the deviating eye is actively suppressed.[2]

Form deprivation. This type of amblyopia results from a physical obstruction on the visual axis that prevents a sharp, clear image from forming in one or both eyes. The obstruction must be present from birth to approximately age eight.[2] The most common cause of form

PEDIG Studies Under the Microscope

The PEDIG's Amblyopia Treatment Studies (ATS) are an integral part of amblyopia treatment paradigm. ATS 1, 2B, 4 and 10 deal with patients younger than 10 with moderate amblyopia. These studies determined:

- Six hours of prescribed daily patching is as effective as daily atropine.[1]
- Two hours of prescribed daily patching is as effective as six hours of prescribed daily patching.[2]
- Weekend atropine is as effective as daily atropine.[3]
- Bangerter foils are as effective as two hours of prescribed daily patching.[4]

Amblyopia Severity Grading[1-6]

Severity	Visual Acuity
Mild	20/25 to 20/30
Moderate	20/40 to 20/80
Severe	20/100 to 20/400

PEDIG also determined that six hours of daily prescribed patching is as equally effective as full-time patching in severe amblyopes ages seven and younger.[5]

For older children (between the ages of seven and 13) with moderate to severe amblyopia, patching can improve visual acuity even if the amblyopia has been treated previously. For children age 13 to 18, patching can improve visual acuity only if the amblyopia has not been previously treated.[6] Knowing the results of these studies provides a practitioner with multiple options for successful amblyopia therapy, even with difficult patients.

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deprivation amblyopia is congenital cataract, although it can also be caused by other conditions such as ptosis and corneal opacities.[2]

In addition to the mechanism driving development, amblyopia is categorized based on severity. The Pediatric Eye Disease Investigator Group (PEDIG) conducted a research series called the Amblyopia Treatment Studies (ATS), which provided not only a severity grading scale, but also guidance on treatment options for various age groups and visual acuities.

A thorough eye exam can help practitioners determine the presence and type of amblyopia. After the diagnosis, a typical treatment progression may include correction of refractive error with spectacles, occlusion therapy and vision therapy. PEDIG's extensive research in various treatment modalities provides practitioners a more uniform and effective level of care, and can act as a clinical guidepost for treating patients with amblyopia.

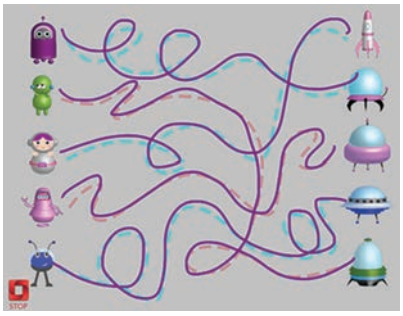
Spectacles

The first step to effective amblyopia treatment is correction of refractive error with spectacles. An accurate spectacle Rx is critical to ensuring a clear retinal image in each eye. The use of cycloplegics is necessary to obtain this information, particularly in pre-verbal children or those unable to successfully participate in the process of subjective refraction.

Skillful retinoscopy is especially useful to determine the presence or absence of anisometric or isoametropic amblyogenic risk factors. If amblyogenic risk factors are present, full-time spectacle wear becomes necessary for treatment.

Research shows spectacle correction alone can provide resolution in roughly 27% of patients ages three to seven with uncorrected anisometropic amblyopia.[4] For patients with strabismic or combined anisostrabismic amblyopia in the same age group, glasses alone were able to provide improvement of visual acuity of greater than two lines in 75% of patients, with 32% showing total resolution.[5] Spectacles can be worn until a plateau occurs in visual acuity. If there is residual amblyopia, additional treatment is necessary.

Photo: Gerull Labs



This is an anti-suppression activity through Gerull Labs' Opto App. It contains a library of activities that can be completed on an iPad without any additional equipment or with either a stereoscope or red/cyan glasses. This allows excellent cancellation and depth perception targets.

Occlusion

Total occlusion is achieved by patching of the better-seeing eye, while partial occlusion is achieved with atropine eye drops, Bangerter foils or contact lenses. All types of occlusion have the same guiding principle: occluding the better-seeing eye forces the amblyopic eye to work, allowing it to develop stronger neural connections in the visual cortex.[2]

Table 1. Anisometropic Amblyogenic Factors

Condition	Diopters
Hyperopia	>1.00D
Myopia	>3.00D
Astigmatism	>1.50D

Table 2. Isoametropic Amblyogenic Factors

Condition	Diopters
Hyperopia	>5.00D
Myopia	>8.00D
Astigmatism	>2.50D

Patching. Each type of occlusion therapy offers benefits and drawbacks, but all are equally effective when prescribed correctly and used with compliance. Adhesive patches are inexpensive, readily available and easy to use. However, these patches can also discourage binocularity, cause allergic reactions on sensitive skin and can be a cause for teasing or bullying for a school-aged child. Compliance also becomes an issue in children older than eight—an age group that can show a 50% rate of noncompliance.[2] Adhesive patches are easily removed and can be easy to peek around if a child is not being closely monitored while wearing the patch. To improve compliance with eye patching, consider decorative patches with child-friendly designs or encouraging patients to decorate their own patches. Prescribing the appropriate amount of patching and thorough education on its benefits can also help to improve compliance.

Atropine. This is only effective if the sound eye is hyperopic. The atropine drop makes near vision blurry in the unaffected eye, forcing the child to rely on the amblyopic eye for near work. Atropine drops are an excellent alternative for patients who are noncompliant with eye patching therapy, as it is impossible to remove or peek around an eye drop once instilled. This treatment modality is excellent for high-energy patients who fuss with an eye patch, children with busy schedules or older patients with cosmetic concerns. This treatment option also promotes binocular vision. Drawbacks of atro-

Vectograms can help assess fusion and expand fusional vergence during therapy. They can reveal how the binocular system is functioning.

Most activities incorporating a Marsden ball help address oculomotor dysfunction. This activity promotes hand-eye coordination and helps with spatial awareness.

pine include being a prescription eye drop that needs to be filled and an intense stinging sensation on instillation. Light sensitivity is also an issue, as atropine produces a dilated pupil.

Bangerter foils. These can be applied to a spectacle lens of the sound eye to produce a blurred image at both distance and near. As vision improves in the amblyopic eye, practitioners can reduce the grade of blur by changing the foils. These also help promote binocularity and are excellent for older children with cosmetic concerns. However, a Bangerter foil should be used with caution on those suspected of noncompliance, as it could be quite easy to peek around the glasses.

Vision Therapy

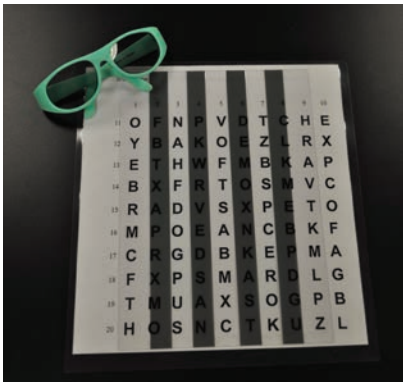
While traditional treatment for amblyopia improves monocular function by providing visual input to the amblyopic eye, vision therapy can assist in treating the underlying binocular dysfunction that accompanies amblyopia.[6] By initiating vision therapy, the doctor can reduce the total amount of therapy time necessary.[2,3,7] Additionally, adding vision therapy to occlusion, partial or total, is considered to be more effective than occlusion alone.[6]

Vision therapy can focus on increasing accommodation, improving accuracy of oculomotor skills, increasing vergence ranges, improvement of spatial perception, and breaking suppression and eccentric fixation.[7,8] The treatment goal is for the patient to effortlessly function with sound visual skills at a high level of binocularity. This high level of binocularity helps improve vision and will prevent the regression of visual acuity after amblyopia treatment has stopped.[2,3,6,9]

Accessing Vision Therapy

If your patient would benefit from vision therapy but you are unable to provide it in your practice, you can find a doctor who practices vision therapy by using the College of Optometrists in Vision Development's (COVD) locate a doctor search function (http://locate.covd.org) or the Optometric Extension Program Foundation's (OEPF) practitioner search map (http://oepf.org/page/map).

To add vision therapy to your practice, you can start by attending OEPF clinical curriculum courses (http://oepf.org/clinical-curriculum) or COVD courses (www.covd.org/?page=annual_meeting). In addition, hiring an optometrist with advanced training or clinical experience in vision therapy can help you incorporate this treatment modality into your practice with confidence.



In this anti-suppression activity, the patient uses a polarized bar reader with polarized glasses.

While all aspects of the visual system need to be addressed, a skilled practitioner can tailor the vision therapy plan for each patient's needs. Plans can be individualized to address those deficiencies observed in an exam, and the signs and symptoms reported by the patient.

Vision therapy consists of planned activities, individually prescribed and monitored to develop efficient visual skills and processing.[10] It typically consists of a 45-minute weekly in-office appointment, and the patient is typically assigned 15 minutes of nightly homework. Most therapies range from several weeks to several months, depending on factors such as the number and severity of diagnoses and patient compliance and motivation. For vision therapy to succeed, compliance is essential, and it is important to stress to the patient the importance of attending weekly therapy sessions and completing the assigned homework. With new computer-based therapies, tracking homework is easier than ever.

Vision therapy uses lenses, prism, filters, occluders and specialized equipment during prescribed activities.[10] Computer programs and interactive tablet apps have taken traditional vision therapy activities and turned them into new, interactive and exciting exercises for our patients. Another new development is the possibility of using virtual reality or binocular video games as a form of amblyopia treatment. PEDIG has been investigating the use of a binocular iPad game to determine if video game play can be as effective as patching. ATS 18 was designed to determine if amblyopic-eye visual acuity improvement treated with a binocular iPad game is as effective as part-time patching. For a younger cohort of patients, the study was inconclusive and unable to establish if iPad game play was worse than two hours of prescribed patching.[11] Research is still pending for an older age group.

Vision therapy activities used to address accommodation should increase accommodative accuracy, ability, amplitude and reaction time. Research shows incorporating lenses into accommodative activities, as in monocular accommodative rock and binocular accommodative rock, is more effective than activities that stimulate accommodation using a target distance as in hart chart near-far rock.[7]

To assist patients in developing normal eye movements and central fixation, the therapy plan can incorporate ocular motor activities to improve eye-hand coordination, accuracy of pursuits, saccades and fixation.[2] To improve the patient's spatial processing, the patient can complete word searches or hidden picture activities.

It is important to incorporate anti-suppression activities in amblyopia treatment, as patients with amblyopia have a complete, but suppressed binocular system.[12] Anti-suppression activities help promote binocularity in normal viewing conditions, improve fusion, expand vergence ranges and develop stereopsis.[6,12] To help lower suppression, the amblyopic eye participates in anti-suppression techniques that require it to become more competitive with the non-amblyopic eye.[8] Under artificial viewing conditions where the amblyopic eye is not suppressing, information from the two eyes can be combined normally.[12] There will be overall improvement the longer the patient is binocular in an artificial environment.[12] Some practitioners suggest introducing anti-suppression therapy early in the treatment plan to allow the amblyopic eye to function in a binocular state and encourage more natural binocular connections.[3]

So far, vision therapy has not been evaluated in randomized clinical trials.[13] PEDIG considered a clinical trial and completed a pilot randomized controlled trial of office-based

Amblyopia Management[2]

These treatment options are in no specific order and are determined by doctor and patient needs:

Anisometropic Amblyopia

- Refractive correction
- Occlusion
- Vision therapy (can reduce treatment time)

Isoametropic Amblyopia

- Refractive correction
- Vision therapy

Form Deprivation Amblyopia

- Removal of obstruction within first two months of life
- Refractive correction within one week after surgery
- Occlusion (if applicable)
- Vision therapy/stimulation activities

These patients need their visual acuity and binocular development followed closely the first year.

Strabismic Amblyopia

- Refractive correction
- Occlusion
- Vision therapy (may reduce therapy time by 50%)

active vision therapy. Unfortunately, there was insufficient recruitment and difficulties with assessing home therapy. PEDIG concluded that a successful clinical trial would need broader eligibility requirements, better methods to assess home therapy and the ability to customize vision therapy based on patient data.[13]

Some studies have looked at perceptual activities with amblyopic children and adults. Perceptual learning studies in children show improvement in visual acuity and contrast sensitivity. Similar improvement was observed when comparing perceptual learning with patching in children. However, perceptual learning required less treatment time to achieve similar results.[14,15] Additionally, a growing number of studies suggest a substantial plasticity in the visual system of adults with amblyopia. Perceptual learning techniques with adults show improvement with visual detection, stereo information and contrast sensitivity function.[14-16]

Many case reports demonstrate that vision therapy works for treating amblyopia in adults and children.[17,18] While amblyopia treatment has a decreased effect for adults compared with children, documented case reports show improvement of vision and binocular function after treating adult amblyopes with vision therapy.[2,3]

The goals for amblyopia therapy should not be based solely on visual acuity, but also on the function of the binocular system. Amblyopia treatment should not be considered complete until the visual acuity of the amblyopic eye is normalized under binocular viewing conditions.[8] To achieve good binocular vision after amblyopia treatment has stopped, it is important to treat all aspects of the binocular system.[12] With this therapy approach in mind, practitioners can be confident their patients with amblyopia will receive the best care and achieve the best visual outcomes. ■

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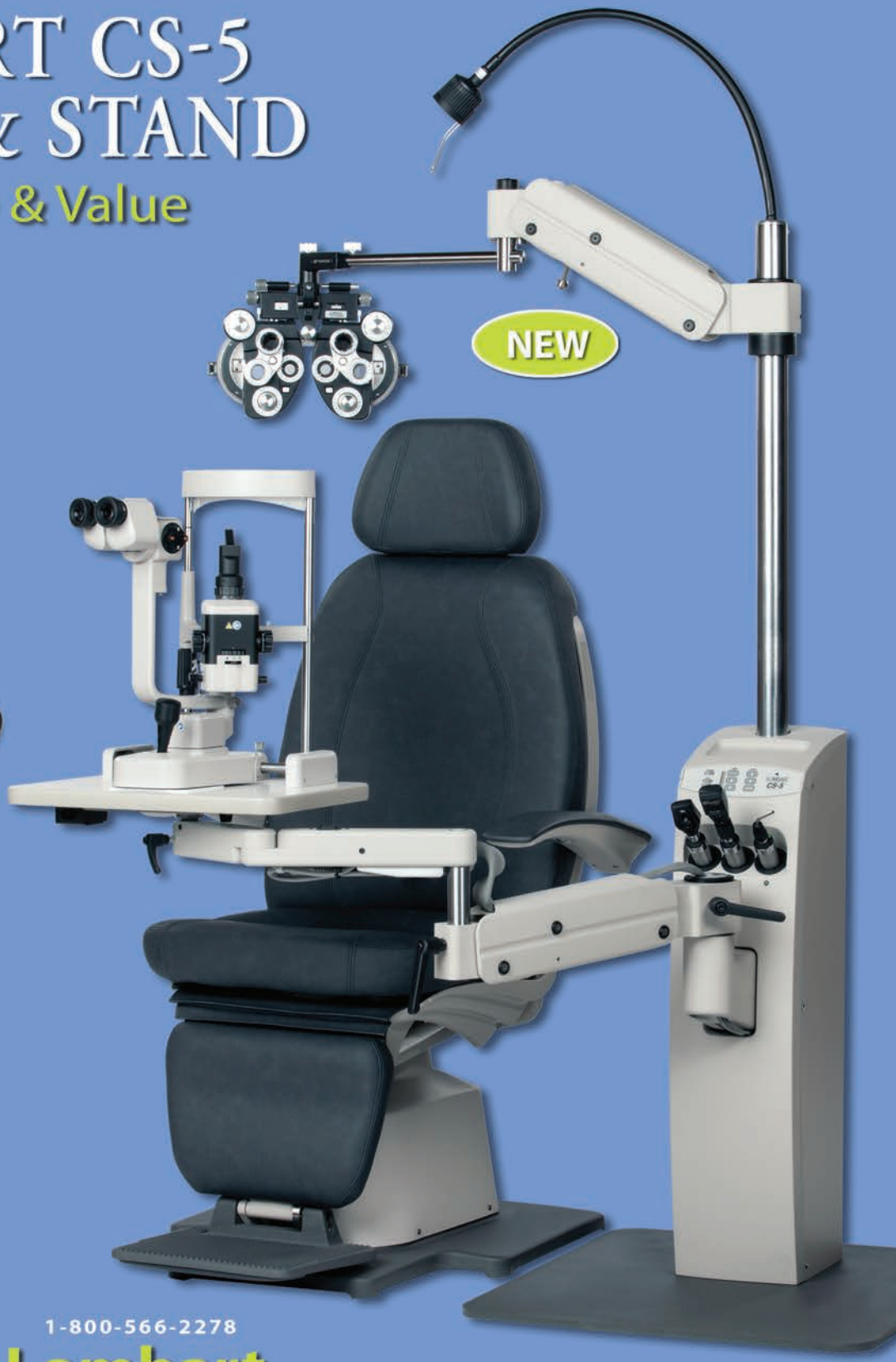

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KIDS AND PHARMACEUTICALS:

HOW TO TAILOR YOUR THERAPY

Treating infections in a pediatric population can be tricky. These tips and tricks can help. **By Rachel A. Coulter, OD, MSEd, and Julie Tyler, OD**

Optometrists are comfortable diagnosing and treating a variety of eye infections in adults, such as blepharitis, hordeola, conjunctivitis, keratitis and preseptal cellulitis. But when the patient is a child, it's a whole different story.

Pediatric patients—a catch-all term that includes infants (birth to up to one year), toddlers (ages one to three), preschoolers (three to five), grade-schoolers (five to 12) and adolescents (12 to 18)—are not simply miniature adults; they differ in their pharmacokinetic responses to medications.[1] Infants and toddlers, in particular, have the greatest differences from adults in drug absorption, distribution, metabolism and excretion, and as a child grows and matures, these differences evolve markedly.[2]

This article discusses managing eye infections in children and highlights how optometrists can tailor the therapy to match the needs of each pediatric patient.

Prescribing Challenges

For clinicians seeking information on appropriate prescribing for specific medications, the first resource should be the Pediatric Use section found in the manufacturer's drug insert.[3] Although randomized clinical trials to assess how infants and children respond to pharmaceuticals continue to lag behind trials assessing adults, drug inserts are more likely than in the past to include pertinent information, including the age for which the drug is approved, any associated side effects and outcomes.[4,5]

When pediatric prescribing information is not available, the Pediatric Use section will state, "Safety and effectiveness in children have not been established."[3,6] In these circumstances, off-label use may be necessary, which can be a significant problem in ocular care. In fact, research shows eye drops and dermatologic preparations are the most likely to be prescribed off-label in the

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Goal Statement: Pediatric patients differ from adults in their pharmacokinetic responses to medications, and as a child grows, these differences evolve markedly, making treatment with pharmaceuticals particularly challenging. This article discusses the many ocular infections common in the pediatric population, as well as how to select the proper pharmaceuticals and calculate pediatric dosing.
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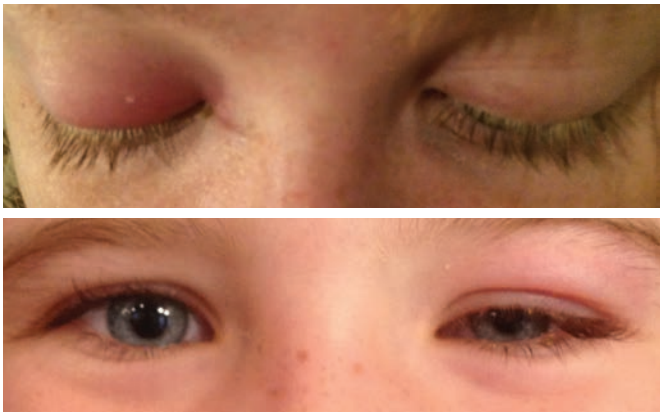
primary health care setting.[2] When practitioners discuss treatment options with the parent or guardian, they should include whether or not the drug has been approved for pediatric use, and document the decision-making process and the parent's/guardian's consent in the medical record.

When prescribing off label, optometrists should first check the package insert and FDA website for warnings or contraindications, as some commonly used drugs have serious side effects specific to pediatric patients. For example, doxycycline and other tetracycline analogs can cause dental enamel abnormalities in some children younger than eight.[7] Likewise, over-the-counter cough and cold medications are associated with an increased risk of respiratory depression, seizures and arrhythmias in children younger than four.[8]



Preseptal cellulitis, here in two different patients, can be caused by sinusitis, chalazia, hordolea, dacryocystitis, trauma or insect bites.

Determining Dosage

The best source for this task is the Dosage and Administration section in the package insert. When pediatric dosage information is not provided for topical ophthalmic drugs, researchers suggest clinicians use half the adult dose for children younger than two and two-thirds the adult dose for children ages two to three.[9,10] Often, clinicians decrease the frequency of dosing of ophthalmic preparations, as drops are difficult to "split."

Despite differences in pharmacokinetics and pharmacodynamics between children and adults, oral medications in the pediatric population are still adjusted according to body weight (mg/kg/day) or, in the case of chemotherapeutic drugs, according to body surface area (mg/m^2).[11] To establish the patient's weight, optometrists should weigh the patient in office and not rely on reported weight. Optometrists considering oral medications for ocular conditions in a pediatric patient—such as amoxicillin suspension for preseptal cellulitis—should take a four-step approach: (1) convert pounds to kg (1kg = 2.2lb), (2) calculate the dose in mg, (3) divide the dose by frequency and when needed for oral solutions/suspensions and (4) convert the mg/dose to mL.

Common Infections

Pediatric patients with an infectious condition might be unable or unlikely to report their symptoms. Consequently, diagnosis is often made based upon findings and the history reported by the parent or caregiver, including the condition's duration, laterality, the presence of pain and photophobia, eye discharge and any history of trauma.[12-14]

Optometrists can choose from a wide variety of antibiotics when managing bacterial eye infections in pediatric patients. Clinicians must consider the type of infection, severity and the age of the child to determine the best choice. Other factors that influence medication selection include the availability of the pharmaceutical in ointment or solution form, length of treatment, number of doses per day and the cost.[15]

Here are some prescribing considerations for several of the more common infections found in the pediatric population:

Blepharitis may be associated with bacterial infections with vessel telangiectasias and hard, fibrinous crusts and scales with occasional misdirected or missing lashes.[16] It most commonly presents in children between ages six and 10.[12] Treatment starts with lid hygiene, but topical and oral antibiotics are added when necessary to resolve severe and chronic cases.[17-19] Lid scrubs may include diluted baby shampoo applied to the lid margins or commercial wipes, including Systane lid wipes (Alcon) or Ocusoft lid scrubs, which are available in a "baby" formulation.[18] As *Staphylococcus* overgrowth is a major causative factor of lid inflammation, crusting and flaking, antibiotics particularly effective in reducing these bacteria are good options.[17,19] A good choice is ophthalmic erythromycin ointment applied one to two times a day for two weeks and discontinued when the condition improves. Because ointments cause blurry vision, applying them close to bedtime may improve compliance.

While AzaSite (azithromycin ophthalmic solution 1%, Akorn) is FDA approved for bacterial conjunctivitis, it has known benefits against lid disease as well. In addition to targeting *Staphylococcus*, it improves meibomian gland function by enhancing the immunomodulatory response.[19,20] AzaSite is typically dosed BID for two days OU and then changed to once a day for the next five days.[19,20] It has a good safety profile and is FDA-approved for children 12

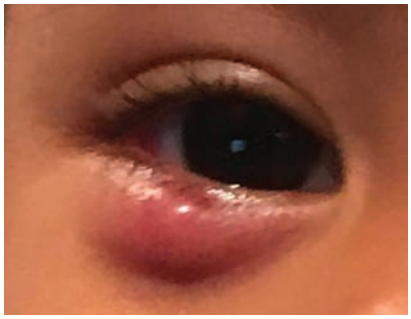


Photo: Christine W. Sindt, OD

Pediatric patients who present with localized swelling, pain and redness may have a hordeolum, as seen here.

months and older. Unfortunately, it is difficult to find in pharmacies and is often expensive.

In severe blepharitis, a topical antibiotic/steroid combination may eliminate inflammation and bacterial overgrowth.[21,22] However, side effects of corticosteroids include elevated intraocular pressure (IOP) and cataract formation, and the risk increases with chronic use.[17] In addition, herpes simplex virus (HSV) may be the culprit on rare occasions, and steroid treatment may worsen the condition and negatively impact the cornea.

Roughly 5% of chronic pediatric blepharitis cases progress to corneal involvement in the form of punctate erosions, punctate keratitis, phlyctenules, marginal keratitis or ulceration; in these cases, more aggressive management is required, which may include oral antibiotics or a topical fluoroquinolone with broad spectrum coverage such as besifloxacin.[22]

Topical antibiotic/steroid combinations in ointment form, including Tobradex (tobramycin and dexamethasone ophthalmic suspension, Alcon) or neomycin/polymyxin B/dexamethasone, may be easier to use in pediatric patients than an eye drop.[2] Clinicians should limit steroid use, and carefully consider the dosing frequency and risks/benefits prior use in children.

When an oral antibiotic is necessary for severe or chronic lid disease, oral azithromycin is a good choice, as it has both anti-inflammatory and antibacterial properties, and the oral formulation is approved for children six months of age and older.[20]

Hordeola, caused by infections of the eyelid glands, can cause localized swelling, pain and redness. Usually caused by *Staphylococcus aureus*, hordeola may initially be treated by applying a warm compress to the eye for 10 to 15 minutes, four times a day.[14] To help children keep the warm compress on the lid/face, the parent might challenge the child to "freeze" it in place using music as a timer. The "game" is to not remove it until the end of the song (or songs). While topical antibiotics generally are not used to treat hordeola, they may be necessary to prevent the spread of infection to other structures when drainage/expression is significant. On occasion, internal hordeola may progress and result in a secondary preseptal cellulitis that requires an oral antibiotic.

Conjunctivitis is a common eye disease, responsible for approximately 1% of all primary health care visits.[23] While infectious conjunctivitis may be bacterial or viral, most conjunctivitis in children is bacterial.[24] Bacterial conjunctivitis is typically self-limiting, resolving in one to two weeks in more than half of all cases.[23] Antibiotic treatment hastens resolution, decreases symptoms and reduces the risk of complications and disease transmission.[25-27]

The causative organisms of bacterial conjunctivitis in pediatrics are usually *Staphylococcus aureus*, *Staphylococcus epidermis*, *Streptococcus pneumoniae*, *Moraxella catarrhalis*, *Pseudomonas* and *Haemophilus*.[21] The most marked symptom is a sticky mucopurulent discharge that coats the eyelashes and causes the eyelids to stick together.

For milder presentations, the initial treatment choice is often a broad-spectrum antibiotic effective against both gram-negative and gram-positive organisms.[14] The most common treatment options include a combination of polymyxin B sulfate and trimethoprim sulfate ophthalmic solution, polymyxin B/bacitracin ophthalmic ointment and erythromycin 0.5%.[14] Clinicians may also use topical aminoglycosides, including gentamicin 0.3% and tobramycin 0.3% drops or ointment. However, aminoglycosides are not as effective against *Staphylococcus* and are associated with a corneal epithelial toxicity reaction after several days of use.[14] For infants, preschoolers and those who struggle with drop instillation, an ointment preparation may be helpful. Erythromycin is FDA-approved for use in newborns, while tobramycin is approved for use in children as young as two months of age.

Clinicians have several treatment options for more moderate presentations of bacterial conjunctivitis. AzaSite is effective against acute bacterial conjunctivitis, and research shows it is superior to tobramycin.[28] It is dosed one drop in the affected eye(s) twice daily, eight to 12 hours apart for the first two days and then once daily for the next five days.[29] This drop's viscous vehicle enhances contact with the eye and enables a caretaker to gently run "excess" drop along the lid margins to provide additional treatment to any infection of the eyelid.

A fluoroquinolone is another option for bacterial conjunctivitis, and an increasing number are FDA approved for use in children older than 12 months. Older generations include ciprofloxacin, ofloxacin, moxifloxacin and gatifloxacin. Ciprofloxacin 0.3% is also available in an ointment form and is approved for use in children two years and older. Dosing should be frequent

with severe cases, particularly during the first days, and the fluoroquinolone choice will determine the exact frequency. For example, ciprofloxacin 0.3% solution should be dosed every two hours while awake for the first two days and then every four hours for five days; moxifloxacin 0.5% solution is dosed two or three times per day for a week, depending on the solution vehicle.

A topical fluoroquinolone may also be a good choice for pediatric patients who are contact lens wearers because of the association between *Pseudomonas* infection and contact lens wear.[14] Children who have conjunctivitis and are contact lens wearers should immediately discontinue contact lens wear, discard their current contact lens case, if applicable, and not return to contact lens wear until the condition is resolved and no indication of discharge exists.

For severe cases of bacterial conjunctivitis, clinicians should consider a newer generation of fluoroquinolone such as Besivance (besifloxacin 0.6% ophthalmic suspension, Bausch + Lomb). Research shows this broad-spectrum antibiotic is safe and effective for children one year and older.[30] Studies also show this chlorofluoroquinolone is effective even against multi-drug resistant *Staphylococci*. Besivance is prescribed TID for seven days for bacterial conjunctivitis.[26]

Optometrists should see patients undergoing treatment for conjunctivitis every two to three days until signs and symptoms resolve. Bacterial conjunctivitis is quite contagious and is often transmitted through hand-to-eye contact.[31] Although cultures are expensive and usually are unnecessary, clinicians can consider gram stain, culture and drug sensitivity to clarify diagnosis and treatment in cases where a child is not improving or there is a concern about resistance.[28] Antibiotic resistance is not uncommon in pediatric conjunctivitis cases, particularly when the child has co-occurring otitis media.[32] Conjunctival discharge may contribute to the spread of antibiotic resistant bacteria.

Patient education regarding ocular hygiene is key.[33] Clinicians should remind parents and caregivers of common practices to reduce the transmission of bacteria:

- Avoid touching the eyes or eye area.
- Use disposable tissues and wipes instead of towels.
- Wash hands immediately after touching the eyes or eye area.
- Don't share towels, washcloths, dishes, cups or eating utensils.
- Play with hard surface toys that can be thoroughly cleaned.

Research shows these hygiene practices can reduce daycare absenteeism among children age three and younger.[34]

Patients may initially present to their primary care physician, which means that a non-eye care provider may make the initial diagnosis and initiate treatment. When a child has a persistently red eye, but no discharge or fever, optometrists should review the chosen treatment with the primary care physician. Gentamicin and neomycin, for example, may be associated with toxic reactions and red eyes that persist beyond the bacterial infection.[14]

Neonatal bacterial conjunctivitis, though not common in optometric practices, may be linked to *Neisseria gonorrhoeae* or other sexually transmitted diseases, which neonates may contract during the birth process. *Neisseria gonorrhoeae* results in a hyperacute bacterial conjunctivitis that develops quickly after birth and rapidly progresses. It is marked by copious, purulent discharge that quickly reforms after being wiped away. The condition requires medical comanagement with a neonatal specialist, ophthalmologist or pediatrician, in cases of hyperacute conjunctivitis or when systemic association is suspected. Aggressive treatment is required, as corneal perforation is a risk with *Neisseria gonorrhoeae* infection.[14] Treatment measures include irrigation of the eye with saline, systemic ceftriaxone (which may be IV in infants) and topical ciprofloxacin.[14]

A recent study compared research on the use of two different topical fluoroquinolones (besifloxacin 0.6% vs. gatifloxacin 0.3%) in 33 neonatal subjects with bacterial conjunctivitis. Clinically, both medications were safe, well tolerated and demonstrated high rates of clinical resolution with TID dosing for one week.[35]

Viral conjunctivitis is usually bilateral—with symptoms starting in one eye and progressing to the second. Infected patients present with red, watery eyes and sometimes small hemorrhages. In children with

Beyond the Prescription Pad

Besides prescribing, providing education and support is key to successful pediatric care:

- Consider family schedules when making treatment recommendations and reviewing the medication and dosing. Asking the caregiver to administer the medication at regular family activities such as breakfast, after school and bedtime may improve compliance.
- Educate the patient and caregiver on proper hygiene practices.
- Discuss the need to keep children out of daycare or school and complete any notes or documentation required regarding absences or reentry.
- When prescribing an oral medication, follow up with a phone call to check on the patient's status and review any concerns. These phone calls should be documented in the patient's record.
- Call or write to the child's pediatrician regarding the condition and any prescribed medication. This may help the overall care and compliance. Be sure to document this communication in the medical record.

a developed immune response, clinicians may see pre-auricular nodes. The most common form of viral conjunctivitis is caused by adenovirus, although HSV and varicella zoster can also be etiologies.[23] In some adenoviral conjunctivitis subtypes, a delayed corneal response of subepithelial infiltrates may occur; therefore, clinicians should emphasize patient and parent education regarding expected outcomes and the importance of follow-up.

Viral conjunctivitis is a self-limiting condition and symptoms usually decrease within the first week. Children should be kept out of school or daycare for several days because the condition is highly contagious.[31] Currently, no ophthalmic agents are approved for adenoviral infections. Moreover, use of an antibiotic does not hasten resolution and is not indicated, unless there is super-infection by bacteria. Clinicians should instruct parents and patients on general preventative hygiene measures and palliative measures such as cool compresses and artificial tears.

Microbial keratitis (MK) is relatively rare in the pediatric patient, with notable exceptions for children who experience ocular trauma or wear contact lenses.[36-38] Additional risk factors for MK include decreased immune system due to early development or an underlying systemic condition, chronic dacryocystitis, canaliculitis or blepharitis, or ocular trauma with a secondary opportunistic infection. Most aspects of a pediatric keratitis presentation will be similar to an adult presentation, including the presence of an epithelial defect—generally overlying a stromal infiltrate—with associated corneal edema and symptoms of blurred vision, pain, tearing and light sensitivity. In children, the most common bacteria responsible for keratitis varies by associated predisposing factor—with gram-positive microorganisms the main agents of infection in children unless associated with contact lens wear, in which case *Pseudomonas aeuriginosa* is most notable.[36-38] Other causative agents of keratitis include viral and fungal infections.

In all cases of keratitis, the goal is to remove the causative/contributing agents, provide anti-infective agents when available and initiate anti-inflammatory support. Removing insulting agents, such as contact lenses, is a key first step. Upon diagnosis, in-office management with a sterile saline lavage, followed by home lavage with saline, artificial tears or both can provide mechanical support and removal of infectious agents and toxic debris. Palliative treatment with cool compresses and possible cycloplegic agents is also helpful.

The common agents used to treat a presumed bacterial keratitis are fluoroquinolones, especially with history of soft contact lens wear. However, the treatment regimens are often off-label, even in adult populations, as only earlier generations have FDA approval and dosing regimens. Fortunately, several of these medications are approved for young children. Clinicians should also consider overnight coverage, and possibly daytime treatment in very young children, with an antibiotic ointment. Patients should be monitored daily until resolution, and if a specific antibiotic regimen is working for a child, clinicians should not alter it until corneal epithelial improvement is marked.

Viral keratitis with a primary corneal defect is most likely due to HSV. Patients may present with conjunctival injection, follicles and corneal staining with rose bengal of a classic dendrite or, early on, as discrete punctate defects. The two primary FDA-approved topical medications for HSV epithelial keratitis include trifluridine 1% drops—generally dosed up to nine times per day and with noted corneal toxicity over time—and Zirgan (ganciclovir ophthalmic gel 0.15%, Bausch + Lomb).[39] Zirgan has lower corneal toxicity, less frequent applications and a gel formulation that may be preferable in some pediatric populations.[39] Research has yet to establish its safety and efficacy in patients younger than age two.

Also, while corneal debridement may be considered in adults with HSV epithelial keratitis, it is not recommended in the pediatric population because the ability to achieve a precise debridement without damaging Bowman's layer in children is a significant challenge.

Fungal keratitis, in general, is difficult to manage because few effective anti-fungal agents exist and the condition is quite invasive.[40] If fungal keratitis is suspected based on history, lack of response to antibiotics, or development of a thickened, feathery, poorly defined corneal lesion, clinicians should consider comanagement with an ophthalmologist. The challenges of managing a fungal infection are amplified in pediatrics due to a dearth of information on anti-fungal agents in this population and, in

Avoid Common Dosing Errors

When determining dosage, the prescriber must identify the correct mg/kg/dose and frequency as well as apply the "maximum dose" ceiling when necessary. Proper use of decimals—often generated by weight-based dose calculations—is essential. An error in keeping place or a dangling zero may result in a dosage ten times greater than indicated.[1] Also, the abbreviation "d" may represent *day* (mg/kg/day) or *dose* (mg/kg/dose) and can cause confusion. Clinicians can include the mg/kg/dose and the patient's weight on prescriptions to allow pharmacists the opportunity to double-check the dosing.[1]

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some cases, a less well-developed natural immune response.

Preseptal cellulitis is an infection of the eyelid and periorbital tissue that is anterior to the orbital septum. Common signs are lid swelling and redness, often with fever and discharge.[41] The orbital septum is a thin, fibrous membrane that serves as a barrier to prevent deeper infection manifesting as orbital cellulitis that requires more aggressive treatment. When the infection involves tissue posterior to the orbital septum, the condition is diagnosed as orbital cellulitis. Signs and symptoms of orbital cellulitis include proptosis, decreased ocular motility, severe pain of the orbit, afferent pupillary defect, optic nerve head edema and decreased visual acuity. Differentiating orbital cellulitis from preseptal cellulitis is important, as orbital cellulitis is potentially a vision- or life-threatening condition.

Causes of preseptal cellulitis include sinusitis, chalazia, hordolea, dacryocystitis, trauma and insect bites.[41] Treatment requires oral antibiotics. Amoxicillin and Augmentin (amoxicillin/clavulanic acid, GlaxoSmithKline) are good options for children without a penicillin allergy.[42] Augmentin, which can be prescribed in several formulations and flavors, is prescribed at levels of 20mg/kg/day to 40mg/kg/day for no more than 10 days. Amoxicillin and Augmentin are approved for use in neonates and infants.[43]

Clinicians can consider prescribing trimethoprim/sulfamethoxazole when patients present with infections resistant to other antibiotics. Septra (trimethoprim/sulfamethoxazole, Monarch Pharmaceuticals) and Bactrim (trimethoprim/ sulfamethoxazole, Roche) are contraindicated for children younger than two months of age, as well as for patients with sickle cell disease or sulfa allergies.[42] Clinicians should note that use of either of these medications is associated with a higher risk of Stevens-Johnson syndrome.[42] Dosage depends on the type and severity of the infection.

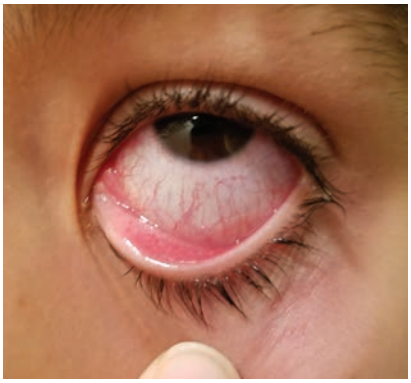

Photo: Alexandra Espejo, OD

This child has bacterial conjunctivitis in the left eye. On upgaze, note the injection as well as discharge around caruncle nasal. Papillae are visible on the lower lid. On downgaze, you can see injection of the bulbar conjunctiva and noticeable redness around the adnexa.

Because pediatric eye infections commonly present in primary care optometric practices, clinicians need to be aware of differences in selecting pharmaceuticals, dosing, patient education and treatment measures. Armed with the right information about prescribing for children, optometrists can create a pediatric following in their practice. ■

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Dr. Tyler is an associate professor and module chief at Nova Southeastern University. She serves as instructor of record for the course, Ocular Disease of the Anterior Segment.

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1. The Pediatric Use section of the package insert includes all of the following, except:
a. Ages for which the drug is approved.
b. Associated side effects.
c. Outcomes of studies and clinical trials.
d. Details on generic versions of the drug.

2. What are some advantages of using AzaSite (azithromycin ophthalmic solution 1%, Akorn) to treat blepharitis and conjunctivitis?
a. It's approved for neonates and infants.
b. It is inexpensive and available in a generic.
c. Dosing requires fewer instillations than other antibiotic agents.
d. It is usually available in most pharmacies.

3. What should an optometrist do when using a drug off-label?
a. Discuss treatment options with the parent or guardian, including whether or not the drug has approval for pediatric use.
b. Document the decision making process.
c. Indicate the parent's/guardian's consent in the medical record.
d. All of the above.

4. Why are doxycycline and other tetracycline analogs contraindicated in patients who are younger than eight years old?
a. They can cause aplastic anemia.
b. They can cause liver toxicity.
c. They can cause dental abnormalities.
d. They can cause dermatitis.

5. What is the best way to determine the pediatric dosage of any medication?
a. Use half the adult dose.
b. Refer to the manufacturer's package insert.
c. Use the same dosage as the adult version but in ointment form.
d. Calculate the dosage using Young's rule.

6. Which of these may be part of the first-line treatment for pediatric blepharitis?
a. Ophthalmic Ciloxan 0.3% ointment.
b. Tobramycin-dexamethasone ophthalmic ointment.
c. Systane lid wipes or Ocusoft lid scrubs.
d. Oral azithromycin adjusted for the child's weight.

7. The causative organisms of bacterial conjunctivitis are usually:
a. *E. coli*, *Staphylococcus aureus*, *Staphylococcus epidermis* and *Borrelia burgdorferi*.
b. *Streptococcus pneumoniae*, *Staphylococcus aureus*, *Staphylococcus epidermis*, *Haemophilus, Pseudomonas* and *Moraxella catarrhalis*.
c. *Clostridium*, *Moraxella catarrhalis*, *Helicobacter pylori* and *Yersinia pestis*.
d. *Pseudomonas*, *Haemophilus*, *Campylobacter* and *Pseudomonas aeruginosa*.

8. Which of the following drugs are available in ointment form and approved for use in children as young as two months of age?
a. Besifloxacin and gatifloxacin.
b. Ciprofloxacin and a combination of polymyxin B sulfate and trimethoprim.
c. Azithromycin and moxifloxacin.
d. Erythromycin and tobramycin.

9. Practitioners should culture and test for gram stain and drug sensitivity:
a. For all pediatric patients who present with an eye infection.
b. In cases in which a child is not improving or there is a concern about drug resistance.
c. For all pediatric patients who present with co-occurring otitis media.
d. In all cases in which the child presents with conjunctival discharge

10. Symptoms of localized swelling, pain and redness are associated with:
a. Viral conjunctivitis.
b. Blepharitis.
c. Hordeloa.
d. Bacterial conjunctivitis.

11. The mainstream method for adjusting topical ophthalmic drug dosing for children younger than two is based on:
a. Differences in pharmacokinetics.
b. Differences in pharmacodynamics.
c. Body weight (mg/kg/day).
d. Use half the adult dose.

12. To avoid errors, clinicians can ____ when prescribing oral medications for children:
a. Include the recommended mg/kg/d and the patient's weight on the prescription.
b. Prescribe all medications using the adjusted mg/kg/m^2.
c. Calculate the expected dosing based on converting pounds to mL.
d. Recommend two-thirds of an adult dose for all children older than eight years of age.

13. Commonly encountered eye infections in children include:
a. Orbital cellulitis, chalazion and HSV epithelial keratitis.
b. Blepharitis, preseptal cellulitis and conjunctivitis.
c. Pseudomonas keratitis, infectious scleritis and granulomatous uveitis.
d. Viral conjunctivitis, fungal keratitis and acanthamoeba.

14. All of these are contributing factors to the development of preseptal cellulitis, except:
a. Sinusitis.
b. Dacryocystitis.
c. Insect bites.
d. Scleritis.

15. Treating preseptal cellulitis requires oral medications that most commonly include:
a. Doxycycline 200mg/kg/day for 10 days.
b. Augmentin 20mg/kg/day to 40mg/kg/day for no more than 10 days.
c. Acyclovir 800mg/kg/day for seven days.

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d. Ibuprofen 80mg/kg/day to 320mg/kg/day for 10 days.

16. To keep a treatment on the lid/face when managing hordeola, children can:
a. Play the "freeze game"—keeping a compress in place using music as a timer.
b. Go to sleep with a "pirate patch" on the eye to limit the infection.
c. Play the "freeze game" by placing ice directly on the eye for one minute.
d. Apply topical antibiotic to the inside of an eye pad and place on the lid.

17. Which of these is associated with a higher risk of Stevens-Johnson syndrome?
a. Bactrim.
b. Augmentin.
c. Prednisone.
d. Acyclovir.

18. Some OTC cough and cold medications, used adjunctively with viral conjunctivitis, have been associated with increased risk of _____ for children younger than age four.
a. HSV epithelial keratitis and uveitis.
b. Respiratory depression, seizures and arrhythmias.
c. Bruising, drowsiness, and arrhythmias.
d. Respiratory depression, stroke and Bell's palsy.

19. Using the suggested modification guidelines, if Polytrim would be prescribed every three hours (6x/day) for an adult, a recommendation for a child younger than two years of age would be:
a. Roughly every five hours (4x/day).
b. Roughly every four hours (5x/day).
c. Roughly every six hours (3x/day).
d. Roughly every two hours (7x/day).

20. Corneal complications in about 5% of chronic blepharitis cases include:
a. Dendritic epithelial keratitis, papilloma or punctate erosions.
b. Marginal keratitis, filamentary ulceration or EBM disruptions.
c. Punctate keratitis, corneal guttata or dendritic epithelial defects.
d. Punctate erosions, marginal keratitis or ulceration.

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20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

Rate how well the activity supported your achievement of these learning objectives:
1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent

21. Improve my clinical ability to manage pediatric infections in my practice. ① ② ③ ④ ⑤
22. Become familiar the pharmaceuticals commonly prescribed to a pediatric population. ① ② ③ ④ ⑤
23. Better understand the challenges that accompany prescribing for pediatric patients. ① ② ③ ④ ⑤
24. Properly calculate dosages for pediatric patients in need of pharmaceutical treatment. ① ② ③ ④ ⑤
25. Better identify pediatric infections based on typical signs and symptoms. ① ② ③ ④ ⑤
26. Inform me of the proper treatment options for each of the various infections common to pediatric patients. ① ② ③ ④ ⑤

Rate the quality of the material provided:
1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

27. The content was evidence-based. ① ② ③ ④ ⑤
28. The content was balanced and free of bias. ① ② ③ ④ ⑤
29. The presentation was clear and effective. ① ② ③ ④ ⑤
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Weak in the Knees: Miller Fisher Syndrome

While rare, this Guillain-Barré variant has distinct clinical characteristics, and good outcomes often prevail. **By Betty Wang, OD, Jenette Cantrell, OD, and Stephanie Cali, OD**

Miller Fisher syndrome (MFS), a rare, self-limiting variant of Guillain-Barré syndrome (GBS), is an anti-GQ1bIgG antibody syndrome that affects the peripheral and central nervous systems.[1] An acute neuromuscular polyneuropathic condition, MFS causes an ascending paralysis with a classic presentation triad of ophthalmoplegia, ataxia and areflexia. Researchers first described the clinical triad in 1932.[1] Two decades later, Canadian stroke specialist Charles Miller Fisher was the first to publish a report in 1956 that described the clinical findings and defined the condition as a limited form of GBS.[2]

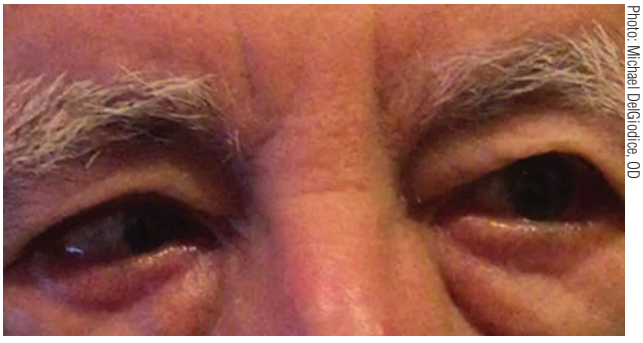

Photo: Michael DelGiodice, OD

Fig. 1. The differential diagnosis following baseline examination included a partial right CN VI palsy, which could indicate a vascular condition, as seen here in another patient.

Symptoms occur over several days, often in winter and spring, typically following a viral infection, and patients often experience diplopia as one of the first symptoms. When a patient presents with symptoms such as intermittent esotropia, impaired horizontal pursuits and fixed mydriatic pupils, and your neurological exam is abnormal, it's time to think of GBS, specifically MFS. The following case illustrates the relevant signs and symptoms and reflects the typical outcome.

History

A 27-year-old Caucasian male presented to the VA Eye Clinic during the spring months reporting acute onset binocular diplopia that started one day prior. The patient stated it was constant but only occurred at distance viewing with objects displaced horizontally. There was no eye pain or blurred vision associated with the diplopia. He also complained of recent dizziness and balance difficulties. He reported a sinus infection with significant paranasal congestion that started one week prior to the double vision. Previous ocular and medical histories were unremarkable; the patient was not currently taking any medications and did not have any medication allergies.

Diagnostic Data

On baseline examination, uncorrected visual acuities were 20/20-2 OD and 20/25-2 OS. Pupils were 5mm equally round and reactive to light with no afferent pupillary defect. Extraocular motilities were full, smooth and accurate in both eyes with no restrictions. Cover test at distance revealed an intermittent four to six prism diopter right esotropia in primary gaze occurring approximately 50% of the time. Performing cover test at near revealed a four prism diopter esophoria in primary gaze with no tropia.

While not undertaken in this case, clinicians should cover test in primary, right and left gaze. In patients with diplopia, cranial nerve six (CN VI) palsy will manifest as non- comitant deviation, worse in right or left gaze, depending on what side the palsy is on. A decompensating phoria will, on the other hand, show comitant deviation.

Refractive error was -0.50D sphere OD and -0.75D sphere OS and vision was correctable to 20/20 in each

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eye. Intraocular pressures were within normal limits in both eyes. Anterior and posterior segment findings were unremarkable in the right and left eyes. The optic nerves appeared healthy with a cup-to-disc ratio of 0.15 in both eyes.

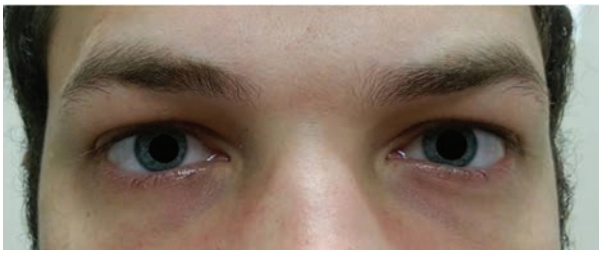

Fig. 2. The patient's pupils were dilated 8.5mm with no reactivity in the right or left eyes.

Differential diagnoses included a partial right CN VI palsy *(Figure 1)*, myasthenia gravis, a compressive process and a decompensating distance esophoria causing an intermittent right esotropia. The patient denied any previous history of binocular diplopia.

Imaging of the brain and orbits was ordered by the eye clinic to rule out brain involvement, including a compressive lesion. Computed tomography (CT) scans of the brain and orbits were performed and interpreted by the staff neuroradiologist. CT was chosen instead of magnetic resonance imaging (MRI) due to timing and availability; testing and results could be obtained the same day with CT in the clinic after hours. The results were unremarkable with no signs of intracranial hemorrhage, compressive lesion or acute infarction.

CT of the paranasal sinuses revealed moderate to severe chronic paranasal sinus disease and complete opacification of the right maxillary sinus. There was no extension into the orbit. The patient's family physician was informed of the patient's symptoms, eye exam findings and CT imaging results and started treatment for maxillary sinusitis with an oral azithromycin five-day dose pack and instructed the patient to follow up with the eye clinic.

The patient returned five days later after completing his oral azithromycin course and stated he experienced no improvement in the distance diplopia; in addition, he experienced the onset of significant photophobia and periocular pain around both eyes. The patient stated that his gait imbalance and walking difficulties were also worsening. On examination, best-corrected visual acuities remained 20/20 in the right and left eyes. Pupils were dilated at 8.5mm with no reactivity to light in either eye *(Figure 2)*. Extraocular motilities revealed lateral restriction with pain upon movement in the right eye and medial restriction with pain upon movement in the left eye consistent with a right gaze palsy *(Figure 3)*. Horizontal and vertical pursuits were saccadic with inaccurate and jerky movements. Anterior and posterior segment findings remained unremarkable OU.

Differential diagnoses after ophthalmologic examination included left internuclear ophthalmoplegia, right gaze palsy and right gaze palsy with left internuclear ophthalmoplegia, which could account for extraocular motility findings. Other differentials include a midbrain lesion or myasthenia gravis. Differential diagnoses for pupil dilation include third nerve (CN III) palsy, trauma and pharmacologic dilation. The patient did not have any other findings consistent with a third nerve palsy and denied trauma or exposure to pharmacological agents.

The patient was referred to neurology for further assessment. Neurological examination reported the patient was fully alert and oriented with no loss of facial sensation. The patient exhibited mild ataxia with difficulty walking in tandem and standing from a squatting position. Proximal weakness in both the upper and lower extremities and abnormal reflexes were present. All sensory modalities were intact. MRI and magnetic resonance angiography (MRA) of the brain and orbits were unremarkable. Cerebrospinal fluid protein was elevated at 75mg/dL (reference range: 15mg/dL to 45mg/dL) with no other abnormalities. Electromyogram testing was unremarkable and showed no abnormalities in nerve conduction from the spine to the feet and hands.

Diagnosis

The patient presented with acute symptoms of diplopia, ataxia and areflexia. Given the intermittent right esotropia, impaired horizontal pursuits, fixed mydriatic pupil, mild ataxia and abnormal reflexes on neurological exam and elevated cerebrospinal fluid protein, the diagnosis MFS was made. The patient was admitted to the hospital to monitor for any further complications. An eye patch was provided to the patient to cover the right eye as needed for diplopia.

No published diagnostic criteria for Miller Fisher syndrome currently exists. The diagnosis is usually made through the presentation of the clinical triad along with imaging and cerebrospinal fluid studies. MRI is typically unremarkable in the condition, although some studies have shown central lesions in the midbrain, pons and lower medulla. Electrophysiological studies show reduced or abnormal peripheral sensory conduction.[17]

Cerebrospinal fluid protein is often elevated with no other abnormal findings. In a landmark study, 64.4%

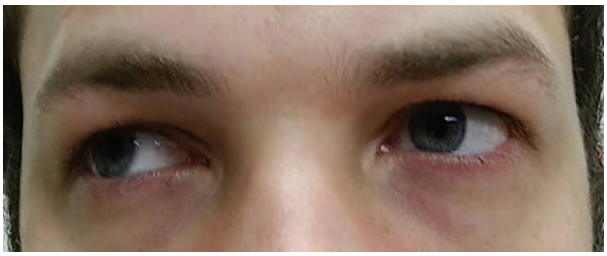

Fig. 3. Extraocular motilities revealed restriction in dextroversion.

of MFS patients showed elevated spinal fluid protein.[8] A positive anti-GQ1b IgG antibody test allows for a definitive diagnosis. In one study, anti-GQ1b IgG antibody was present in up to 95% of patients with the condition and absent in controls.[3]

The neurologist diagnosed the patient with MFS based on the clinical triad of ophthalmoplegia, ataxia and areflexia, an unremarkable MRI and MRA of the brain and elevated cerebrospinal fluid protein. Therefore, anti-GQ1b IgG antibody testing was not performed in this case.

Several clinical features of MFS are also seen in Guillain-Barré syndrome and Bickerstaff's brainstem encephalitis, making the diagnosis challenging. Patients with only Guillain-Barré present with limb weakness, sensory loss, cranial neuropathy and areflexia with no ophthalmological manifestations. Patients with Bickerstaff's brainstem encephalitis typically present with ophthalmoplegia, ataxia, hyper-reflexia and a disturbed consciousness. Since several symptoms and signs are present in all three conditions, anti-GQ1b IgG antibody titer testing is helpful for a more definitive diagnosis. Anti-GQ1b IgG antibody is positive in a large majority of Miller Fisher syndrome patients compared with 66% in Bickerstaff's brainstem encephalitis and 26% of Guillain-Barré syndrome patients.[16]

Treatment and Follow-up

The patient was evaluated the day after admission, when he stated he experienced no visual changes or improvement in balance. Binocular diplopia was still constant; therefore, the right eye remained patched. Eye pain with far gazes and fast eye movements were still present. On limited bedside examination, best-corrected visual acuities remained 20/20 in the right and left eyes. Pupils were dilated at 8.0mm in both eyes with minimal reactivity, which was slightly improved from the day prior. Color vision was normal in the right and left eyes. Extraocular motilities were full in all quadrants with pain still present on far horizontal

gazes. Horizontal pursuits remained saccadic with jerky movements and endpoint nystagmus. The optic nerves appeared healthy with distinct margins and a cup to disc ratio of 0.15 in both eyes.

Two days after, the patient stated an improvement in all symptoms and that his eyes "feel better today than they have been in a while." Eye pain and binocular diplopia were both resolved. Visual acuities remained 20/20 in the right and left eyes. Pupil sizes were 7.5mm in dark and 7.0mm in light with improved direct and consensual responses in both eyes. Extraocular motilities were full in all quadrants with no eye pain. Horizontal pursuits remained saccadic with jerky movements and endpoint nystagmus. Neurological exam revealed abnormal reflexes, which remained stable. The treatment options for MFS include plasmapheresis and intravenous (IV) immunoglobulin IgG, but these were withheld given the improvement in ophthalmoplegia and ataxia.

Four days later, the patient reported all symptoms were still improving, with some difficulty focusing and tracking while using the computer. Balance was 80% resolved and there were few occurrences of distance diplopia. Acuities remained 20/20 OU. Pupils were 5.5mm in dark and 4.0mm in light with 3+ direct and consensual responses OU. Extraocular motilities were full in all quadrants with no eye pain. Horizontal pursuits were improved, with no saccadic movements. Monocular accommodation was normal, which was measured 8.5 and 9 prism diopters OD and OS, respectively.

One week later, the patient reported his vision returned to near normal with no diplopia and mild difficulties focusing and tracking. Balance was 100% resolved. Visual acuities remained 20/20 in the right and left eyes. Pupil sizes were 5.5mm in dark, 4.0mm in light with 4+ direct and consensual responses OU. Extraocular motilities were full in all quadrants with no eye pain. Horizontal pursuits improved with no observed saccadic movements. Ocular health was unremarkable OU.

Treatment was withheld given the resolution of ophthalmoplegia and ataxia with stable areflexia. Full recovery of opthalmoplegia and ataxia took two weeks. Management included close monitoring and follow up in four weeks; the patient was advised to return sooner if any of his symptoms returned.

Just How Common is MFS?

Epidemiological data on Miller Fisher syndrome is limited. The incidence of MFS is quite rare, occurring in 0.09 per 100,000 people annually.[3] MFS occurs more commonly in patients with Guillain-Barré syndrome, from 3% to 25%.[4,6,7] This association occurs more in patients of Far East descent, suggesting a possible genetic component to MFS. The mean age of onset is 43, with a range of 13 to 78.[5] MFS affects males twice as much as females with a ratio of two to 1.03.[5] MFS occurs more in the winter and spring seasons.[5,6,8] The exact cause of the seasonal predilection has not been determined, but is likely associated with the post-infectious nature of the condition as bacterial and viral infections have been found to trigger an autoimmune response causing production of the anti-GQ1b IgG antibody in MFS.

Clinical Features

In well-described studies of MFS, all patients presented with the clinical triad of ophthalmoplegia, ataxia and areflexia.[4,5] Onset of symptoms typically occur over several days, and patients suffer a viral infection one to four weeks prior to the onset of clinical symptoms.[4,5] According to one study of 50 patients with the condition, the mean interval between infection onset and development of neurological symptoms is eight days.[4] Other signs of MFS include slurred speech, difficulty swallowing and an abnormal facial expression with an inability to smile or whistle.

Ophthalmological features. Practitioners should be on the lookout for mydriasis, lid retraction, acute angle closure and diplopia secondary to nerve palsies affecting CN III, IV and VI.[3,8,10,22,24] Diplopia is the most common ophthalmological symptom reported in many MFS studies.[8,16,25,26,27] Diplopia is also the initial symptom reported in 38.6% of 223 patients in one landmark study and 65% of 466 patients in a second study.[8,27] In the second study, 100% of patients exhibited external ophthalmoplegia, while internal ophthalmoplegia was present in 35% of patients.[27] External ophthalmoplegia refers to impairment of external extraocular muscles. Internal ophthalmoplegia refers to impairment of the pupillary sphincter and ciliary muscle. Complete ophthalmoplegia affects both external and internal muscles.

In a retrospective study of 19 patients with MFS, all presented with one or more nerve palsies affecting the extraocular muscles.[25] Other studies show MFS patients may experience multiple cranial nerve palsies, which can manifest bilaterally.[22,24,25] One common finding found among anti-GQ1b IgG positive disorders is abduction deficit consistent with a unilateral or bilateral CN VI palsy.[26,28] Internal ophthalmoplegia causes pupil mydriasis, where pupil constriction to light and/or near stimulation can range from minimal to absent.[8,26] In one report, mydriasis was present in 42% of patients. Involvement of CN VII occurred in approximately 45.7% of MFS patients causing facial

weakness, inability to smile or whistle.[8]

Ataxia. The cause of this clinical feature in MFS is not fully understood. Debate exists on whether ataxia is caused by dysfunction centrally in the cerebellum or peripherally. The first hypothesis, proposed by Dr. Fisher, postulated the involvement of Ia-afferent neurons.[2] This was supported by a second study decades later, which showed abnormalities of Ia-afferent fibers in MFS.[11] Abnormal peripheral nerve involvement has also been proposed with a mismatch between proprioceptive input from muscle spindles and kinesthetic information from joint receptors.[12]

There has also been supporting evidence that ataxia originates in the cerebellum. A predominance of anti-GQ1b IgG antibody in the cerebellum was found using immunocytochemical staining of the human cerebellum and further confirmed with western blot studies, which showed increased anti-GQ1b IgG antibody in MFS patients compared with control, although the mechanism for cerebellar involvement has not been studied.[13,14] In 2015, researchers published a case report of a MFS patient who underwent MRI. There was a reduced N-acetylaspartate/creatine ratio, suggesting cerebellar dysfunction, which returned to normal 2.5 months following recovery.[23]

Areflexia. This clinical feature, along with depression of deep tendon reflexes, is a sign of peripheral nervous system involvement in MFS. Electrophysiological studies have validated abnormal peripheral nerve conduction in GBS and MFS.[3] In one study, 81.6% of MFS cases presented with arefexia.[8] Another landmark study showed all patients experienced areflexia, which was still present six months after onset.[4]

Pathophysiology

The pathophysiology of Miller Fisher syndrome is not well understood; however, several hypotheses exist from immunological and histological studies. It is well known that the condition lies within the spectrum of anti-GQ1b IgG antibody syndromes along with Guillain-Barré syndrome and Bickerstaff's brainstem encephalitis.

The ganglioside GQ1b is a group of complex lipids involved in the central and peripheral nervous systems. GQ1b is a component of the plasma membrane structure in cranial nerves that supply the extraocular muscles and is involved in cell function at the presynaptic neuromuscular junction.

An autoimmune mechanism from a preceding triggering infection will produce the anti-GQ1b IgG antibody, which damages ganglioside GQ1b function,

causing demyelination. Histological studies have shown demyelination and axonal swelling in peripheral and oculomotor cranial nerves.[15] The anti-GQ1bIgG antibody is absent in healthy patients, but positive in over 90% of patients with MFS.[16,25]

Bacterial and viral infections have been found to trigger an autoimmune response, causing production of the anti-GQ 1b IgG antibody. The following infective agents that have been associated with MFS include mycoplasma pneumonia, HIV, *Campylobacter jejuni, Hemophilus influenza, Helicobacter pylori* and Epstein-Barr virus.[4,16] In one report that looked at 466 MFS patients, 90% had an antecedent illness, including upper respiratory infection, diarrhea or both.[27]

Prognosis and Treatment

Miller Fisher syndrome is a self-limiting disease and has an overall positive prognosis. Symptoms generally improve after a few weeks with full recovery typically occurring in two to three months. In one study, recovery started at a median of 13 days from symptom onset, and complete resolution of ophthalmoplegia and ataxia occurred in six months.[4,5] Relapses have been found to occur in less than 3% of cases.[4,5]

Although MFS is typically self-limiting, systemic complications have been associated with the condition. Upper respiratory infections have been found in 56% to 76% of MFS patients, which can progress to respiratory failure requiring mechanical ventilation.[4,18,19] Other rare serious complications include cardiomyopathy, lactic acidosis and coma.[3,20]

The treatment options for MFS include plasmapheresis, IV immunoglobulin IgG, and monitoring without treatment. Researchers postulate that plasmapheresis and IV IgG may be effective to speed resolution time since antibody Gq1b is IgG in class and the half-life of IgG is approximately 21 days, which is longer than the five to six day half-lives of IgM and IgA.[19] However, no randomized double-blinded placebo-controlled studies investigating these treatments have been conducted.

One retrospective study failed to show plasmapheresis changed the chance of full recovery and the time to resolve ataxia and ophthalmoplegia; further, the interval from onset to start of plasmapheresis treatment did not affect time to resolution or the severity of symptoms at onset. However, one case report indicates that plasmapheresis is indicated when profound ataxia, motor and respiratory impairment is present.[21]

Patients should be monitored closely for risk of developing serious systemic complications such as upper respiratory failure. Studies show no significant improvement in recovery time with IV IgG or plasmapheresis treatment in MFS, so monitoring may be a practitioner's best option.

While rare, the condition need not go undiagnosed. By recognizing the key clinical features, practitioners can reveal the cause of their patient's symptoms and gain confidence in a full recovery. ■

Drs. Wang and Cantrell are staff optometrists at the Orlando VA Medical Center. Dr. Cali is a staff optometrist at the Lee County VA Clinic. *The authors wish to thank Joseph Miller, OD, Paul Gruosso, OD, and Vanessa Santos, OD, for assistance with this manuscript.*

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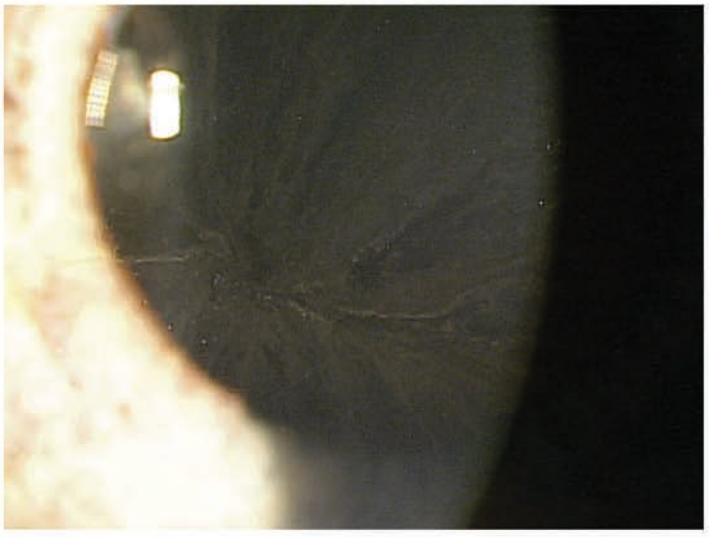

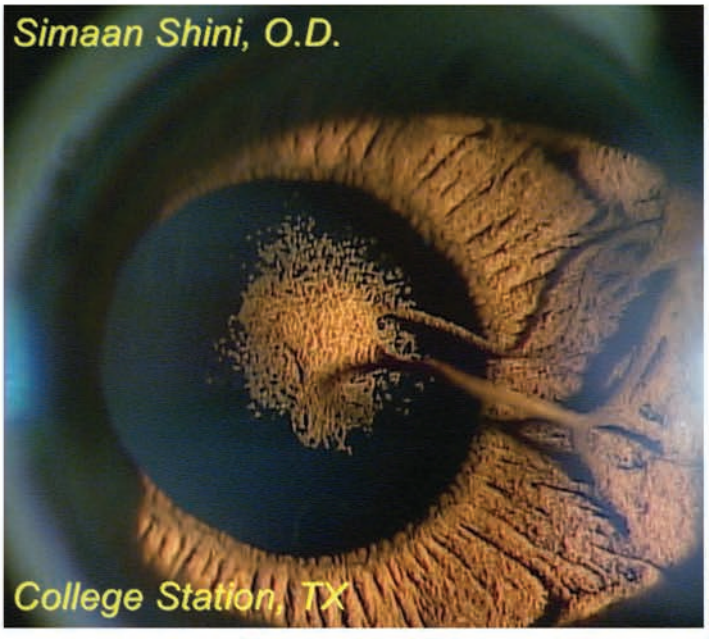

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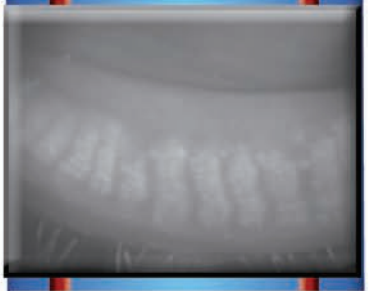

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A Sector-Specific Approach

This option can give practitioners more control when fitting sclerals–and improve some patients' success. **Edited by Joseph P. Shovlin, OD**

Q I have read that sector-specific scleral lenses are available.[1] Considering the need for a complete corneal vault and proper peripheral scleral landing when using these lenses, can you describe a situation in which they might make sense?

A "Scleral lenses can sometimes be modified in the optical zone or central zone, midperiperal/limbal zone and peripheral zones, depending on the manufacturer,"and these are known as sector-specific scleral lenses, says Stephanie L. Woo, OD, Scleral Lens Education Society vice president.

According to Jason Jedlicka, OD, clinical associate professor and chief of the Cornea and Contact Lens service at the University of Indiana, these designs help to better align the lens to the eye in cases where the corneal or scleral shape is highly irregular. "The 'sector-specific' aspect can be related to the cornea, to help normalize the vault in cases of high irregularity or to the scleral alignment zone, where the lens rests on the sclera," says Dr. Jedlicka. "A lens with poor scleral alignment will move with the blink, gather debris under the lens and cause visual issues due to the unstable lens position. In some instances, adding toricity is simply not adequate to resolve this, and a sector-specific landing zone should be used."

"One instance you might need a quadrant-specific design would be for a post-graft patient," says Dr. Woo. If the graft is tilted or elevated more in a certain area, that area may need more sagittal depth than the rest of the cornea, according to Dr. Woo. "For instance, if you have great clearance in all areas except the nasal area of the cornea, you could change the sagittal depth or base curve in that area of the lens to give you equal amounts of central clearance throughout the whole lens," she says.

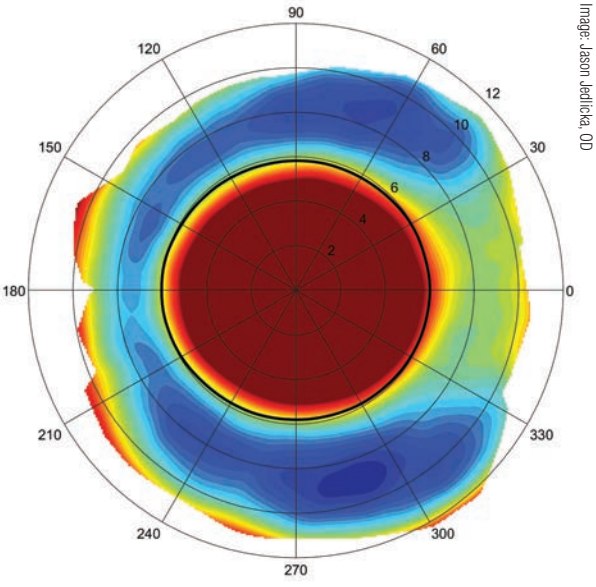

Image: Jason Jedlicka, OD

This scleral elevation map shows an asymmetric, atoric sclera, which can lead to poor scleral alignment with spherical or toric haptic lenses. In this case, a sector-specific lens may provide an improved fit.

Sector-specific scleral designs could also be helpful when dealing with the peripheral area of the scleral lens. "Perhaps the edge of the lens or landing zone is aligning perfectly with the entire sclera except the nasal portion, which has some impingement," says Dr. Woo. "The lab could modify just that quadrant of the lens to achieve better alignment."

Despite the various instances in which this technology can be beneficial, Dr. Jedlicka says that without a scleral topographer, "it is difficult to know when a sector-specific design is needed or would be helpful, and when it would require advanced fitting skills or troubleshooting to make the determination."

Who's Making Them?

Currently, a number of scleral labs offer sector-specific lens designs. According to Dr. Woo, many top companies offer this technology, but smaller labs may have sector-specific options available as well. If your lab doesn't list it on its website, try reaching out. "To find out if your scleral lens lab can fabricate a sector-specific scleral lens, I would suggest calling consultation and asking them," says Dr. Jedlicka. "Many may have the capability but do not necessarily advertise it." ■

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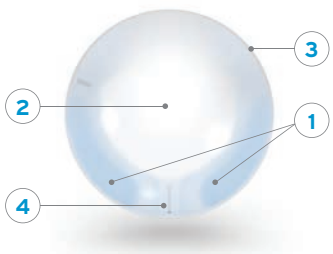

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Worm Your Way Out of This

What's lurking behind this young woman's vision loss?

By Angela Diamantakos, OD, and Mark T. Dunbar, OD

A 26-year-old Hispanic female, from the Dominican Republic, presented with a 12-year history of decreased vision in the right eye. She said the vision loss was painless and progressive. Other than the early decreased vision, her ocular and medical histories were unremarkable.

Her best-corrected visual acuities were counting fingers in the right eye and 20/20 in the left eye. Her pupils were equal and round and, although her left eye was reactive, her right had a sluggish response to light. We noted a strong afferent pupillary defect in the right eye. Ocular motility was full in both eyes. Confrontation visual fields were constricted in all fields in the right eye and full to finger counting in the left eye. Intraocular pressures were 15mm Hg OD and 13mm Hg OS. The patient had a 15 prism diopter constant exotropia of the right eye.

An anterior segment examination was unremarkable. Dilated fundus examination of the right eye revealed significant findings (*Figure 1*). A dilated fundus examination of the left eye revealed clear vitreous and a healthy optic nerve, macula and peripheral retina. An optical coherence tomography (OCT) scan of the macula was taken, as were fundus autofluorescence images (*Figures 2 and 3*).

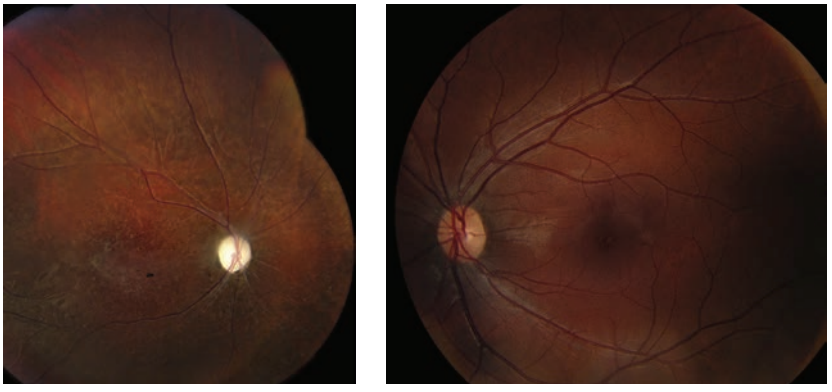

Fig. 1. Fundus photo of the right eye (at left) during the late stage with optic atrophy, retinal arterial narrowing and degenerative changes in the retina. The left eye appears normal.

Take the Retina Quiz

1. How would you describe the optic nerve?
a. Diffuse pallor.
b. Optic atrophy.
c. Advanced cupping.
d. Disc swelling.

2. What is the most likely diagnosis?
a. Unilateral retinitis pigmentosa.
b. Presumes ocular histoplasmosis.
c. Diffuse unilateral subacute neuroretinitis.
d. Post-traumatic chorioretinopathy.

3. What additional test would be helpful to confirm the diagnosis in the acute setting of this condition?
a. Fluorescein angiography.
b. Electroretinogram.
c. SD-OCT.
d. All of the above.

4. What is the most likely etiology?
a. Genetic mutation.
b. Nematode.
c. Trauma.
d. Fungus.

5. In which patients does this condition typically occur in?
a. Elderly.
b. Children.
c. Young adults.
d. B and C.

For answers, see page 112.

Diagnosis

We noted extensive optic atrophy in the patient's right eye in addition to a narrowing of the retinal arteries and pigmentary changes throughout the retina of that eye. The left eye was completely normal.

Given the patient's demographics, history of vision loss occurring at a young age and the appearance of the fundus, we diagnosised her with diffuse unilateral subacute neuroretinitis (DUSN). The patient presented to the clinic in the late stage of the disease, limiting treatment to observation.

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Discussion

DUSN, originally termed "unilateral wipe out syndrome," is a progressive ocular infection caused by various types of nematodes.[1] The most common nematodes that induces DUSN are *Ancylostom caninum, Baylisascaris procyonis* and *Toxocara canis*.[2] Infection results in persistent inflammation and degenerative changes in the retina. It typically occurs in otherwise healthy children and young adults with no significant ocular history. It usually affects only one eye, but a few bilateral cases have been reported.[3] It can lead to severe visual impairment and is a cause of blindness in children, especially in tropical areas, including the southeastern United States, the Caribbean and parts of South America. Additionally, cases have been reported in parts of Europe.[3]

Clinical presentation is divided into two stages, early and late. The early, or acute, phase of DUSN is characterized by loss of central vision, vitritis, vasculitis, optic nerve swelling and recurrent crops of evanescent, multifocal, white-yellow lesions at the level of the outer retina and retinal pigment epithelium (RPE).[4] Left untreated, the late phase of this condition results in severe vision loss, typically 20/200 or worse.[1] Findings in the late stage include optic atrophy (with subsequent afferent pupillary defect), narrowing of the retinal arteries and marked focal and diffuse degenerative changes in the retina and RPE.[2] Rarely, choroidal neovascularization can occur.

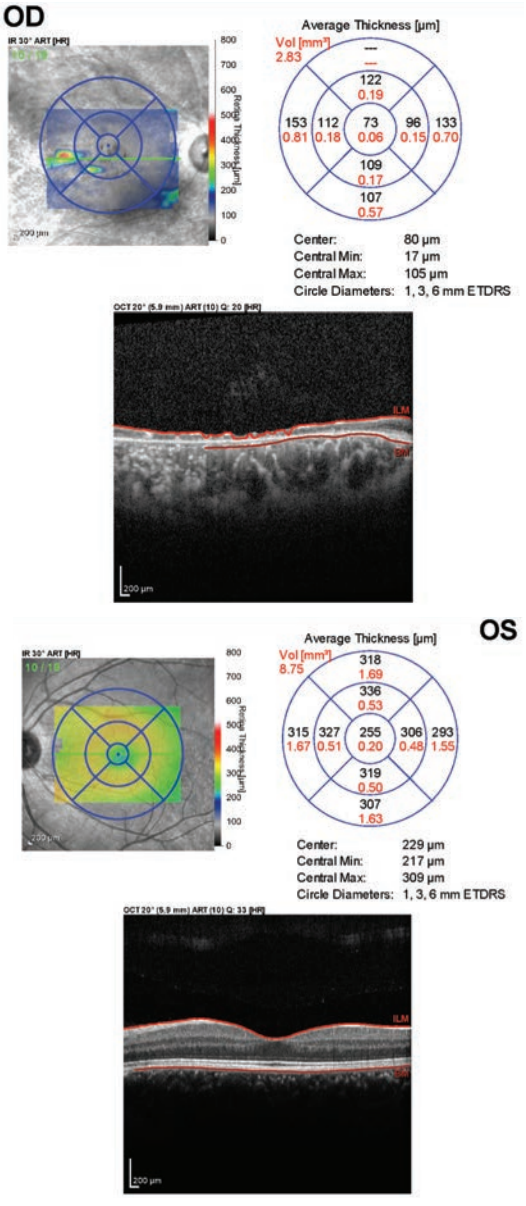

Fig. 2. This OCT of the macula of the right eye (above) shows severe atrophy of both inner and outer retina. The OCT of macula of the left shows a normal foveal contour.

The mechanism of dysfunction in this condition may involve a local toxic effect on the outer retina by the nematode itself, as well as a diffuse inflammatory and toxic reaction affecting both the inner and outer retina.[1] Definitive diagnosis is made by actual visualization of the nematode in the subretinal space. This only occurs in about 25% to 30% of patients.[3] Differential diagnoses in cases where the worm is not visualized include retinal sarcoidosis, syphilic chorioretinitis, toxoplasmosis, presumed ocular histoplasmosis, multifocal posterior placoid pigment epitheliopathy and multiple evanescent white dot syndrome in the early stage.[3]

In the late stage, differentials include retinitis pigmentosa, post-traumatic chorioretinopathy and occlusive vascular disease.[3]

Monitoring and Treatment

Fluorescein angiography can be useful in the early stages of DUSN. It can reveal hypofluorescence of the retinal lesions followed by late staining.[1] Leakage can be seen around the optic disc. Serologic testing, stool examinations and peripheral blood smears are of little value in diagnosing DUSN.[2] Electroretinography (ERG) can also be performed in these patients. Most patients with DUSN have an abnormal ERG, even when tested early in the disease course.[2] Changes commonly seen in the ERG are a decrease in rod and cone function, with a B-wave being more affected than the A-wave.[4] OCT may also be useful in assessing patients with DUSN. It is common to find a decrease in the retinal nerve fiber layer as well as the central macular thickness.[4] Disruptions of both the inner and outer retinal layers can be observed. Additionally, it may be useful to help identify the location of the worm.

When visible, treatment of

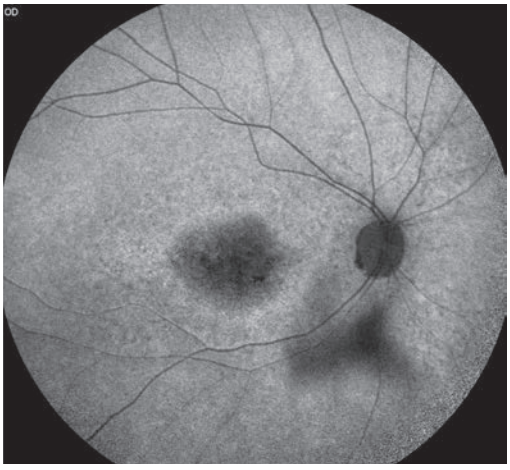

Fig. 3. This fundus autofluorescence of the right eye shows areas of hypofluorescence corresponding to the areas of RPE atrophy seen clinically.

When visible, treatment of choice is the eradication of the parasite with laser photocoagulation.[1] The worm is usually found near the retinal lesions. In patients where it cannot be identified, high-dose oral anti-helminthic therapy, such as albendazole, is recommended. Oral corticosteroids may help decrease the inflammatory response. Researchers also believe that laser photocoagulation adjacent to the retinal lesions may aid in ocular penetration of albendazole by disrupting the blood-retina barrier.[3] Some improvement in visual acuity is expected in these patients following laser photocoagulation. In cases presenting during the late stage of this disease, vision loss is irreversible and does not improve with medical or surgical treatments.

Our patient was made aware of the diagnosis. Unfortunately, she would not benefit from any treatment as damage to the retina and optic nerve has already occurred and cannot be recovered. We did recommend that she take monocular precautions and stressed the importance of wearing protective eyewear. ■

Dr. Diamantakos is a resident at Bascom Palmer Eye Institute.

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All for Nothing... and Everything

Just how much testing is necessary for a glaucoma suspect in need? All you can offer.

By James L. Fanelli, OD

In August, a 62-year-old black female was referred to our office for evaluation of her eyes, by a well-respected diabetes clinic that treats patients who cannot afford care and have no insurance or means to pay for services. It's also known for encouraging timely eye care.

The patient reported that she had noticed difficulty with her distance and near vision since losing her glasses approximately six weeks earlier. She reported that her last visit to an eye care provider was four years earlier. Her medications included Januvia (sitagliptin), Tylenol as needed and a hypertensive medication that she takes once daily. She reported no allergies to medications.

The patient informed us that her primary care doctor was "not happy" with her A1c level obtained in June, but she did not know that value. She does not self-monitor with finger-stick glucose readings. She mentioned also that her doctor was considering starting her on insulin, which tells me that there was not consistent and adequate control of her glucose levels. She was not sure of exactly when she began diabetes medication, nor what her pretreatment glucose levels were, but she said that she thought she had been diagnosed about 10 years earlier.

Examination

Uncorrected distance acuities were 20/60- OD and 20/50 OS, pinholing to 20/40 OD and OS. Best-corrected visual acuities through hyperopic astigmatic correction was 20/40 OD, OS, and 20/40+ OU. Pupils were equal, round and reactive to both light and accommodation, with no afferent pupillary defect noted.

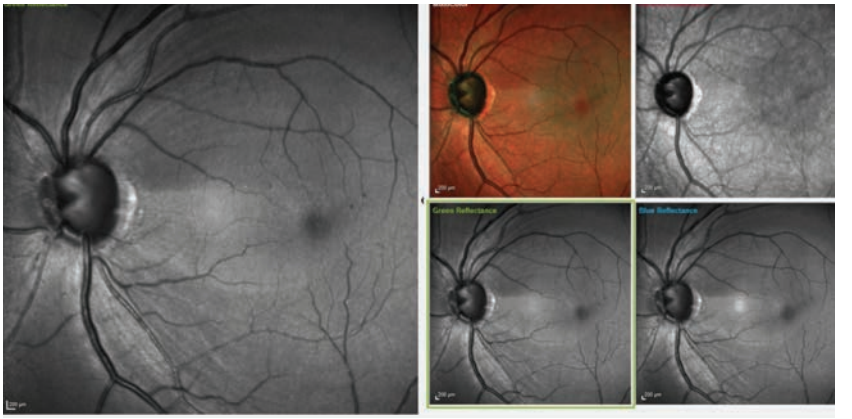

Fig. 1. These images show the significant RNFL wedge defect located between the one and two o'clock positions, as well as a diffuse RNFL loss in the four to five o'clock positions. Microaneurysms are also visible perifoveally.

A slit lamp exam of her anterior segments was unremarkable, with no evidence of iris neovascularization. Her anterior chamber angles were open. Applanation tensions were 20mm Hg OD and 23mm Hg OS at 2:30pm.

The patient was dilated with phenylephrine and tropicamide. Through dilated pupils, her crystalline lenses were characterized by mild cortical and nuclear cataracts, and there were bilateral early posterior subcapsular cataracts. I estimated that these lenticular changes accounted for a best-corrected visual acuity of about 20/30 OD and OS.

Our team noted bilateral posterior vitreous separations. Her cup-to-disc measurements were estimated at 0.7 x 0.7 OD and 0.75 x 0.85 OS. A retinal nerve fiber layer (RNFL) wedge defect was visible in her left eye consistent with glaucomatous damage. Her maculae were characterized by scattered microaneurysms, greater in the left eye than the right, with no discernible leak observed at the slit lamp, and the maculopathy did not meet ETDRS guidelines.

Retinal vasculature was characterized by mild arteriolar attenuation and mild crossing changes in both eyes. Her mid-peripheral retinal evaluation was characterized by scattered, and minimal, intraretinal hemorrhages consistent with mild non-proliferative diabetic retinopathy. Her peripheral retinal evaluations were unremarkable.

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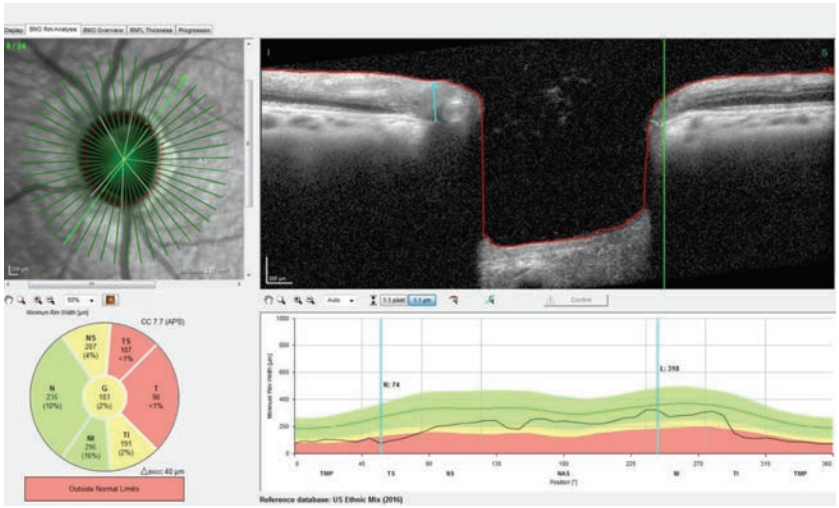


Fig. 2. You can see the thinned superotemporal Bruch's membrane opening radial scan with minimal rim tissue thickness in this scan at 74µm, well outside the reference database for normals.

Since this was our first visit together, and diabetic maculopathy and uncontrolled glaucoma were noted, I chose to image her optic nerves and maculae with multicolor imaging as well as OCT that consists of: three diameter circle RNFL scans, Bruch's membrane opening (BMO) radial scans and macular scans with segmented retinal layers.

As she will need further evaluation, I scheduled a follow up visit in a couple of weeks, for threshold 24-2 visual fields, pachymetry, gonioscopy and Heidelberg Retina Tomograph (HRT 3) scanning of the optic nerves.

Imaging Results

On multimodal laser imaging of the left posterior pole, not only was a rather large wedge defect seen in the superior portion of the papillomacular bundle and arcuate region, but diffuse retinal ganglion cell loss was also noted inferiorly in the left eye (*Figure 1*). Superior temporal BMO in the left eye demonstrated a minimum rim width (MRW) of only 74µm in the area of the wedge defect (*Figure 2*).

The RNFL circle scans of that eye demonstrated a statistically significant aberration in the temporal and inferotemporal Garway-Heath sectors in the 3.5mm and 4.1mm RNFL circle scans.

Macular OCT scans of both eyes demonstrated ganglion cell layer defects in the areas consistent with the multicolor images and the wedge defect and RNFL diffuse defects seen clinically (*Figure 3*). In essence, all the pieces of the puzzle fit together perfectly.

Diagnosis

At the completion of this first visit, I determined that the patient had mild nonproliferative diabetic retinopathy, no clinically significant diabetic macular edema and uncontrolled glaucoma in both eyes, but more so in the left than the right.

She presented for follow up as scheduled. At that visit, visual field studies demonstrated field defects consistent with the clinical picture seen at the previous visit, with above and below arcuate defects in her left eye encroaching upon fixation.

Her pachymetry readings were 530µm in the right eye and 535µm in the left. Applanation tensions at this follow up visit were 21mm Hg OD and 22mm Hg OS at 2:45pm. HRT 3 studies confirmed the clinically estimated cup-to-disc ratios and correlated well with the OCT BMO profiles obtained earlier in both eyes. A gonioscopic exam

demonstrated open angles, with a flat iris approach and moderate trabecular pigmentation in both eyes.

After the visit, the patient was educated about a firm diagnosis of open-angle glaucoma, and a sample of Travatan Z was given to her with instructions on drop instillation and directions to use one drop in both eyes at bedtime. As of this writing, she is scheduled for follow up in two weeks to assess the efficacy of this medication as well as tolerability.

First, Do No Harm

So, what's the catch here? This seems like a straightforward case of a new patient presenting with undiagnosed glaucoma, who also happens to have diabetes with mild retinopathic findings. The patient was imaged using the latest technology and software, underwent threshold visual fields, and findings from the structure, function and clinical exams all added up and made sense. She was exhaustively tested to confirm what I suspected from the first visit: this patient has glaucoma that needs to be treated. Both visits demonstrate straightforward, textbook findings, imaging and management.

But this case contained one key difference.

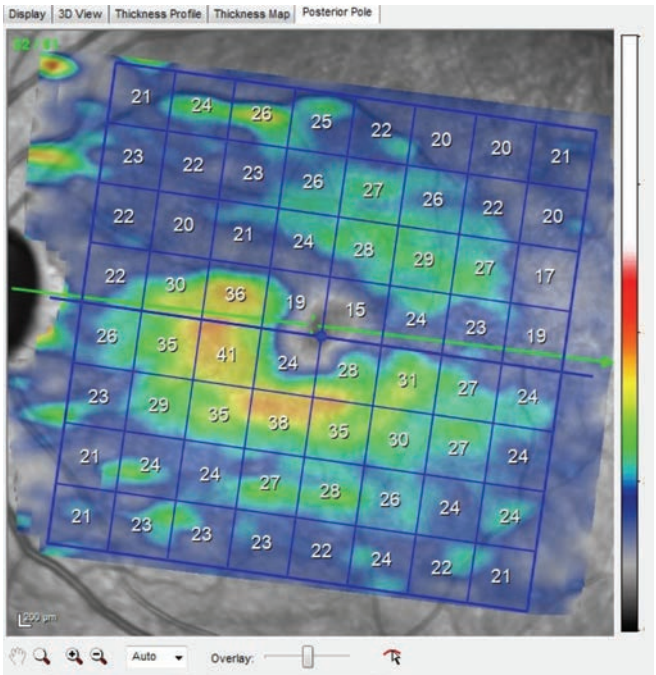


Fig. 3. Note the thinned ganglion cell layer above the horizontal meridian that matches perfectly with the wedge defect seen in the multicolor image shown earlier. This macular ganglion cell layer thinning also matches up to the thinned superotemporal BMO scan also shown earlier.

The patient was referred from a clinic in town that sees patients who fall through the cracks. We've all seen patients like these where we comp our services, in the interest of providing good care to all people, independent of whether or not we get paid. I'm no hero for seeing this patient; every doctor does some measure of this as circumstances allow. Sometimes doctors offer patients in these situations services, to be sure—but with a reduced level of care. With all the technology available to us, we could have skipped many of the studies that we typically run our glaucoma patients through. We certainly have tests that could have been eliminated in this case that would not have affected her ultimate diagnosis. But to what end? To save a few bucks? To save some time? Although the patient could not pay for her services, she still deserves the best care I can provide. No need to shortchange her.

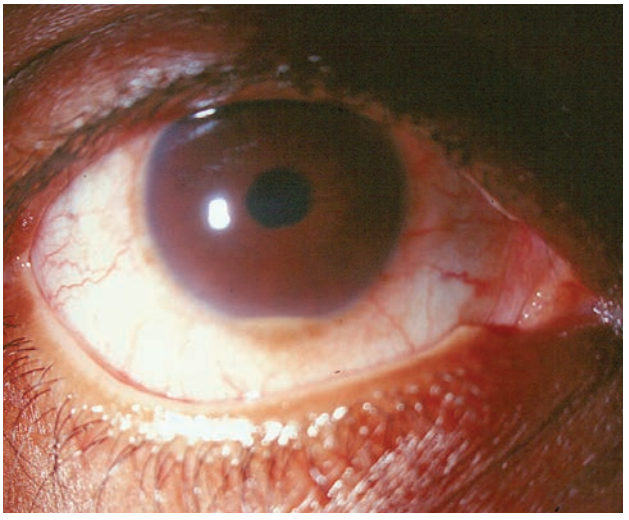
Do what is right. We did it all, we got paid nothing, but, by going all-out, we both have everything to gain. ■


Master Uveitis Prescribing

We have numerous options, but cost, efficacy and access can vary considerably.

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

Two recent patients with anterior uveitis underscore new considerations for managing this condition. The first was a 36-year-old man who has had repeated bouts of anterior uveitis in his right eye. Each episode was mild to moderate in intensity, and he responds well to topical steroids and cycloplegia. Well-versed in his symptoms, he readily self-treats with a steroid if he cannot immediately get into the office to be seen. However, he was troubled by the recurrent nature of his condition, which flares up every three to four months. He has had detailed medical evaluations with a rheumatologist who has yet to fund an underlying cause. In an effort to reduce recurrence, his rheumatologist put him on the immunosuppressant agent methotrexate. His rheumatologist communicated that he wanted to ensure there was no clinical evidence of an infectious etiology and to be alerted if the uveitis flared again while the patient was on methotrexate so that he could have justification to put the patient on Humira (adalimumab injection, Abbvie).



This 36-year-old patient was troubled about his recurrent hypopyon uveitis flare ups.

The second patient is a 54-year-old man who had been originally referred for evaluation of elevated intraocular pressure (IOP). He had not gotten around to scheduling that appointment, but when he developed a red, increasingly painful left eye, he presented on an emergent basis. He was extremely photophobic and manifested a grade 3 bulbar injection with circumlimbal flush. He also complained of hazy vision in his left eye, though the visual acuity was 20/20 OU. He had a moderate anterior chamber reaction in the left eye with a significant level of flare. No vitritis was seen. He was diagnosed with acute anterior uveitis of the left eye and prescribed generic prednisolone acetate Q1H and atropine BID. He returned for his scheduled follow up and was feeling much better. His ocular redness had disappeared and anterior chamber reaction much dissipated.

He reported that the two medications gave him significant relief on the first day. When asked, he reported that these two generic medications, prednisolone and atropine, cost him $60 and $121, respectively, without insurance.

The Basics

Uveitis is most commonly encountered in those between 20 and 60 years old.[1,2] Patients typically complain of pain, photophobia and hyperlacrimation. The pain is characteristically described as a deep, dull ache, which may extend to the surrounding orbit. Associated sensitivity to light may be severe, and often these patients will present wearing dark sunglasses. In the earliest stages of anterior uveitis, visual acuity is minimally compromised. Often, patients report that their vision is 'hazy' or 'smoky' and this represents protein in the anterior chamber (flare). Should the condition persist, or initial treatment delayed, accumulation of cellular debris in the anterior chamber and along the corneal and lenticular surfaces may result in subjectively blurred vision.[3,4]

Clinical inspection of uveitis patients will reveal a deep perilimbal injection of the conjunctiva and episclera, although the palpebral conjunctiva will remain unaffected. The cornea will display mild stromal edema upon biomicroscopy and, in more severe or protracted reactions, keratic precipitates may be noted on the endothelium. The hallmark

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signs of non-granulomatous anterior uveitis are cells and flare. Cells represent leukocytes liberated from the iris vasculature in response to inflammation and are observable and freely floating in the convection currents of the aqueous. When present, flare gives the aqueous a particulate, or smoky, appearance. When the inflammation is profound, white blood cells will settle, creating what is known as hypopyon uveitis. Whenever there are sufficient cells in the anterior chamber, convection currents can carry some cells behind the iris into the anterior vitreous. This is termed "spillover" and must be differentiated from an intermediate or posterior uveitis.

Numerous etiologies may induce uveitis, ranging from blunt trauma to widespread systemic infection (e.g., tuberculosis) to generalized ischemic disorders (e.g., giant cell arteritis).[5-10] Some other well-known systemic etiologies include ankylosing spondylitis, rheumatoid arthritis, psoriatic arthritis, juvenile idiopathic arthritis, sarcoidosis, systemic lupus, Behçet's disease, inflammatory bowel syndrome, multiple sclerosis, syphilis, Lyme disease, histoplasmosis and herpetic diseases.[5-10]

Management

The primary goals in managing anterior uveitis are to: (1) immobilize the iris and ciliary body to decrease pain and prevent exacerbation of the condition; and (2) quell the inflammatory response to avert detrimental sequelae such as maculopathy, cataracts and glaucoma. Cycloplegia is a crucial step in achieving the first goal. Unfortunately, options today are severely limited. With the outright discontinuation of the intermediate potency cycloplegic scopolamine and homatropine not stocked in most pharmacies (obtained through special order in one to two days), practitioners now only have two readily available options: Cyclogyl (cyclopentolate 1%, Alcon) and atropine 1%. Cyclogyl is best used for cycloplegic refractions due to its mild action and short duration; however, it is typically not potent enough to achieve adequate cycloplegia in the inflamed eye and should be avoided.

That only leaves atropine 1%. Many practitioners shy away from this cycloplegic due to its long duration of action. Indeed, in a healthy eye, a drop of atropine will cause mydriasis and cycloplegia for a week or longer. However, in an inflamed eye, this medication needs to be dosed at BID to TID to achieve an adequate therapeutic response.[11] Today, practitioners are often forced to choose between a medication which may be too weak and one that may be too strong. We advocate choosing atropine to treat uveitis. Alternately, in-office administration of atropine 1% may give adequate cycloplegia until the patient can obtain homatropine 5%.

Steroids

Numerous steroids can treat anterior uveitis. The most important consideration in choosing which steroid to use is identifying one with the proper potency. Fluorometholone steroids typically are not potent enough for patients with anterior uveitis. Lotemax (loteprednol 0.5%, Bausch + Lomb) is an acceptably potent steroid with a lower propensity (though not negligible) to elevate IOP. For many years, the most commonly used steroid for uveitis management was Pred Forte (prednisolone acetate 1%, Allergan). Cost and insurance often dictate that we use generic prednisolone acetate 1%, which is perfectly acceptable in most cases.

Topical corticosteroids should be administered in a commensurate fashion with the severity of the inflammatory response. In pronounced cases, dosing every 15 to 30 minutes may be appropriate; however, at minimum, steroids should be instilled every three to four hours initially.

In recent years, however, many clinicians have recognized the usefulness of Durezol (difluprednate 0.05%, Alcon) in controlling anterior uveitis.[12-14] Clinical trials show that difluprednate 0.05% can be dosed at roughly half the frequency as prednisolone acetate 1% while achieving the same clinical efficacy.[22,23] Our successes have made this medication one of our favorite steroids when managing anterior uveitis. We have seen difluprednate manage uveitic conditions that previously failed on other steroids, including Pred Forte.

Recalcitrant cases of anterior uveitis that are unresponsive to conventional therapy may necessitate the use of systemic immunosuppressants such as cyclophosphamide, methotrexate, azathioprine, mycophenolate mofetil, cyclosporine, tacrolimus, interferon or infliximab.[15]

Only well-trained clinicians who are able to manage complications should prescribe these. In any other case, comanagement with a rheumatologist or internist is a must.

What is Humira?

Humira is a tumor necrosis factor (TNF)-α blocker. TNF-α is a specific source of inflammation that appears to have a role in uveitis. By blocking it, the inflammatory effect of uveitis is reduced. Uveitis (specifically noninfectious intermediate and posterior uveitis and panuveitis) is the 10th indication for Humira, joining a list that includes rheumatoid arthritis, juvenile idiopathic arthritis, psoriatic

arthritis, ankylosing spondylitis, adult Crohn's disease, pediatric Crohn's disease, ulcerative colitis, plaque psoriasis and hidradenitis suppurativa. It appears that rheumatologists are also broadly applying the uveitis indication to anterior uveitis as well.

Cost

Anterior uveitis can be an expensive disease to treat. Branded steroids can run from approximately $130 to $213 per bottle. Whenever possible, we typically use generic medications, but this does not always guarantee an affordable option. Against recommendations, the second patient presented here unfortunately went to the most convenient but expensive pharmacy and ended up paying $180 for two generic medications, whereas he could have had both for about $40 had he gone elsewhere. It behooves practitioners to help patients find the best drug prices and discount coupons using any number of online drug pricing sites. In the case of Humira, rheumatologists need evidence of failure on another immunosuppressant in order to get the medication approved by insurance. Otherwise, Humira therapy will cost upwards of $4,500 per month. ■

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Is That an IOL on Your Retina?

Lenses can dislocate for any number of reasons, and patients will need your expertise to see them through the complicated fix.

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

There are numerous reasons why an intraocular lens (IOL) can drop into the posterior chamber during or after cataract surgery. Weak zonules or a history of trauma are two likely causes, but we see this complication in completely unanticipated cases as well. Unfortunately, the fix is rarely simple. These four steps can help you identify the complication and guide patients through the process.

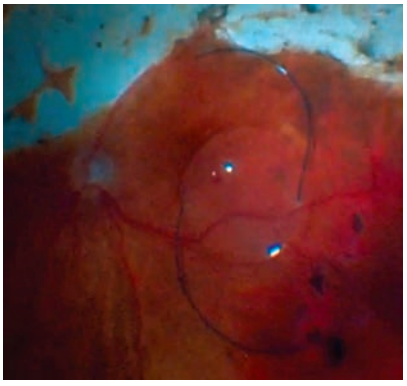
Step One: Find the Problem

Symptoms of a dislocated IOL include a sudden decrease in vision, distorted vision or flashes of light. You will likely also notice a large hyperopic prescription during autorefraction.

Any sudden change in vision should prompt dilation because the anterior chamber can often look normal with the lens behind the iris. Unless the patient is dilated, the dislodged lens is easy to miss. It will most likely settle on the inferior retina, but a careful 360-degree funduscopic examination is necessary to rule out a posteriorly dislocated IOL. Patients often look fine superficially and have minimal intraocular inflammation.

Note: If this is the first time you are seeing a patient, the absence of an IOL does not always mean something is wrong; there are several situations during which the surgeon may elect to leave a patient aphakic.

To see a video of this procedure, visit www.reviewofoptometry.com, or scan the QR code.



An advanced technique uses retinal surgery trochars as anchor points for securing a three-piece IOL's haptics to the eye in a minimally invasive fashion.

Step Two: Alert the Team

Upon discovering a posteriorly dislocated IOL, you must immediately inform the surgeon and a local retina specialist. However, this does not necessary constitute a true emergency. IOLs are sterile, and surgical intervention—which is complex and usually requires a fair bit of planning—can often be delayed several days without any detrimental effect.

Step Three: Surgery

The cataract and retina surgeons can choose several different methods of retrieving the IOL and either replacing or securing it. It can be a one-step procedure or split into two: IOL removal and then implantation and securing. In addition, the surgeons may decide to do the combined procedure together, or the retina surgeon may be able to do it alone. To remove the IOL, it must first be cut up in the posterior chamber and then taken out through the small scleral ports.

After an IOL has been dislocated, no adequate capsular bag exists to hold a lens. The two most common options to secure the IOL into position is suturing it to the iris or the sclera. Suturing to the iris can damage the iris and limit the patient's ability to have a dilated exam in the future. Securing the lens to the sclera usually carries fewer long-term complications, but is a much trickier surgical option.

Step Four: Follow up

Postoperative management after IOL reposition and fixation is not significantly different from routine cataract surgery. The medications are the same, and the patient should have a relatively quiet eye. However, you need to keep an eye on the retina for any residual inflammation or possible subclinical tears.

Previously an invasive and traumatic procedure, IOL reposition and fixation has been refined with newer technologies and techniques so that today's patients are quite functional the next day—and optometrists are now an integral part of the pre- and postoperative care team. ■

The authors would like to thank Alan Franklin, MD, PhD, for contributing this month's video.

October 2017

■ **15-17.** *CE in Italy—Edinburgh.* Apex Hotel, Edinburgh, Scotland. Hosted by: James L. Fanelli. Key faculty: Lorraine Lombardi, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinItaly.com, call (910) 452-7225 or go to www.ceinitaly.com.

■ **20-22.** *Envision New York.* SUNY College of Optometry, New York City, NY. Hosted by: SUNY Office of Continuing Professional Education. CE hours: 50 total, 21 per OD. For more information, email Betsy Torres at ce@sunyopt.edu, call (212) 938-5830 or go to www.sunyopt.edu/cpe.

■ **21-22.** *VOA Fall Conference.* Omni Richmond, Richmond, VA. Hosted by: Virginia Optometric Association. CE hours: 8. For more information, email Bo Keeney at office@thevoa.org, call (804) 643-0309 or go to www.thevoa.org.

■ **21-22.** *CE in Fort Worth.* Dallas Fort Worth Marriott Hotel & Golf Club, Forth Worth, TX. Hosted by: University of Houston College of Optometry. CE hours: 16. For more information, email optce@central.uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu.

■ **21-22.** *Georgia Optometric Association Fall Education Conference.* University of Georgia Hotel and Conference Center, Athens, GA. Hosted by: Georgia Optometric Association. CE hours: 18. For more information, email Vanessa Grosso at vanessa@goaeyes.com, call (770) 961-9866 ext. 1 or go to www.goaeyes.com.

■ **21-23.** *2017 Annual Education Conference.* Mystic Marriott Hotel & Spa, Mystic, CT. Hosted by: Connecticut Association of Optometrists. Key faculty: Ron Melton, Randall Thomas. CE hours: 18. For more information, email tadb11@aol.com.

■ **22.** *Applebaum Symposium.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: Marshall B. Ketchum University. CE hours: 8. For more information, email Antoinette Smith at asmith@ketchum.edu, call (714) 872-5684 or go to ketchum.edu/ce.

■ **25-27.** *CE in Italy—Florence.* Hotel Silla, Florence, Italy. Hosted by: James Fanelli. Key faculty: Lorraine Lombardi, Joseph Pizzimenti, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinItaly.com, call (910) 452-7225 or go to www.ceinitaly.com.

■ **26-28.** *Idaho Optometric Physicians 2017 Annual Congress.* Sun Valley Resort, Sun Valley, ID. Hosted by: Idaho Optometric Physicians Association. CE hours: 26 total, 19 per OD. For more information, email Randy Andregg at execdir@iopinc.org, call (208) 461-0001 or go to idaho.aoa.org.

■ **29-31.** *CE in Italy—Sicily.* Hotel Villa Schuler, Taormina, Sicily. Hosted by: James Fanelli. Key faculty: Lorraine Lombardi, Joseph Pizzimenti, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinItaly.com, call (910) 452-7225 or go to www.ceinitaly.com.

November 2017

■ **2.** *Envision University Low Vision Grand Rounds.* Envision, Wichita, KS. Hosted by: Envision University. CE hours: 2. For more information, email Michael Epp at michael.epp@envisonus.com, call (316) 440-1515 or go to www.envisionuniversity.org.

■ **2-5.** *EastWest Eye Conference.* Global Center for Health Innovation and Convention Center, Cleveland, OH. Hosted by: Ohio Optometric Association. Key faculty: Ben Gaddie, Diana Shechtman, Leo Semes, Jeff Gersona, Carl Hilliar. CE hours: 250+ total, 26 per OD. For more information, email Keith Kerns at kkerns@ooa.org, call (800) 874-9111 or go to www.eastwesteye.org.

■ **2-5.** *Optometric Management Symposium.* Disney's Yacht & Beach Club, Orlando, FL. Hosted by: Optometric Management & Eyecare Business. Key faculty: Joseph Sowka, Greg Caldwell, Scot Morris. CE hours: 60 total, 25 per OD. For more information, email Maureen Trusky at maureen.trusky@pentavisionmedia.com, call (215) 628-7754 or go to www.omconference.com.

■ **2-5.** *Kansas Fall Eyecare Conference.* Doubletree by Hilton, Wichita, KS. Hosted by: Kansas Optometric Association. CE hours: 13. For more information, email Todd Fleischer at todd@kansasoptometric.org, call (785) 232-0225 or go to www.kansasoptometric.org.

■ **2-6.** *Art & Science of Optometric Care—A Behavioral Perspective.* Western University, Pomona, CA. Hosted by: Optometric Extension Program Foundation. Key faculty: John Abbondanza. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call (410) 561-3791 or go to www.oepf.org.

■ **2-6.** *VT/Visual Dysfunctions.* Office of Lynnette Burgess, Grand Rapids, MI. Hosted by: Optometric Extension Program Foundation. Key faculty: Robert Hohendorf. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call (410) 561-3791 or go to www.oepf.org.

■ **3-5.** *New Technologies & Treatments in Eye Care—Philadelphia.* Loews Philadelphia, Philadelphia, PA. Hosted by: *Review of Optometry*. Key faculty: Paul Karpecki. CE hours: 19. For more information, email Lois DiDomenico at reviewmeetings@jobson.com, call (866) 658-1772 or go to www.reviewofoptometry.com/philadelphia2017.

■ **3-5.** *Toronto Annual Fall Conference.* Hilton Suites Hotel, Donald Cousens Conference Centre, Markham, Ontario. Hosted by: Vision Institute of Toronto. Key faculty: Ron Melton, Randall Thomas, Rich Madonna, Bruce Onofrey, Stuart Richer, Ralph Chou. CE hours: 20. For more information, email visioninstitute@globalserve.net or go to visioninstitutecanada.com.

■ **5.** *Glaucoma Grand Rounds.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: Marshall B. Ketchum University. CE hours: 8. For more information, email Antoinette Smith at asmith@ketchum.edu, call (714) 872-5684 or go to ketchum.edu/ce.

■ **8.** *Annual Winter Seminar.* Jumping Brook Country Club, Neptune, NJ. Hosted by: New Jersey Academy of Optometry and Salus University. CE hours: 2. For more information, email Dennis Lyons at dhl2020@aol.com or call (732) 920-0110.

■ **10-11.** *WOA Primary Care Symposium.* The Country Springs Hotel, Waukesha, WI. Hosted by: Wisconsin Optometric Association. CE hours: 10. For more information, email Joleen Breunig at joleen@woa-eyes.org, call (608) 824-2200 or go to www.woa-eyes.org.

■ **10-12.** *2017 Fall Congress.* Hilton Sedona Resort & Spa, Sedona, AZ. Hosted by Arizona Optometric Association. For more information, call (928) 284-4040.

■ **10-12.** *NCOS Fall Congress.* Grove Park Inn, Asheville, NC. Hosted by: North Carolina Optometric Society. CE hours: 18. For more information, email Paul Kranze at paul@nceyes.org, call (919) 977-6964 or go to www.nceyes.org.

■ **12.** *Fall 2017 Education Conference.* Fredericksburg Hospitality House Hotel & Conference Center, Fredericksburg, VA. Hosted by: Virginia Academy of Optometry. Key faculty: Bruce Prum. CE hours: 4. For more information, email Angela Tsai at antsaiod@gmail.com.

■ **13.** *Illinois Optometric Association Winter CE Series.* Thelma Keller Conference Center, Effingham, IL. Hosted by: Illinois Optometric Association. Key faculty: Mile Brujic. CE hours: 6. For more information, email Charlene Marsh at ioabb@ioaweb.org, call (217) 525-8012 or go to www.ioaweb.org.

■ **18-19.** *Everything Therapeutic: San Antonio.* Westin Riverwalk, San Antonio, TX. Hosted by: University of Houston College of Optometry. Key faculty: Bruce Onofrey. CE hours: 16. For more information, email at optce@central.uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu.

December 2017

■ **2-3.** *34th Annual Cornea, Contact Lens & Contemporary Vision Care Symposium.* Westin Memorial City, Houston, TX. Hosted by: University of Houston College of Optometry. Key faculty: Jan Bergmanson. CE hours: 16. For more information, email optce@central.uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu.

■ **3.** *Clinical Topics in Optometry.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: Marshall B. Ketchum University. CE hours: 8. For more information, email Antoinette Smith at asmith@ketchum.edu, call (714) 872-5684 or go to ketchum.edu/ce.

■ **7.** *UABSO Evening of Education.* University of Alabama Birmingham School of Optometry, Birmingham, AL. Hosted by: University of Alabama Birmingham School of Optometry. CE hours: 2. For more information, email Katherine Clore at kclore@uab.edu, call (205) 934-5700 or go to www.uab.edu/optometry/home/uabso-ce.

■ **7-11.** *VT/Learning Related Visual Problems.* Southern College of Optometry, Memphis, TN. Hosted by: Optometric Extension Program Foundation. Key faculty: Paul Harris. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call (410) 561-3791 or go to www.oepf.org.

■ **23-30.** *Considerations in Ocular Disease Management and Treatment.* Norwegian Cruise Line's Norwegian Epic, Western Caribbean Cruise, round-trip Orlando (Port Canaveral), FL. Hosted by: Dr. Travel Seminars and the New Jersey Society of Optometric Physicians. Key faculty: Mark Dunbar. CE hours: 16. For more information, email info@drtravel.com, call (800) 436-1028 or go to www.drtravel.com.

January 2018

■ **6.** *Glaucoma Symposium.* Willows Lodge, Woodinville, WA. Hosted by: Pacific University College of Optometry. Key faculty: Howard Barnebey, Murray Fingeret. CE hours: 7. For more information, email Michelena Buckingham at mikibuckingham@pacificu.edu, call (503) 352-2985 or go to www.pacificu.edu/future-graduate-professional/colleges/college-optometry/continuing-education.

■ **12-14.** *AZOA 2018 Bronstein Contact Lens & Cornea Seminar.* Hilton Scottsdale Resort & Villas, Scottsdale, AZ. Hosted by: Arizona Optometric Association. CE hours: 15.5. For more information, email Kate Diedrickson at kate@azoa.org or go to www.azoa.org/Connect.

■ **13-15.** *Kraskin Invitational Skeffington Symposium on Vision.* Embassy Suites Hotel at the Chevy Chase Pavilion, Washington D.C. Hosted by: Optometric Extension Program Foundation. CE hours: 19. For more information, email Jeffrey Kraskin at jlkraskin@rcn.com, call (202) 363-4450 or go to www.skeffingtonsymposium.org.

■ **14-20.** *2018 Island Eyes Conference.* Ritz-Carlton Kapalua, Kapalua (Maui), Hawaii. Hosted by: Pacific University College of Optometry. Key facutly: Mark Andre, Carlo Pelino, Alan Reichow, Tracy Doll, Walt Whitley, Fraser Horn. CE hours: 29. For more information, email Jeanne Oliver at jeanne@pacificu.edu, call (503) 352-2740 or go to www.pacificu.edu/future-graduate-professional/colleges/college-optometry/continuing-education.

■ **22.** *Day at the Capitol & Winter CE.* Boise Centre, Boise, ID. Hosted by: Idaho Optometric Physicians. CE hours: 4. For more information, email Randy Andregg at execdir@iopinc.org, call (208) 461-0001 or go to idaho.aoa.org.

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Product Review

Devices and Equipment

New Punctal Plug

The VeraPlug Flexfit by Lacrivera simplifies and improves punctal occlusion for both doctor and patient, the manufacturer says. The plug's nose is designed to collapse when inserted and, according to the company, the device allows for simple sizing, easy insertion, superior retention and excellent patient comfort. It comes in four sizes—extra small, small, medium and large—in sterile preloaded and non-sterile bulk packaging.

Visit lacrivera.com.

Pediatric Screening Device

Practitioners now have a new screening device to consider for their pediatric eye exams. With the Plusoptix S16 mobile vision screener, you can obtain reliable measurements for refraction (spherical equivalent), pupil diameter, pupil distance and symmetry of corneal reflexes, according to the company. The S16 is designed for stationary use. In addition to producing hardcopy reports via any networked printer, it can connect to a server for data exchange to an EHR system, Plusoptix says.

Visit www.plusoptix.com.

Ultra-Widefield Fundus Imaging

The Clarus 500 from Zeiss offers practitioners a new option for widefield retinal photography. It combines improved color accuracy with high resolution imaging—down to 7µm—within a 133-degree field of view, Zeiss says. High-definition scans are merged into a single 200-degree image, and the system provides annotation and caliper measurement tools. The Clarus 500 also integrates with Zeiss analysis software to review findings alongside other ophthalmic images and exam data.

Visit www.zeiss.com/us/clarus.

Software

Patient Management System

For help with patient booking and retention as well as office efficiency, the Prime Nexus software suite from EyeCare Prime allows practices to more easily schedule and confirm appointments, connect with EHR systems, integrate with the practice's website and conduct personalized email campaigns, the company says.

Visit eyecareprime.com.

Myopia Calculator

A new evidence-based tool from the Brien Holden Vision Institute will help clinicians educate patients about managing myopia appropriately. The free, web-based calculator merges individual patient information with different optical and pharmacological treatment options to illustrate the impact on their future level of myopia. Practitioners input patient age and level of refractive error when beginning therapy and select a treatment option. The calculator then demonstrates the effect of the treatment on myopia progression and how it is likely to progress without treatment.

Visit www.brienholdenvision.org.

Vitamins

Easy-to-Swallow Minigels

For patients who may have difficulty swallowing larger pills, Bausch + Lomb now offers a minigel vitamin with concentrated fish oil to help reduce the size of the pills. The Ocuvite Adult 50+ formula minigel daily eye vitamins, which will replace the current Ocuvite Adult 50+ soft gels, are 25% smaller and easier to swallow, according to the company.

Visit www.bausch.com.

Contact Lenses and Lens Care

Monthly Toric Lens

Optometrists have a new choice to offer their patients with astigmatism who wear monthly lenses. The Acuvue Vita for Astigmatism is a daily wear lens designed for reliable comfort throughout the month, according to Johnson & Johnson Vision. The lens uses a non-coated silicone hydrogel formulation, balanced to help maximize and maintain hydration, according to the company. The design works naturally with the eyelids, helping to keep the lens in the correct position.

Visit www.acuvue.com.

New MPS Package Stickers

Alcon's packaging of its Opti-Free multipurpose solution now comes with a bold pink sticker to help patients easily identify it on retail shelves, according to Alcon. To differentiate Opti-Free products from generics, the new sticker highlights that the product offers an exclusive formula compared with store brands, according to the company.

Visit myalcon.com. ■

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Founded in 1951, the National Board of Examiners in Optometry (NBEO), is one of the few national boards in any profession with a repertoire of exams which include computer-based tests, an advanced competence exam, and clinical skills test using standardized patients at the National Center of Clinical Testing in Optometry. The mission of NBEO is to serve the public and profession of optometry by developing, administering, scoring and reporting results of valid examinations that assess competence.

The Executive Director role will report to the Board of Directors and will have the opportunity to lead the organization in strategic planning.

This candidate's past experiences should demonstrate a history of progressive leadership development and an ability to direct the human, financial, and physical resources of the organization. The person for this position must be a motivated individual with a commitment to setting the course for best psychometric and testing practices for the future. This individual will be required to relocate to Charlotte, NC. A license to practice optometry is preferred, but not required.

A complete description for this position will be available on the NBEO website at www.optometry.org by September 15. Applications will be accepted starting September 15, 2017 through October 25, 2017.

Applicants should send a cover letter and CV to jill.martinson-redekopp@optometry.org

Diagnostic **Quiz**

Plumbing the Depths

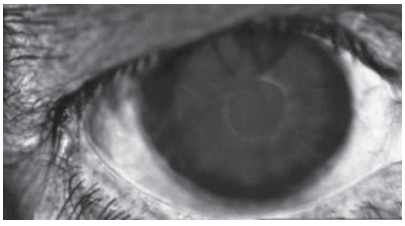
By Andrew S. Gurwood, OD

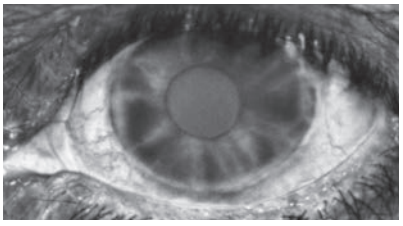
History

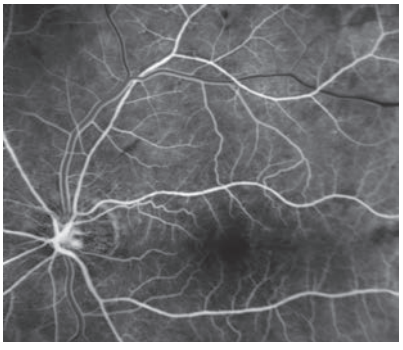
A 69-year-old Caucasian male presented with a chief complaint of blurry vision in the left eye for one month. Associated symptoms included redness, photophobia and a dull ocular ache. The patient denied trauma. His ocular history was remarkable for cataract surgery in both eyes a year earlier. His medical history included hypertension, hypercholesterolemia and depression, for which he was properly medicated with amlodipine, chlorthalidone, lisinopril, atorvastatin and fluoxetine. He also reported current tobacco use. His resting blood pressure reading was 156/79mm Hg. He denied allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities with -1.00D spheres were 20/20 OD and 20/80 OS with improvement upon use of the pinhole to 20/25 OS. Extraocular muscles and confrontation fields were full in both eyes. His right pupil was round and demonstrated a reactive reflex to light with no afferent pupillary defect. The left was mid-dilated, exhibiting a grade 3+ afferent pupillary defect. Color desaturation testing demonstrated a 30% red desaturation in his left eye, and brightness testing uncovered 30% diminished light sensitivity. Refraction uncovered -1.25D spheres in both eyes, improving acuity in the right to 20/60.







For additional images for this case, visit www.reviewofoptometry.com, or scan this QR code.

Can any of these images help point to the diagnosis for this 69-year-old patient who underwent cataract removal a year earlier?

Biomicroscopy of the right eye was unremarkable. The left eye exhibited 1+ diffuse conjunctival injection, a clear cornea, a quiet anterior chamber and open 2x2 Van Herick angles. A critical anterior segment finding is illustrated in the photograph. Intraocular pressures by Goldmann tonometry measured 10mm Hg OD, 20mm Hg OS.

A dilated fundus examination found optic nerves that were flat with 0.4 round cup-to-disc ratios and healthy rim tissues in both eyes. An isolated splinter hemorrhage was visible superotemporal to the optic disc in the right eye. Maculae were flat and intact in both eyes. The periphery in the right eye was normal, but the left revealed mid-peripheral intraretinal hemorrhages, dilated veins and arteriolar narrowing in all quadrants.

Your Diagnosis

Based on the information provided, what's the likely diagnosis? To find out, please visit us at www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 100):* 1) b; 2) c; 3) d; 4) b; 5) d.

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References: 1. Alcon data on file, 2017. **2.** Alcon data on file, 2008. **3.** Alcon data on file, 2011. **4.** Angelini TE, Nixon RM, Dunn AC, et al. Viscoelasticity and mesh-size at the surface of hydrogels characterized with microrheology. *Invest Ophthalmol Vis Sci.* 2013;54:E-abstract 500.

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