Ocular Surface Review: Hyaluronic Acid for Dry Eye, *PAGE 80*

REVIEW® OF OPTOMETRY

March 15, 2018

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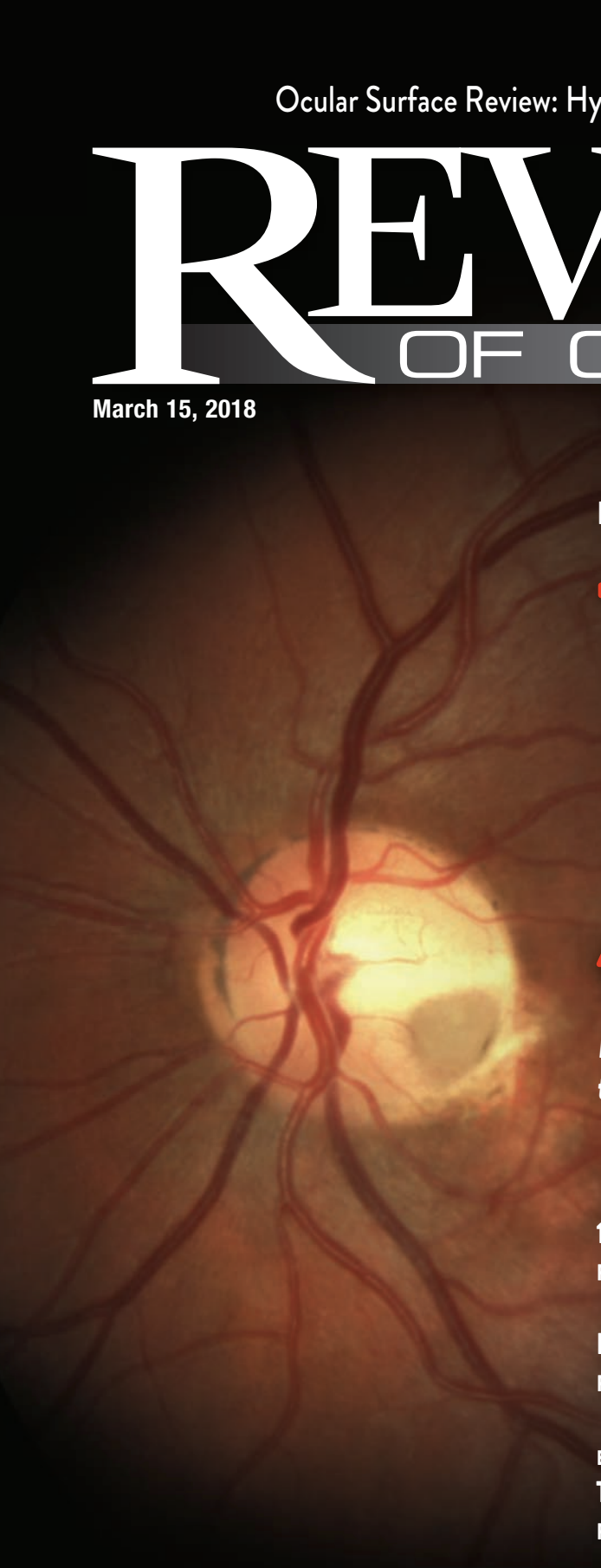

DIAGNOSTIC SKILLS & TECHNIQUES

Test Your Diagnostic Acumen

Do you have what it takes to solve these five puzzlers? *PAGE 34*

EYE-OPENINGLY DIFFERENT

TAKE A CLOSER LOOK

at Biotrue® ONEday—the fastest growing family of daily disposable hydrogels[1*]

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

	Biotrue® ONEday	1-DAY ACUVUE MOIST	DAILIES AquaComfort Plus
Moisture content	78%	58%	69%
Oxygen level†	42 Dk/t	25 Dk/t	26 Dk/t
Spherical aberration control‡	✓		
UVA/UVB protection	✓	✓	
Patient rebate§	$200	$100	$120

Give your patients who are new to daily disposable lenses a smart combination of performance and value.

*Lens sales between January 2016-October 2017 among traditional hydrogels.
†Oxygen levels for single vision spherical (SVS) lenses only.
‡In SVS and toric lenses only.
§Annual supply rebate as of Q3 2017 for existing SVS wearers.

REFERENCE: 1. Data on file. Bausch & Lomb Incorporated. 3rd Party Industry Report. 2016-2017.

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VOL. 155 NO. 3 ■ MARCH 15, 2018

IN THE NEWS

A recent study from the United Kingdom found **patients with diabetes are twice as likely to develop cataracts** as those without the condition. The researchers analyzed the medical records of more than 56,000 diabetes patients older than 40 and discovered those between ages 45 and 49 were 4.6 times more likely to develop cataracts, while those between ages 50 and 54 had a 5.7 times higher risk than healthy individuals.

Becker C, Schneider C, Aballéa S, et al. Cataract in patients with diabetes mellitus—incidence rates in the UK and risk factors. Eye. February 1, 2018. [Epub ahead of print].

Researchers have discovered **increased upper-body strength in men is associated with higher baseline intraocular pressure (IOP)**. After taking baseline IOP measurements, researchers asked 65 men to perform an incremental loading test in the ballistic bench press. Results show a strong correlation between baseline IOP and relative maximum force and power, as well as relative one-repetition maximum—which may have implications for managing ocular conditions in patients who participate in resistance training programs, study authors say.

Vera J, Jiménez R, García-Ramos A, Cárdenas D.Muscular strength is associated with higher intraocular pressure in physically active males. Optom Vis Sci. 2018;95(2):143-9.

Investigators are calling for validated classification criteria in the field of uveitis after a review of 5,766 uveitis cases revealed **agreement among uveitis experts on diagnosis is moderate at best**. Five committees of nine experts each reviewed cases from a database of 25 uveitic diseases, and only after committee consensus conference calls were they able to reach agreement in 99% of cases.

Jabs DA, Dick A, Doucette JT, et al. Interobserver agreement among uveitis experts on uveitic diagnoses: the standardization of uveitis nomenclature experience. Am J Ophthalmol. 2018 Feb;186:19-24.

Retinal Infarction: Better Safe Than Sorry

Too few patients presenting with retinal stroke are receiving follow-up testing, new research shows.

By Francesca Crozier-Fitzgerald, Associate Editor

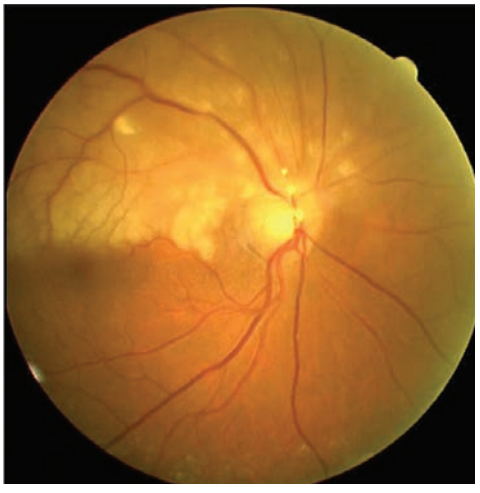
As the retina and brain receive blood from a shared source, retinal infarction, or stroke in the eye, should serve as a clear warning sign for more severe, underlying systemic health risks or potential future stroke.[1] While these high-risk cases should be referred to a neurologist or sent for a full basic stroke examination, a new study shows these critical steps are not taking place as often as they should.[1]

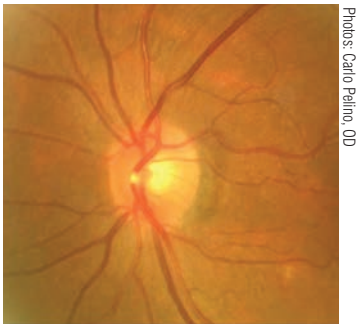
Researchers at the American Heart Association (AHA) followed 5,688 patients presenting with retinal infarction and found only one-third were sent to receive a basic stroke workup, and less than one in 10 were referred to a neurologist. Not surprisingly, within 90 days of their initial retinal infarction, the study shows that one in every 100 patients from this group experienced ischemic stroke.[1]

"Due to the nature of the claims data, we didn't specify what type of retinal infarction they had, but included any patient with central retinal artery inclusion (CRAO), branch retinal artery occlusion (BRAO) or amaurosis fugax," says lead study author and neurologist Alexander Merkler, MD.

In a video interview with the AHA, Dr. Merkler noted, "We're really not doing an appropriate job evaluating those patients with retinal infarction for stroke risk factors, and therefore we may not be preventing future full-blown ischemic stroke in these patients."

(continued on page 7)

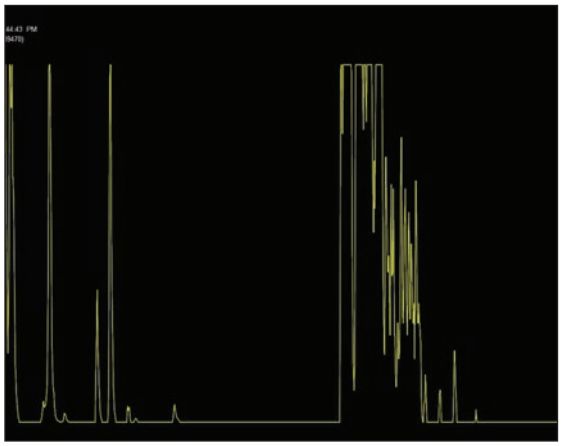




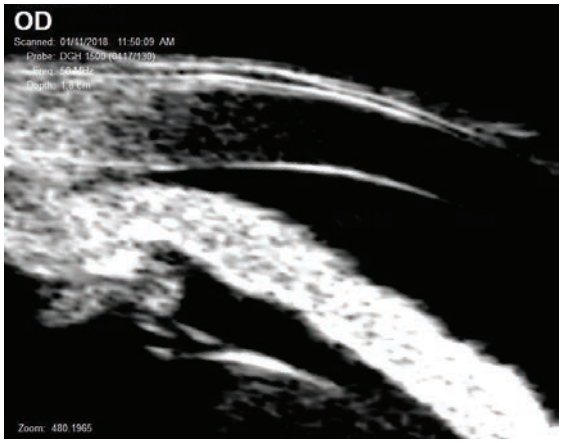
Photos: Carlo Pelino, OD

If a patient presents with BRAO with emboli, as seen here, clinicians should refer them immediately for a full stroke workup.

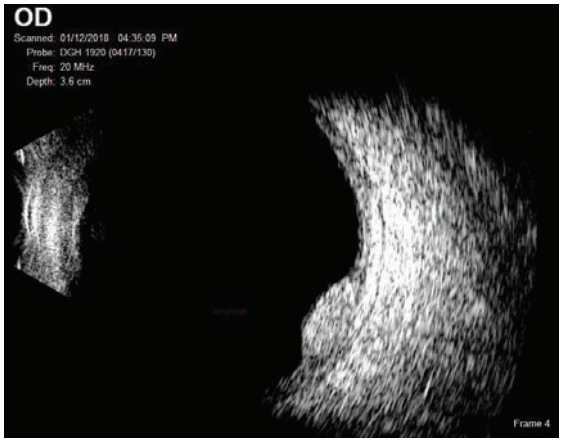
ScanMate FLEX



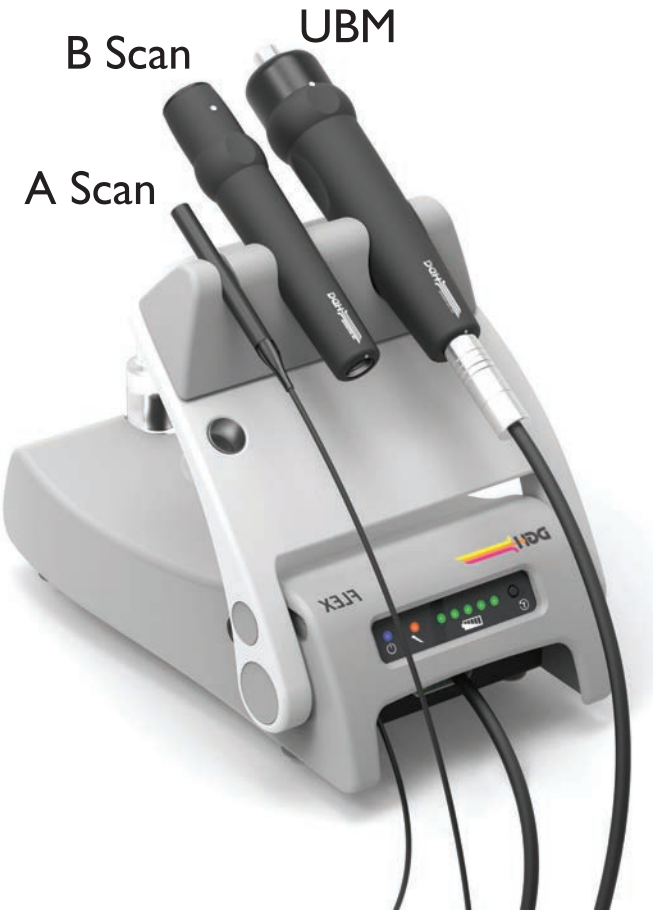
A Scan Measurement (Immersion Mode)



UBM Image with Plateau Iris and Closed Angle



B Scan Image with Choroidal Hemangioma



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Amblyopia Therapy: Skip the iPad

The Pediatric Eye Disease Investigator Group (PEDIG) has been looking at the efficacy of a binocular iPad game for amblyopia therapy, and the second round of results, this time with participants in their teens, isn't promising: 16 weeks of therapy with a binocular iPad game was no better than patching therapy—and possibly worse.[1]

"Doctors working with the pediatric population were hopeful that iPad therapy would provide an additional treatment option to manage amblyopia," says Kara Tison, OD, an assistant clinical professor and chief of pediatrics at the University of the Incarnate Word Rosenberg School of Optometry. "It was disappointing to learn that the iPad therapy was unsuccessful."

The researchers randomized 100 participants between the ages of 13 and 16 to 16 weeks of either one hour of a binocular iPad game daily or patching of the fellow eye for two hours a day. Results showed those in the iPad group had a mean ambloyic eye visual acuity (VA) increase of 3.5 letters from baseline, while those in the patching group had a mean improvement of 6.5 letters. The difference, after adjusting for baseline VA, was -2.7 letters in favor of patching therapy.[1]

An earlier PEDIG study with younger participants ages five to 12 provided inconclusive results, and was unable to establish if iPad game play was worse than two hours of prescribed patching.[2]

Working from such lackluster results, the new finding shouldn't come as a huge shock. What was surprising was one of the possible reasons behind the results: compliance. In the age of technology, you would think teens would do well with a vision therapy program that includes video games. However, the data shows only 13% of participants completed more than 75% of the prescribed treatment.

"Unfortunately, teenagers found the iPad game uninteresting, and the study was stopped early due to poor compliance and visual acuity improvement favoring patching," Dr. Tison says.

"With evidence that binocular treatment of amblyopia can improve visual acuity for adults, there is still hope that, with the right game, we can see improvement with binocular treatment for the pediatric population," Dr. Tison says. PEDIG is already recruiting for another binocular iPad game, ATS20 Binocular Dig Rush Game Treatment for Amblyopia, which will hopefully be more appealing and lead to better treatment adherence, according to Dr. Tison.[3]

1. Manh VM, Holmes JM, Lazar EL, et al. A randomized trial of a binocular iPad game versus part-time patching in children aged 13 to 16 years with amblyopia. Am J Ophthalmol. 2018 Feb;186:104-15.
2. Holmes JM, Manh VM, Lazar EL, et al. Effect of a binocular iPad game vs part-time patching in children aged 5 to 12 years with amblyopia. JAMA Ophthalmology. 2016;134(12):1391-400.
3. PEDIG. ATS20 - Binocular Dig Rush Game Treatment for Amblyopia. http://pedig.jaeb.org/Studies.aspx?RecID=506. Accessed February 20, 2018.

Worm Your Way Into This Diagnosis

A 26-year-old Oregon woman has been hailed as the first case of a *Thelazia gulosa* infection and the first reported case of human thelaziasis in North America in more than two decades.[1]

While on a salmon fishing expedition in Alaska, Abby Beckley, lifelong rancher and travel enthusiast, experienced a red, irritated left eye with a foreign body sensation, droopy eyelid and migraines. Once on shore, she found a mirror and proceeded to pull a worm from her eye. Clinicians at Oregon Health and Science University in Portland sent samples to the Centers for Disease Control and Prevention's (CDC) Parasite Diagnostics and Biology Laboratory for identification.[2] That's when researchers realized this was not just a rare case, but an unprecedented discovery.[1] She was diagnosed with parasitic infiltration of the left periocular tissues and a secondary bilateral papillary reaction of the upper and lower palpebral conjunctivae.[2] The infiltrate was *T. gulosa*, which, until now, was known to only infect cattle.[2]

The few previous cases of human eyeworms reported worldwide implicate two species of Thelazia: *T. callipaeda* in Asia and Europe and *T. californiensis* in the United States.[1] While a common veterinary infection, Ms. Beckley's infection is the first reported case of this specific species of *Thelazia* to jump species—via a fly vector—and infect a human.[1]

Clinicians chose to manually remove the worms over a 20-day period.[1] Because the worms remain on the surface of the eye, there is little concern for systemic complication; however, some reports suggest corneal scarring, opacity and blindness without proper treatment.[1,2]

Luckily, Ms. Beckley had a team of physicians, optometrists and CDC experts to help her through the ordeal—and make history.[1]

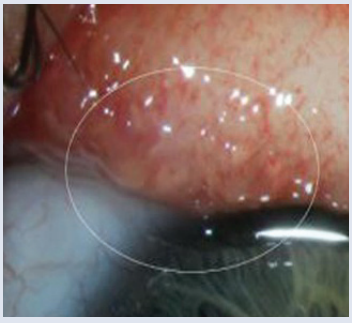

Photo: CDC.gov/DPDx.

***T. gulosa* on the surface of a patient's conjunctiva (circle).**

1. Bradbury RS, Breen KV, Bonura EM, et al. Case report: conjunctival infestation with Thelazia gulosa: a novel agent of human Thelaziasis in the United States. Am J Tropical Medicine and Hygiene. February 12, 2018. [Epub].
2. LaMotte S. 'I looked at it, and it was moving': Worm in woman's eye leads to unique discovery. CNN. February 12, 2018. www.cnn.com/2018/02/12/health/human-eye-worms/index.html. Accessed February 13, 2018.

Retinal Stroke

(continued from page 4)

And it's not that the gravity of these cases is being overlooked.

"As optometrists and ophthalmologists, our responsibility is to recognize the risks of retinal artery occlusion in a holistic way, as it serves as a warning sign for threats that go beyond the patient's vision health," says Carlo Pelino, OD, chief of Optometric Retina Service at the Eye Institute of Philadelphia.

"When we get a patient with BRAO or CRAO in our chair, we know to send that case, as soon as possible, to the nearest stroke center or emergency room. These urgent cases should receive a diffusion-weighted imaging (DWI) MRI and subsequent blood work to rule out other potentially life-threatening conditions, like ischemic or hemorrhagic stroke," Dr. Pelino says.

Research shows DWI imaging is an ideal tool for diagnosing acute ischemic stroke.[2]

"Patients with retinal infarction should be cared for in the same manner as patients with infarction of the brain," Dr. Merkler says.

The study further illuminates the importance of interprofessional collaboration between optometrists, ophthalmologists and neurologists. Better communication and proactive follow-up testing is crucial to help ensure clinicians identify early warning signs, implement necessary precautions and prevent future risks as quickly as possible.

1. American Heart Association. Too few with stroke of the eye are treated to reduce future stroke. ScienceDaily. January 25, 2018. https://newsroom.heart.org/news/too-few-with-stroke-of-the-eye-are-treated-to-reduce-future-stroke. Accessed February 12, 2018.
2.Okorie CK, Ogbole GI, Owolabi MO, et al. Role of diffusion-weighted imaging in acute stroke management using low-field magnetic resonance imaging in resource-limited settings. West Afr J Radiol. 2015;22(2):61-6.

Ears use Eyes to Anticipate Sound

A recent study reveals that the visual and auditory systems are far more coordinated than previously predicted.[1]

The researchers placed 16 study participants in a dark room, presented them with visual LED light stimuli and asked them to follow the light with their eyes while maintaining a fixed head position. Highly sensitive microphones placed in the ear canal tracked the concurrent eardrum vibrations.[1]

The results showed a clear correlation: simply moving the eyes from one side to the other induced parallel movement in the subject's eardrums. When the participants' eyes made larger, more exaggerated movements, a greater vibration was tracked in the ear canal. This auditory response occurred even in silent environments, suggesting eye movements and auditory response are connected with or without the presence of sound.[1]

Surprisingly, results show vibrations in the ear occurred slightly before (10ms) the eyes began tracking, leading researchers to believe the brain's desire to align stimuli from the visual and auditory pathways is significant quite early in the process.

The study results may help researchers better understand the role vision can play in hearing-impaired individuals, as well as the ways hearing can aid in the perception of stimuli for the visually impaired.

1. Gruters KG, Murphy DLK, Jenson CD, et al. The eardrums move when the eyes move: A multisensory effect on the mechanics of hearing. Proceedings of the National Academy of Sciences. 2018;201717948. [Epub ahead of print].

New OD Surgical Fellowship

The University of the Incarnate Word's Rosenberg School of Optometry recently launched a new Fellowship in Ocular Disease and Optometric Surgery that focuses on "ocular disease and minor surgery through intensive hands-on clinical experience and concentrated disease exposure," according to the school's program guide.

It emphasizes surgical procedures optometrists in some states are currently allowed to perform, such as laser iridotomy, trabeculoplasty, YAG capsulotomy, procedures involving amniotic membranes and excision of dermatologic lesions and chalazia, as well as subcutaneous and subconjunctival injections.

"It's something we think is necessary," says Fellowship Program Coordinator Kyle Sandberg, OD. "We look at what's happening in eye care and the population, and we see there's going to be a need for medical eye care."

Although the school is in San Antonio, the fellows will complete the 12-month program at the DeSoto Parish Regional Eye Institute and Surgery Center in Logansport, La., and will follow up with patients out of the optometric practices of Dennis Golden, OD, in Carthage, Texas.

Currently, one optometric fellow's career is already taking off as a result of the new program. "So far, I've probably done close to 200 laser procedures and removed 500 lumps and bumps," explains the program's pilot fellow Katherine Dronka, OD, who also educates students and trains technicians as part of the fellowship. This knowledge base has provided her a launching pad to the next step in her career, teaching optometric surgical procedures at the University of Pikeville in Kentucky, a position she's stepping into in July.

A Piece of the Puzzle

On the surface, the program gives individual optometrists a deep background in performing optometric procedures; but the program has an additional motive: expanded scope of practice. While Texas neighbors Oklahoma and Louisiana have gone as far as to indicate laser procedures for optometric use, Texas remains one of the most restrictive states for optometrists in the country. But Dr. Sandberg is working to change that by meeting with legislators and addressing their concerns, in part by by discussing Rosenberg's surgical fellowship.

"I am part of the Texas Optometric Association, and our goal is to push scope expansion in 2019," Dr. Sandberg says. "The challenge has always been 'where do optometrists get that training?'"

He's hoping this fellowship will show that ODs are, in fact, receiving intensive training and hospital privileges and ODs in Texas can add lasers and minor procedures to their lineup. "We've got tons of clinical data that shows complication and success rates. I'm confident that our data will show that we're at the same levels as ophthalmologists," he says. ■

BEAR IN MIND
THE FORMULATION OF LOTEMAX® GEL

- **ENGINEERED TO ADHERE TO THE OCULAR SURFACE**[1,2]
  - Adaptive viscosity: Gel at rest, viscous liquid on the eye
  - Drug-related blurred vision was rarely reported (0.25%, 2/813)
- **~70% LESS PRESERVATIVE than LOTEMAX® SUSPENSION (loteprednol etabonate ophthalmic suspension) 0.5%**[2,3,5]
- **DOSE UNIFORMITY–EVERY DROP, EVERY TIME**
  - No shaking required to resuspend drug[2-4]
- **pH OF 6.5 CLOSE TO THAT OF HUMAN TEARS**[2]
- **CONTAINS 2 KNOWN MOISTURIZERS**[3]
  - Glycerin and propylene glycol

~80% unrestricted managed care access on commercial plans*

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

LGX.0114.USA.16

Based on 9269101/9269201 Revised: 08/2016

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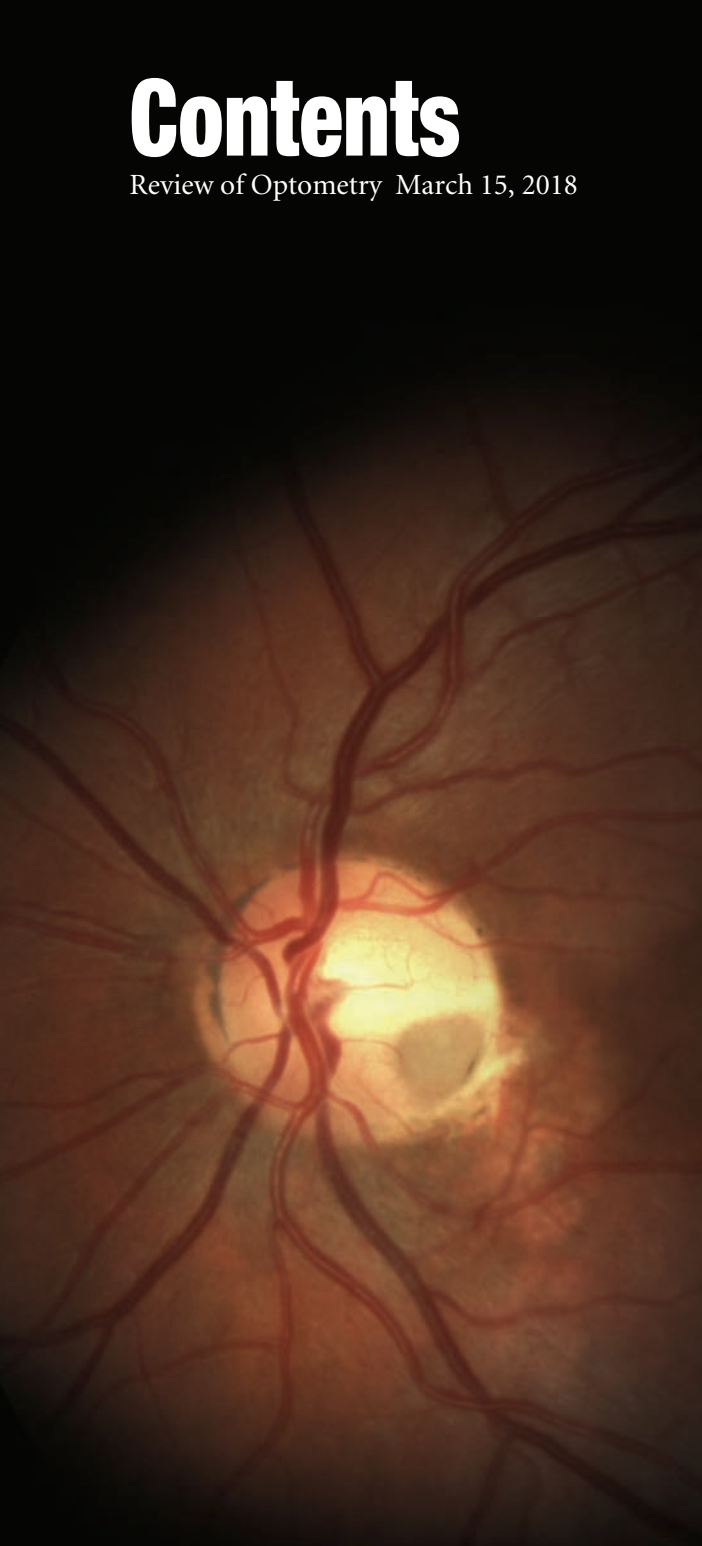
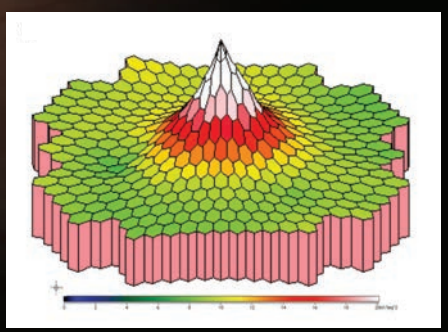


DIAGNOSTIC SKILLS & TECHNIQUES



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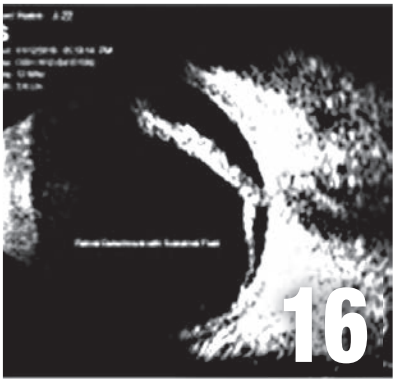
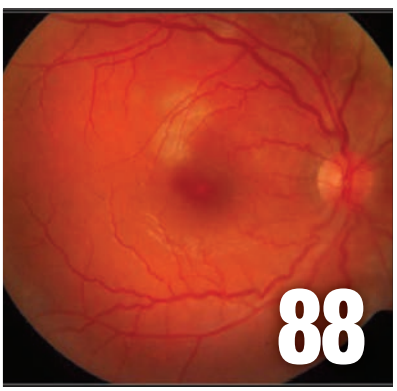
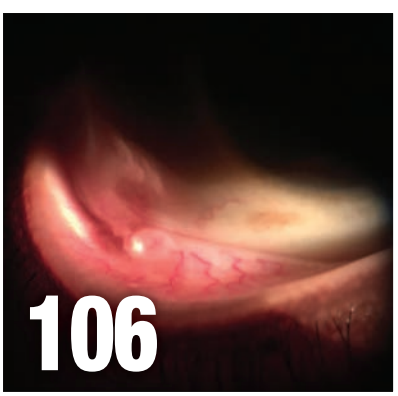
Review of Optometry March 15, 2018







REVIEW OF OPTOMETRY

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Outlook

By Jack Persico, Editor-in-Chief

This Year's Model

Technology advances by leaps and bounds but your responsibility remains the same.

Walking the exhibit floor at the recent SECO conference, I was struck again by how revolutionary optometric technology has become, especially in glaucoma. A colleague and I stopped to watch a demo of one of the latest OCT devices, which could integrate data and images from retinal nerve fiber layer and ganglion cell layer scans, visual field testing and fundus photography. With the press of one button, serial findings popped up onscreen for instantaneous trend analysis. The device compared the results to a normative database and gave its assessment of the risk of glaucoma. It seemed like you didn't need to know anything about the disease or the patient to have a pretty good idea of their status.

And that's the problem. Technology never has all the answers—you do, or at least you need to.

Just a few days earlier, this magazine had given its annual career achievement award to Tom Lewis, OD, retired former president of Pennsylvania College of Optometry and a leading light in glaucoma education throughout his distinguished career. In his day, he had to make do with perimetry, tonometry, a fundus exam and little else. As incredible as OCT is, I can't help but feel that clinicians of Dr. Lewis's generation have an ability to intuit the status of a glaucoma patient better than someone who never learned how to make a diagnosis "the hard way."

OCT is now among the standard of care for glaucoma management. Just a decade ago, it was mostly confined to retinal disease and the specialty practices that needed it. Now, it seems, *everyone* needs one.

The ascent of OCT is one of eye care's biggest success stories, with incalculable benefits for practitioners and patients. If you don't have one yet, run, don't walk, to your nearest exhibit hall and plunk down your credit card. But when you do, remember that it's nothing more than a tool and you're the doctor. As you start using it in your practice, try doing the assessments mentally and then see if your results sync up with what the device tells you. Even if it's just an exercise, that feeling of having your neck on the line can force you to learn the craft more deeply.

Technology often occludes knowledge that way. I sometimes marvel at the realization that my two-year-old son might never drive a car. The big automakers and tech companies are all working fiercely to perfect self-driving vehicles. Will they get there in the next 14 years? There's a good chance they will. No matter what, the experience will be safer, simpler and more reliable than today. As a parent, I'm relieved that he'll be protected in ways I never was at that age. But I do feel a twinge of loss on his behalf at the things he'll lose as automobile travel shifts from an active to a passive experience. There's a thrill, and a responsibility, to being behind the wheel.

Younger ODs may be better equipped—in the sense of literally having better equipment—than older ones, but diagnosis should never become passive and automated. No matter how fancy your gear gets, you'll always be in the driver's seat. ■

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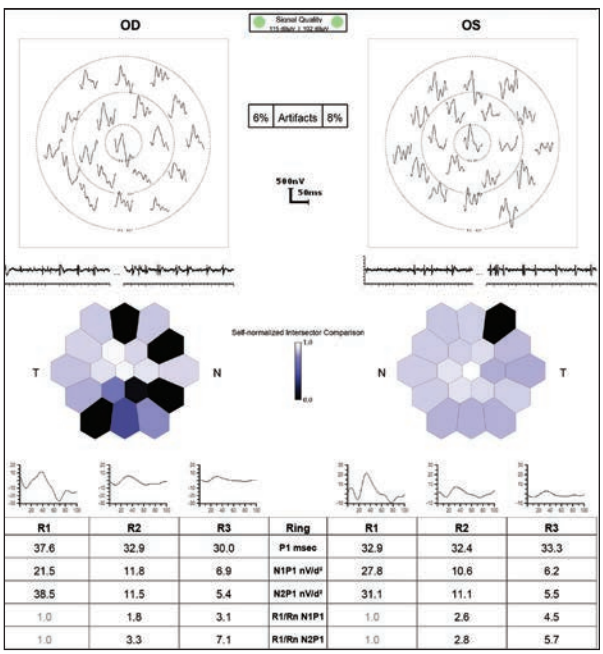
- Monitor retinal function loss and recovery with objective, quantitative metrics[2-3]
- Co-manage patients more efficiently for more timely changes to treatment

The American Academy of Ophthalmology (AAO) recommends the use of multifocal electroretinography for chloroquine (CQ) and hydroxychloroquine (HCQ) retinopathy screening.[4]

Plaquenil Toxicity Example



R1	R2	R3	Ring	R1	R2	R3
37.6	32.9	30.0	P1 msec	32.9	32.4	33.3
21.5	11.8	6.9	N1P1 nVid²	27.8	10.6	6.2
38.5	11.5	5.4	N2P1 nVid²	31.1	11.1	5.5
1.0	1.8	3.1	R1/Rn N1P1	1.0	2.6	4.5
1.0	3.3	7.1	R1/Rn N2P1	1.0	2.8	5.7

See for yourself at Vision Expo East Booth MS4527 or visit Diopsys.com/multifocal

1. Hood, DC, et al. ISCEV Standard for clinical multifocal electroretinography (2011 edition). Doc Ophthalmol 124:1–13. **2.** Dettoraki M, Moschos MM. The Role of Multifocal Electroretinography in the Assessment of Drug-Induced Retinopathy: A Review of the Literature. Ophthalmic Res 2016;56:169–177. **3.** Talamini CL, et al. Abnormal multifocal ERG findings in patients with normal-appearing retinal anatomy. Doc Ophthalmol 2011;123(3):187-192. **4.** Marmor, M, et al. Recommendations on Screening for Chloroquine and Hydroxychloroquine Retinopathy (2016 Revision). Ophthalmology 2016;123:6:1386-1394.

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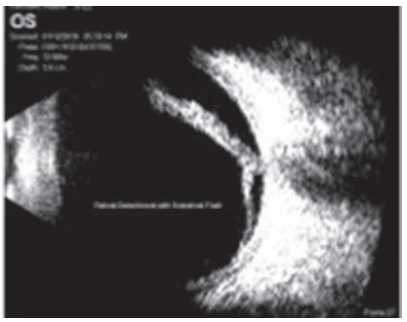
It's *All* Primary Eye Care Today

Before, you could only find advanced diagnostics in the secondary care fields. Today, they belong in every practice. **By Paul M. Karpecki, OD, Chief Clinical Editor**

When I started out as a fresh new OD, I thought 'primary eye' care meant doing the basics of vision care—refractions, spectacles, contact lenses and regular eye exams. Boy, was I wrong. It didn't take me long to realize it means managing *everything* related to the eyes and vision that isn't secondary care (e.g., a subspecialist) or tertiary (e.g., transplants, oncology). That's a lot to be in charge of, as it includes everything from eye alignment to neurology to the diagnosis of rare diseases and malignant tumors. These days, primary care ODs even realize that electrodiagnostics and other advanced tools are essential to providing these key services. Here's a look at some of the new technologies gracing a primary eye care office:

SightSync. This can detect and measure eye alignment in less than two minutes. The technology uses peripheral focus, and is objective and accurate for measuring horizontal phorias. It is also designed to transfer measurements into a spectacle lens called a neurolens, essentially a progressive prism created to correct the problem. In its pivotal study, 93% of patients had a reduction in symptoms and 74% stated the symptoms were eliminated or decreased substantially.[1]

Ultrasound. More than a decade ago when an A-scan/B-scan/ultrasound biomicroscope combination cost six figures, it didn't seem to fit with primary eye care. Today, because of advances in technology,



This B-scan, taken with the Scanmate Flex, reveals a retinal detachment.

more ODs are adding this, gaining high resolution imaging for a tenth of the cost. Now it fits squarely into primary eye care and may become an essential piece of equipment necessary to practice at a high level.

Take, for example, something we see often: a choroidal nevus. How do you determine if it truly is just a nevus rather than a malignant melanoma? Two important determinants are the basal diameter and the elevation, both of which can be determined easily and accurately with B-scan ultrasound. A basal diameter that increases over time or an elevation over 2mm are significant signs of a potential melanoma (other signs include lipofuscin on the surface of the lesion, touching the optic nerve or the presence of subretinal fluid).

Ultrasound can help you confirm a retinal detachment, visualize the retina when there is no clear view (e.g., vitreous heme), assess axial length for cataract surgery, determine angle closure and narrow angles and detect iris cysts and tumors, to name a few. In my practice, we routinely have every patient diagnosed with a moderate or large nevi undergo ultrasound measurements and monitor for progression.

VEP/ERG. Visual evoked potential measures the signal passing through the optic nerve to the brain—an ideal tool when determining neuropathy. Another electrodiagnostic test, electroretinogram, can also help assess retinal function. Both technologies are now available in smaller, portable options, and are far easier to use than previous iterations. They can be integral to detecting and following glaucoma, early retinitis pigmentosa and diabetic retinopathy, for ex-ample, without the need for dilation.

Pupil testing. The days of the difficult swinging flashlight test may finally be behind us. Doctors and staff have always had difficulty with this, given that some patients have dark irides or the relative afferent pupil defect (RAPD) was small. A new device called EyeKinetix (Konan Medical) takes about 30 seconds and can determine the presence of a 0.5 RAPD, helping with an overall neuro assessment screening for the potential diagnosis of glaucoma (often being an asymmetric neuropathy).

Given these impressive technological advances, it may be time to consider advanced testing as truly primary eye care tests. ■

Note: Dr. Karpecki is a consultant for many companies mentioned here.

1. Miles C, Krall J, Thompson V, Colvard DM. A new treatment for refractory chronic daily headache. eyeBrain pivotal trial. January 2016.

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1-Star: Obsessed with My Eyes

Those online reviews can be real knee-slappers—as long as they're someone else's.

By Montgomery Vickers, OD

Man, we all feel like we will live and die depending upon our online patient reviews, don't we?

These days, any patient who feels they did not have a stellar experience can easily and anonymously decide to burn you—and there are few, if any, repercussions.

Even if we have 600 five-star reviews, we all sit there and obsess over the single one-star review like this is the one review that just might take food off the table. And, truth be told, prospective patients do read those low reviews too.

The worst part of this is that the review does not even have to be true. The reviewer can say you turned into a green blob of antimatter during the examination and get away with it. Luckily, in some demographics the green blob deal might actually build your practice. You may want to post the review on a Comicon blog page.

Starting Over

When I moved to Texas, I was given a clean slate. Crazy reviews from West Virginia don't seem to be important to Texans. So, the lady "back home" who wrote that I "coldly did not care" about her eyes held no weight in Dallas.

Of course, I totally understand why she would presume I did not care about her eyes. After all, I did refuse to give her a new monthly trial contact lens so she could wear it for the next year like she had done the year before.

Ones for the Record Books

My team of researchers has found some of the most interesting optometrist reviews from the past year. Any names have been changed to protect the innocent—and the guilty:

- Dr. Schmolt has a very weird attitude. I would highly recommend him.
- Do you wear glasses? This office has them.
- My glasses gave me diarrhea.
- I never had to wear glasses until I went to Dr. Giffo's office.
- I did not like the glasses I did not get at Dr. Hylife's office.
- I just wish we could kidnap Dr. Woof and take him to an office with a decent staff.
- Since they cleaned my glasses I see much better.
- My sister used to date an eye doctor and she said these guys should never prescribe eye drops.
- I'm only giving him two stars, but my whole family goes every single year.
- I was allergic to the nosepieces of my new glasses. Went back four times and got four new glasses, all with allergic nosepieces.
- I recently had my very first eye exam, and this is the worst eye office I have ever seen.
- I always thought my eye doctor was cute. Then I got new glasses.
- Dr. Mimi gave me some good advice and I found my old glasses.
- I can only think of one reason you shouldn't go to Dr. Greasy: if you're not living.
- Despite the hassle and overcharges, they are awesome!
- There's no place like Crisper's Eye Care! Thank God!

We all love a great review. On the other hand, a not-so-great review can sometimes be very entertaining and educational, especially if it's about someone else. ■

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Zoster or Imposter?

With a virus known to masquerade, a detailed history and full exam could be your only shot at an early diagnosis. **Edited by Paul C. Ajamian, OD**

Q A 71-year-old female underwent cataract surgery and noticed swelling on the first day post-op. She believed she was having an allergic reaction to the prescribed eye drops. The swelling worsened by the following day, and to accompany it, she developed a red irritation on her left scalp. How can we connect the dots to home in on an early diagnosis before things get out of hand?

A While it was plausible for the patient to assume the swelling could point to an allergic reaction, it's the optometrist's job to ignore red herrings and to be on the lookout for the early signs of other conditions, especially those known to masquerade like herpes zoster ophthalmicus (HZO). Catching it in its prodromal phase can aid in preventing a full-blown outbreak by getting treatment started as soon as possible. It requires a discerning eye and thorough sleuthing.

Devil in the Details

"To start, we need to ask more questions," says Emily Love, OD, of Ophthalmic Consultants of Connecticut. "With any patient over 60 years old, we should ask if she received the Zostavax (Merck) vaccine. Maybe she has a history of cold sores or herpes? Perhaps she remembers experiencing tingling sensations prior to the appearance of the skin lesion?" asks Dr. Love. "It's also worth asking about other HZO triggers such as any recent exposure to sun, stress, extreme heat or cold or history with alcohol intake."

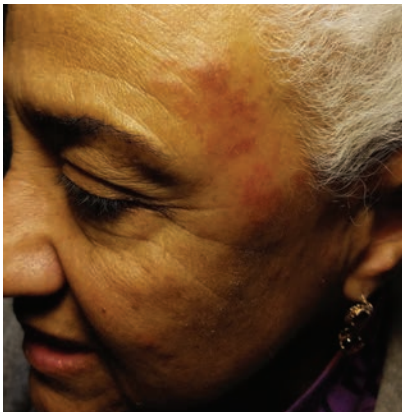

Fig. 1. HZO can manifest as a painful red skin irritation that always respects the midline.

Depending on how the patient responds, you can start crossing things off the list. "My differentials list for every patient includes tumor, herpes and fungal infection, and yes, I check them off every single time. Those are the three that will bite you in the butt if you miss them!" says Dr. Love. Here, making an early diagnosis of HZO is the goal. Once it's full blown, the virus manifests as an extremely painful vesicular rash.

"If, however, the patient is not yet showing HZO's characteristic presentation, you can continue to narrow your search by checking the patient's intraocular pressure," says Dr. Love. If it is in fact HZO, this test will most likely reveal elevation in the eye of the affected side. "Corneal sensitivity could also be decreased, and slit lamp might reveal previous corneal neovascularization, scarring or subepithelial infiltrates," says Dr. Love.

"Once it's full-blown, you can't miss it because HZO always, always respects the midline," she adds (*Fig. 1*). "Also, the herpes family manifests through the V1 ophthalmic division of the trigeminal nerve. If, however, the nasociliary nerve is involved, the patient would have a lesion on the end of the nose. That would be your other dead giveaway."

Table 1. Oral Antiviral Dosage

Famcyclovir	Acyclovir	Valcyclovir
500mg TID	800mg 5x/day	1000mg TID

The FAV Treatment

Once HZO is unveiled, the patient needs to start antiviral therapy immediately. "I've found that my zoster patients with chronic recurrences are okay with taking a pill everyday for prophylactic purposes," says Dr. Love. She even created a simple mnemonic—"Herpes is my FAV"—to recall the names of the three key drugs: *f*amcyclovir, *a*cyclovir and *v*alacyclovir. "That says it all," she adds. "Sometimes I'll even sing that phrase to my patients to lighten the mood, before discussing their treatment plan."

All joking aside, keep in mind that the road ahead can be rocky. "Don't forget that post-herpetic neuralgia can be incredibly painful for your patients," warns Dr. Love. "Recurrences can be common for a lot of folks, so prepare them, educate them and treat them immediately," she says. With a quick diagnosis and proactive treatment plan, the initial shock can dissipate with ease. ■

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A TIMELESS DESIGN

CooperVision® celebrates its revered toric lens design with the launch of MyDay® toric at SECO 2018

Designs can often be fleeting. In industries such as fashion, interior design, and architecture, new designs are adopted, replicated, popularized, and then they're out. But every so often, a design is so revolutionary—and so ahead of its time—that it lives on for much longer. It's timeless.

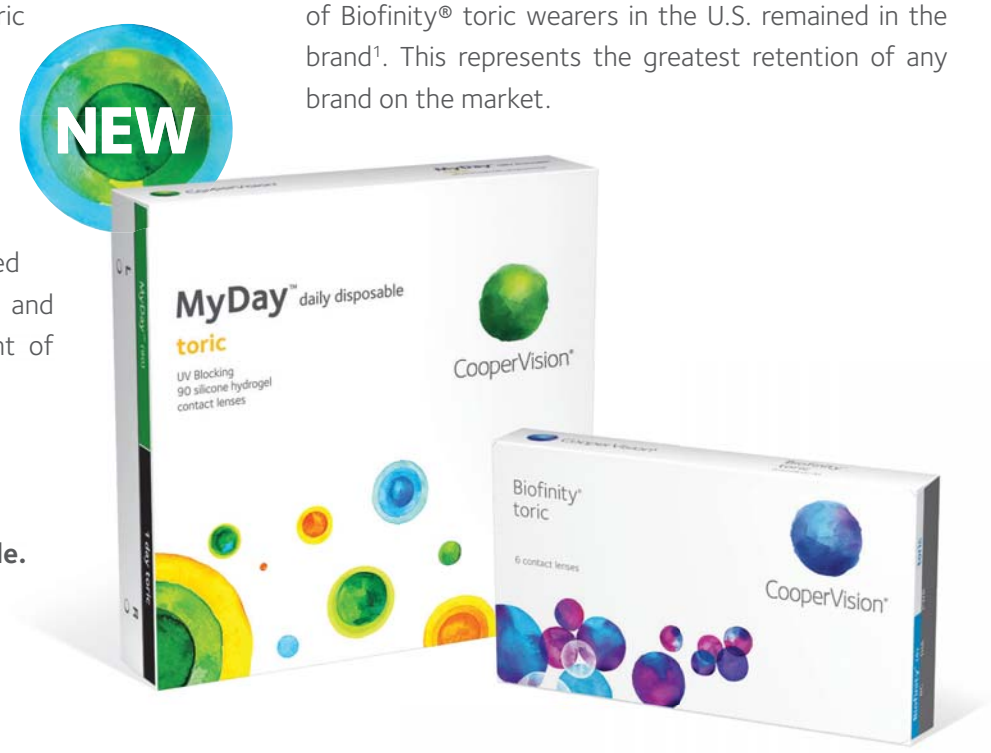
Such is the case with CooperVision's proven and widely respected Biofinity® toric design, known as Optimized Toric Lens Geometry™, which has been key to Biofinity® toric's trajectory to becoming the most prescribed toric lens in the United States[1]. Now, CooperVision has taken those trusted toric design features and applied them to its premium silicone hydrogel 1-day toric contact lens, recently unveiling the long-awaited MyDay® toric.

The celebration was on at SECO 2018 in Atlanta, where CooperVision showcased the innovation of MyDay® toric by honoring the timeless toric design that brought this new lens to life. The booth was adorned with conversation-starters, including a head-turning performance car, ageless fashion accessories, and an iconic smartphone. It all highlighted the concept of timeless design and its importance in the development of MyDay® toric.

But what makes a design timeless? Essentially, it boils down to four characteristics: **Simple. Memorable. Meaningful. Relevant.**

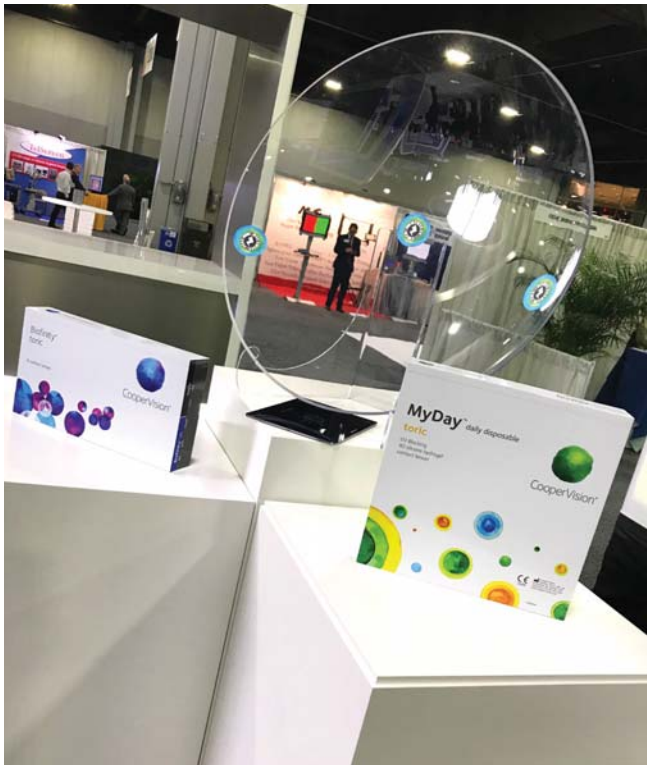
When CooperVision introduced Biofinity® toric, it was its Optimized Toric Lens Geometry™ that set it apart from every other lens on the market[2]. It provides uniform ISO thickness, an optimized ballast band design, a large toric optic zone, and a smooth, continuous surface. The design itself is complex, but this unique combination of components results in a stable, **simple** fit for practitioners. And Biofinity® toric set a new standard in toric contact lenses, one that remains unmatched today. It has proved to be **memorable**, and its timelessness is what inspired CooperVision to bring the same design features into the 1-day modality with MyDay® toric.

For patients, CooperVision's toric design delivers improved vision stability, greater visual acuity, faster lens settling, and excellent rotational recovery[2]. Biofinity® toric continues to keep doctors and patients satisfied; over the last 18 months, more than 93% of Biofinity® toric wearers in the U.S. remained in the brand[1]. This represents the greatest retention of any brand on the market.



And MyDay® toric daily disposable contact lenses are made with Smart Silicone™ chemistry, which transports oxygen with such efficiency that the lens is just 4.4% silicon—the lowest percentage of silicon found in a silicone hydrogel, 1-day lens. By using less raw silicon, there is more room in the lens for hydrophilic (water-loving) material, resulting in improved surface wettability, higher water content, and lower modulus for a softer, more comfortable lens-wearing experience. In fact, MyDay® is CooperVision's softest ever silicone hydrogel 1-day contact lens.

All of these benefits add up to a **meaningful** lens-wearing experience.



With an increasing number of patients moving into 1-day lenses—or wanting to because of their comfort, convenience, and healthier lens-wearing experience—eye care practices are presented with an opportunity to grow their practices with a groundbreaking toric lens. There has never been a greater need for a lens like MyDay® toric, an advanced contact lens that builds upon a long-revered toric design that remains **relevant** today.

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1. Q3 2017 US industry data on file 2. Comparison of fitting stability of the different soft toric contact lenses. Contact Lens & Anterior Eye 37 (2014) Hamed Momeni-Moghaddam et al. Optimised Toric Lens Geometry™ compared to available prism ballast, precision balance and accelerated stabilization toric lens designs.

A Sign of Trouble

How to locate and identify a telltale finding that could prevent a near-future retinal detachment. **By Bisant A. Labib, OD**

In cases of acute posterior vitreous detachment (PVD), we need to be vigilant for any indication that a retinal problem may be present or imminent. Careful retinal exam is of utmost importance in determining the risk of any PVD-associated pathology, such as a tear, detachment or vitreomacular adhesion. While advanced diagnostic techniques require skill to master and are time-consuming to perform, there is also a quick, reliable way optometrists can identify a retinal tear or break: Shafer's sign. Once you know what you're looking for, you can't miss it, and it might just make all the difference.

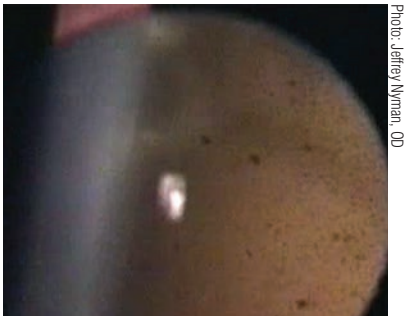

Photo: Jeffrey Nyman, OD

Shafer's sign, or "tobacco dust," found in a slit lamp ophthalmoscopy test.

In the Wake of PVD

Acute PVDs occur in 63% of patients over age 69, and 18% of these cases will experience an associated retinal break.[2] Typically, patients complain of flashes or floaters. The latter result from aggregation of collagen into visible fibers, blood in the vitreous cavity or the glial remnant (Weiss ring) following detachment around the optic disc. While the occurrence of flashes is less understood, they are likely due to vitreous traction, specifically the temporary separation of vitreous and retina induced by eye movements.[3]

While these common patient-reported symptoms are critical to the diagnostic process, a subsequent retinal exam is warranted in patients with acute PVD.[4] Without taking immediate action, the persistent vitreoretinal traction that caused the initial tear can continue to lift the retina from underneath and cause a detachment.

To identify these breaks, eye care practitioners may choose to perform a retinal peripheral examination using three-mirror gonioscopy or binocular indirect ophthalmoscopy with scleral depression. However, these procedures require great clinical skill and practice, and that's why we turn to Shafer's sign for help.

Shafer's Sign

Also called "tobacco dust," Shafer's sign refers to the presence of a collection of brown pigmented cells in the anterior vitreous following a PVD. First identified in 1965, the sign is best observed through slit lamp exam by sending a narrow, bright beam behind the posterior lens to focus on and illuminate the dark vitreous cavity.[5] By finding and accurately identifying this collection of cells, the optometrist is often one step ahead of patient symptomology and can further secure their diagnosis of a retinal break.[6] From here, it's time to set out on the road to preventing a full detachment.

Heed the Warning

While the origin of these pigment granules is unknown, they are thought to come from the shearing force of the break in the retinal pigment epithelium (RPE). During a PVD, as the vitreous forcefully tugs on the retina, the retinal break can occur. Next, the liquefied vitreous breaks down intercellular bonds in the RPE, causing brown pigment to be released into the vitreous cavity. The mark left by these free-floating cells is what we know today as Shafer's sign.[5,7] This theory has been supported by fluorescein angiography testing, revealing a break in the RPE in cases of anomalous PVD.[7]

Table 1. Types of Cells Found in the Anterior Vitreous and their Clinical implications

Abnormal Vitreous Cells	Source	Clinical Indication
Brown (Shafer's sign) cells	Pigment from RPE of retina	Retinal break
Red cells	Red blood cells from hemorrhage	Retinal break or proliferative retinal process
White cells	Inflammatory white blood cells	Vitritis, pars planitis

Many studies suggest that Shafer's sign is pathognomonic for a retinal break, assuming the patient has not previously undergone ocular surgery.[1,2,8] If and when these pigment granules are observed in the vitreous, the likelihood of an associated retinal tear is 52 times higher than in cases where there is absence of these signs.[2] Additionally, as 25% to 90% of retinal tears are expected to proceed to a retinal detachment, missing this warning sign could put your patient's vision in grave danger.

Keep in mind that while the brown pigment granules in Shafer's sign point to retinal breaks, red-pigmented cells in the vitreous will indicate vitreous hemorrhage. When these red cells are found following an acute PVD, there's a 70% correlation with retinal tears. For this reason, both brown and red pigmented cells are causes for concern and further investigation.[5] Instead, if white, or non-pigmented, cells are observed in the anterior vitreous, you are not dealing with a retinal break or tear. These white cells may indicate an inflammatory condition.[7]

Lastly, know that while the presence of Shafer's sign confirms the presence of this associated retinal pathology until proven otherwise, the absence of Shafer's sign in patients with acute PVD does not necessarily exclude the possibility of a retinal break or detachment.[5] Additional testing should carry on as needed in these cases, whether or not their slit lamp exam shows the telltale tobacco dust.

Going Forward

While advanced technical procedures such as binocular indirect ophthalmoscopy with scleral depression may require years of practice, Shafer's sign offers a quick, reliable method for identifying breaks and tears before they cause irreversible damage. As it can be easily identified through routine slit lamp ophthalmoscopy, all eye care practitioners possess the skill required to locate Shafer's sign and heed its warning. ■

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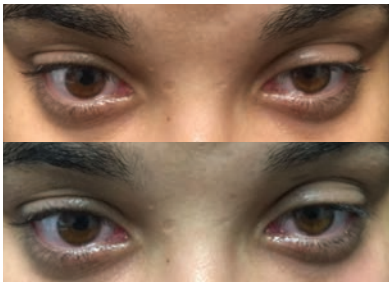
The Upside of a Drooping Lid

A series of noninvasive in-office diagnostic tests can take the "grave" out of myasthenia gravis. **By Michael Trottini, OD, and Michael DelGiodice, OD**

A 20-year-old Hispanic female presented as an emergency office visit with complaints of progressive drooping in her left upper eyelid of 11 months' duration. She noted it seemed to worsen by the end of the day, particularly after computer use. She denied ocular pain, headache, diplopia, muscle weakness, difficulty swallowing and breathing. Her medical, ocular, social and family histories were unremarkable. Unaided visual acuity was 20/20 OD and OS. Interpalpebral apertures were 7mm OD and 5mm OS. Marginal reflex distance was 5mm OD and 3mm OS, and levator function was 15mm OD and 12mm OS. Pupils were equal, round and reactive to light without relative afferent defect; they measured 6mm and 3mm in dim and bright illumination in both eyes. She demonstrated 10/10 color plates in each eye. Intraocular pressure measured 13mm Hg OD and 16mm Hg OS. Confrontation testing was normal. With the exception of left upper eyelid ptosis and mild, incomitant left abduction deficit, the anterior segment exam was unremarkable.



Top photo: Left lid before ice test. Bottom: Same lid after test, showing minimal improvement.

Oh, MG!

Patients presenting with variable weakness of the eyelids and eye muscles who show no signs of cranial nerve palsy raise flags for myasthenia gravis (MG). The patient also fit the demographic profile for the condition, occurring most commonly in women under age 40. As she demonstrated key symptoms in her left eyelid, we considered ocular myasthenia gravis (OMG) and ran a series of in-office tests to narrow and confirm a diagnosis. Aim to employ these when working up an OMG suspect:

• ***Lid-fatigability test.*** This quick, non-invasive exam helps determine whether and to what degree the affected lid experiences fatigue. Ask the patient to sustain a prolonged upgaze fixation and observe eyelid position. Our patient's left ptotic lid showed considerable increase in ptosis after one minute. The orbicularis muscle was also assessed.

• ***Cogan's lid twitch.*** Next, we asked her to look down and then move the direction of gaze back up to her primary position.[1] If a patient overshoots upward before returning to their resting position, they've tested positive—as ours did.

• ***Eyelid retraction.*** Another telltale sign of OMG manifests in eyelid retraction, occurring when one lid rests in ptotic position and the other retracts. As Hering's law says, yoked muscles work synergistically. So, while the ptotic eyelid sits low, the patient is constantly using the orbicularis muscle to raise the affected lid. This extended effort causes over-action of the contralateral, unaffected eyelid, which will manifest as contralateral lid retraction. Manually closing the ptotic eyelid resolves the contralateral lid retraction. Our patient tested positive.

• ***Orbicularis "squeeze test."*** Ask the patient to forcefully close their eyes while you attempt to open them. In our patient, both eyelids were easily opened despite her strong attempt to squeeze them shut. This confirmed bilateral weakness of the orbicularis muscle. Version testing further revealed subjective horizontal diplopia during extreme left gaze. A basic cover test carried out in the nine cardinal positions of gaze found her orthophoric in all positions, except for six prism diopters of left esotropia in extreme left gaze.

• ***The sleep and ice tests.*** At home, the patient can self-administer the sleep test by closing the eyes for 30 minutes and then noting whether there is any improvement in ptosis, diplopia or both. If so, there is a strong indication for OMG. The ice test involves placing an ice-pack over the affected eyelid for two to five minutes.[2] If there is at least a 2mm elevation, or improvement, of the ptosis, the test is positive. Cooling the eye reduces acetylcholinesterase (AchE), thereby increasing acetylcholine (Ach) within the neuromuscular junction (NMJ). The sensitivity and specificity of the ice test is 76.9% and 98.3%, respectively.[3]

Communication Breakdown

Myasthenia gravis is an autoimmune disease of the NMJ, the site of communication between nerve bundles and muscle fibers. It causes classical symptoms of variable muscle weakness worsened by fatigue. MG may affect any age group but is less likely in patients over age 70 and more common in females.[2,10]

There are two forms of the disease: generalized and ocular. The former involves the bulbar, limb and respiratory muscles.[2] The latter is a subtype confined to the EOMs, levator muscle and orbicularis oculi.[2]

In all forms of MG, antiacetylcholine receptor antibodies (AChR-Abs) block receptors on muscle fibers from receiving Ach molecules. This results in defective signal transmission and poor muscle contraction. Because EOMs have fast-twitch fibers that require constant binding of Ach to muscle fibers, the earliest stages of the disease may present with variable ptosis and diplopia. These are the initial signs in more than 50% of MG patients.[11] Within two years, 50% to 80% of those affected with OMG will convert to GMG.[12,13]

In addition to ptosis and orbicularis weakness, ocular motility deficits are common symptoms of OMG. All EOMs may be affected, with the medial rectus and superior rectus showing up as the most common.[14] The motility pattern is often variable and fatigable but can mimic nerve palsies, gaze palsies or internuclear ophthalmoplegias.[11] Additional testing of saccades may reveal intrasaccadic fatigue and a decline in saccadic velocity.[15]

Running both tests produces a larger change in lid position than the sleep test alone.[4] Here, we ordered the ice test to evaluate improvement in the left upper ptosis and diplopia, which confirmed ocular symptoms of OMG.

• ***Pharmacologic testing.*** Edrophonium (Tensilon) prevents the breakdown of Ach by inhibiting AChE within the NMJ. In the so-called Tensilon test, following administration of the drug, OMG patients will typically demonstrate improved muscle strength in either the levator or the affected extraocular muscle (EOM). The sensitivity of the Tensilon test is 95% in generalized myasthenia gravis (GMG) and 86% for OMG.[5] Tensilon must be administered by a provider licensed to perform intravenous injections, typically a neurologist. If the diagnosis is still in question following the Tensilon test, neostigmine—which has a longer duration of action—can be given as an intramuscular injection by appropriately qualified staff.[2]

• ***Neurostimulation tests.*** Repetitive nerve stimulation studies and single-fiber electromyography (SFEMG) involve stimulation of nerve bundles and recording action potentials. SFEMG is the more sensitive test for detecting abnormal neuromuscular transmission. It has an 85% to 100% sensitivity for OMG when used on the frontalis or orbicularis muscle, and a sensitivity of 91% to 100% in GMG.[7,8]

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Next Steps

Given our patient's progressive, fatigable left upper lid ptosis, incomitant left abduction deficit, orbicularis muscle weakness, positive ice test, normal pupils and lack of respiratory or peripheral muscle weakness, we diagnosed OMG. We then ordered AChR-Ab levels as well as thyroid function tests, since thyroid disease can accompany MG, and referred the case to neurology. Two weeks later, the neurologic exam confirmed our findings by Tensilon testing.

Treatment aims to improve both general and ocular muscle weakness, achieve disease remission and slow or prevent progression to GMG.[9] Corticosteroids are the most commonly used medication for both OMG and GMG. Our patient is being managed on a regimen of 100mg pyridostigmine (an AChE inhibitor) and 20mg oral prednisone.

Despite treatment, many patients still exhibit bothersome diplopia, ptosis or both. Supportive measures include temporary prism or occlusion therapy. Surgery can be considered in patients whose symptoms are stable for a minimum of six months and immunosuppressive therapy can be considered in all patients with MG as monotherapy or adjunct therapy with corticosteroids and AChE inhibitors.

The optometrist's role is to monitor disease status and manage the ophthalmic symptoms with prism, occlusion therapy or surgical referral. ■

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The Buzz on Electrodiagnostics

While increasingly useful in the clinic, don't assume coverage is automatic.

By John Rumpakis, OD, MBA, Clinical Coding Editor

In the world of medical diagnostic testing, eye care professionals are fortunate to have some of the most interesting and progressive innovation. Two examples are visual evoked potential (VEP) and electroretinography (ERG). What were once cumbersome and inconvenient testing routines are now delivered daily in many eye care offices with ease.

Unfortunately, coverage policies from third party carriers don't always enjoy the same level of progress as the technological innovations. Thus, be aware of carrier policies regarding VEP and ERG before you administer the tests so you can follow the appropriate rules for non-covered services.

Keep the Patient Informed

If a service is non-covered or you have a specific reason for believing a service will be non-covered, you must inform the patient and give them a choice prior to performing the test with an advanced beneficiary notice (ABN) form. The ABN allows you to identify the test being performed, the reason you believe it will not be covered and the cost the patient can expect to pay should it not be covered or submitted for coverage. This form requires that the patient specify if they want you to proceed with the testing with full knowledge, indicated by their signature, that they may bear full financial responsibility for the cost.

When you have a completed ABN form, you would typically add modifier -GA or -GX to the CPT code describing the test, indicating that you have a signed ABN on file. This allows the carrier to properly transfer financial liability to the patient should the claim be filed and subsequently denied.

Policies and Expectations

When it comes to electrodiagnostics in eye care, claims are often denied, as the scientific evidence of clinical applicability is still playing catch-up to coverage policies, or vice versa.

Typical policy coverage for VEP—CPT code 95930. This is appropriate for:

- Confirming diagnosis of multiple sclerosis when clinical criteria are inconclusive.
- Detecting optic neuritis at an early, subclinical stage.
- Evaluating optic nerve diseases, such as: ischemic optic neuropathy; pseudotumor cerebri; toxic or nutritional amblyopia; neoplasms compressing the anterior visual pathways; optic nerve injury or atrophy; and to rule out hysterical blindness.
- Monitoring the visual system during optic nerve (or related) surgery (short-latency evoked potential studies).

Typical policy coverage for ERG. This includes: to diagnose loss of retinal function or distinguish between retinal and optic nerve lesions such as: toxic retinopathies; diabetic retinopathy; ischemic retinopathies such as central retinal and branch vein occlusion and sickle cell retinopathy; autoimmune retinopathies such as cancer- and melanoma-associated retinopathies and acute zonal occult outer retinopathy; retinal detachment; assessment of retinal function after trauma, especially in conditions where the fundus cannot be visualized; absent b-wave indicates abnormality in the bipolar cell region; retinitis pigmentosa and related hereditary degenerations; retinitis punctata albescens; Leber's congenital amaurosis; choroideremia; gyrate atrophy of the retina and choroid; Goldman-Favre syndrome; congenital stationary night blindness; X-linked juvenile retinoschisis; achromatopsia; cone dystrophy; disorders mimicking retinitis pigmentosa; and Usher syndrome.

Some carriers specifically deny VEP or ERG for glaucoma diagnosis or management as investigational in nature. While you may rightly believe these tests are critical to the early diagnosis of glaucoma or for the management of other diseases, the carrier may not agree with you—and their policies are what you are governed by. Thus, properly providing correct information to your patient and giving them a choice prior to providing the tests is critical to remain compliant under your provider agreement.

Electrodiagnostics are an exciting opportunity, and knowing the rules allows you to provide the very best care while preserving your patients' right of choice to assume financial responsibility of their care. ■

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Zeaxanthin: The Super Antioxidant

What is Zeaxanthin?

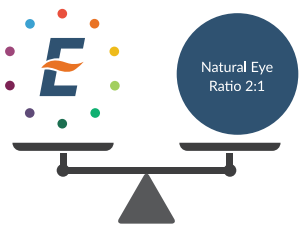
Zeaxanthin is a member of a group of compounds known as carotenoids.* As a group, the carotenoids function like internal sunglasses, shielding underlying light-sensitive tissues from harmful blue light. **Zeaxanthin is the key nutrient responsible for protecting crisp, clear vision needed for reading, seeing faces, and driving at night.** The retina actively seeks zeaxanthin because it's necessary to protect the tissues responsible for central vision. As an antioxidant, zeaxanthin works within the layers of photoreceptors to protect their longevity. In general, Americans don't get enough dietary zeaxanthin from food alone, and many find it easier to take a daily supplement to fulfill this need. To best support their eyes, patients will need to take at least 8 mg of dietary zeaxanthin per day.

Is It Safe?

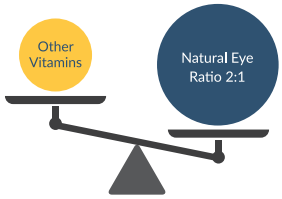
Zeaxanthin can be found in several commonly eaten foods, including kale, peppers, and broccoli, and has been safely consumed for hundreds of years. After reviewing the clinical evidence, a panel of experts working with the World Health Organization[1] (WHO) established an acceptable daily zeaxanthin intake of up to 2 mg per kilogram of body weight. Based on the weight of an average American, the upper limit is 180 mg per day.** Along with an extensive "New Dietary Ingredient" (NDI) application, there are numerous published clinical studies that support the safety of higher doses of dietary zeaxanthin. [2,3,4]

Why 8 mg?

Zeaxanthin is used selectively by the retina to protect the delicate tissues responsible for central vision, meaning the eye naturally places importance on this super antioxidant. **The eye's natural ratio of zeaxanthin to lutein is 2:1.** For optimal protection, it's best to prescribe nutraceuticals that mimic this ratio, or at least contain the amounts of zeaxanthin to match those of lutein.



EyePromise Restore provides 12 mg of Zeaxanthin and Lutein



Other vitamins lack the amount of dietary zeaxanthin necessary to mimic the eye's natural ratio.

EyePromise® is the only line of eye health nutraceuticals that offers this amount of dietary zeaxanthin, providing patients premium protection.

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The Clinical Impact of Higher Levels of Dietary Zeaxanthin

In a study[5] published in 2012, researchers tested 10 mg of dietary zeaxanthin and lutein compared to two other lutein-only study arms among patients with early age-related macular degeneration (AMD). In one year, the study authors found that the group administered both zeaxanthin and lutein had the greatest retinal function improvement when measured with multifocal electroretinography (MfERG).

In the year-long Zeaxanthin and Visual Function Study[6] (ZVF), participants supplementing with a higher dose (8 mg) of dietary zeaxanthin daily achieved:

- Improved high contrast near visual acuity of 8.5 letters or 1.5 lines
- Clearing of central scotomas
- Improved foveal shape discrimination
- Improved night driving skills

These results demonstrate positive changes in visual function as well as structural improvements.

A 2-year, 521-subject study published by Dr. John Herman[7] administering 8 mg of zeaxanthin showed the following results:

- 97.8% of participants improved or stabilized their AMD status.
- 88.3% of participants achieved an MPOD increase of at least 30%.
- 67.9% reported improved glare recovery.
- 62% reported contrast improvement.

This study also demonstrates enhancements in structure and function.

Drs. Herman, Richer, and their colleagues have tested higher levels of dietary zeaxanthin with their patients and have seen improvements with both subjective and objective testing. These results are encouraging and demonstrate potential for improving patients' quality of life.

Leo Semes, OD, earned his OD from Pennsylvania College of Optometry (PCO) and completed residency at The Eye Institute of PCO. He is a former Professor of Optometry at UAB, a fellow of the AAO, and a member of the AOA. Dr. Semes is a founding fellow of the Optometric Retina Society. In 2015, he was recognized as one of the 50 "most influential individuals in eye care" by Optometric Management magazine and received the Educator of the Year award from the Alabama Optometric Association. Dr. Semes was recognized with the Dean's Distinguished Service award from the UAB School of Optometry and is an editorial board member for Review of Optometry.

Financial Disclosures: Dr. Semes serves on the EyePromise Scientific Advisory Board and received honoraria and consulting fees from EyePromise.

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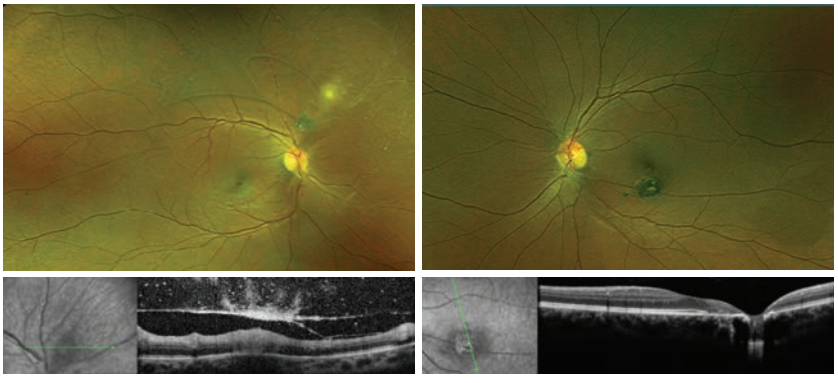
**https://www.google.com/search?client=safari&rls=en&q=what+is+the+average+weight+of+a+50+year+old+american&ie=UTF-8&oe=UTF-8. Accessed January 12, 2018.

Test Your Diagnostic Acumen

Do you have what it takes to solve these five puzzlers?

By Mohammad Rafieetary, OD, and Jessica Haynes, OD

Complex features of retinal and chorioretinal diseases can turn the diagnostic process into a challenge. That's why learning to identify the nuances that distinguish one disease from another is so critical. In this article, you'll find five puzzling cases, each accompanied by a self-test designed to build your retinal disease diagnostic chops. (*For answers, see pg. 42.*)



Figs. 1 & 2. Fundus photo and OCT of the right eye (left) and left eye (right).

CASE 1: Float On

A 34-year-old black female presented to the clinic for new-onset blurry vision and increased floaters in the right eye. She reported that she was in good medical health and was not on any systemic medications. She was nearsighted and remembered doctors' reports from prior eye examinations noting retinal scars in both eyes. She noted that this had not changed for a number of years.

The patient had an airbag-related head injury as a result of a motor vehicle accident (MVA) six months prior to this examination. Entering visual acuities were 20/25 OD, 20/25 OS. Chair skills were normal with full to finger counting in both eyes, she demonstrated full extraocular motilities in both eyes, and her pupils were equally reactive to light with no afferent pupillary reaction.

Intraocular pressures (IOPs) were 12mm Hg OD and 10mm Hg OS. The patient's anterior segment examination was normal, while an evaluation of her posterior segment did reveal abnormal findings (*Figures 1 and 2*).

1. Which of the following could best explain the new-onset complaint of increased floaters in the right eye?
a. She has had an acute posterior vitreous detachment.
b. The floaters represent normal age-related vitreous condensation.
c. She has developed a rhegmatogenous retinal detachment.
d. The floaters arose from vitritis associated with the pre-existing retinal findings.

2. What is the most likely description of the creamy white-colored lesions superonasal to the optic nerve in the right eye?
a. Superficial retinitis.
b. Cotton wool patch.
c. Coalesced drusen.
d. Retinal exudation.

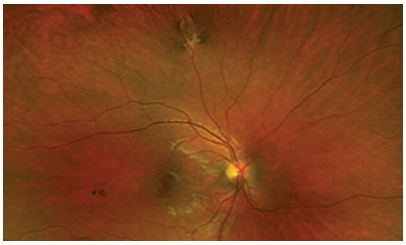


Fig. 3. Retinal imaging of right eye.

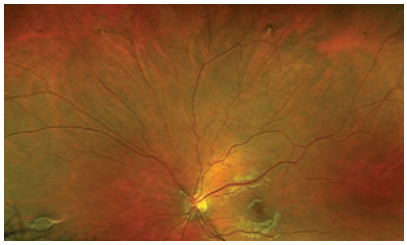


Fig. 4. Retinal imaging of left eye.

3. What is the most likely cause of the funduscopic findings?
a. Retinal vascular disease.
b. Infectious disease.
c. Genetic condition.
d. Blunt force trauma.

4. Which of the following is the most likely cause of this symptomatic event?
a. The recent history of MVA.
b. Natural aging process.
c. Reactivation of a preexisting condition.
d. No associated cause.

5. Which of the following laboratory tests would be most useful in confirming the patient's diagnosis?
a. Angiotensin-converting enzyme.
b. Antinuclear antibodies.
c. Toxoplasmosis serology.
d. Histoplasmosis serology.

Case 1 Discussion

The patient has a reactivation of ocular toxoplasmosis, the most common infection of the posterior segment.[1] In its active phase, ocular toxoplasmosis causes a superficial retinitis and associated vitritis.[2] This typically manifests as a fuzzy or cloudy white overlay on the infected retina, commonly recognized as "headlights in the fog."[2] Retinal vasculitis may also be present.[2]

When the superficial retinitis eventually advances into deeper tissue, it causes chorioretinal scarring.[2] New areas of infected retina often flare near old chorioretinal scars, as in this patient, and are referred to as satellite lesions.[2]

This condition is caused by the parasitic protozoan *Toxoplasma gondii*.[1,2] Toxoplasmosis can be acquired at any stage of life or contracted congenitally.[2] Transmission to humans can occur through undercooked meat, compromised water sources in endemic areas, ingesting dirt or through exposure to cat feces.[2] Congenital cases occur via transplacental transmission from an infected mother to the unborn fetus.[2]

Typically, ocular toxoplasmosis will be diagnosed based on clinical findings alone, but serologic testing is performed to confirm the first-time diagnosis. IgG and IgM antibodies for toxoplasmosis can be ordered in cases with atypical presentations, while IgA can be helpful in determining congenital cases.[3] IgG antibodies appear within one to two weeks of infection and reach their peak one to two months post-infection.[3]

Table 1. Serologic Testing and Diagnosis

IgM	IgG	Interpretation
Negative	Positive	Past infection
Negative	Negative	No infection; early infection
Positive	Negative	Early infection
Positive	Positive	Current infection; reactivation of chronic infection

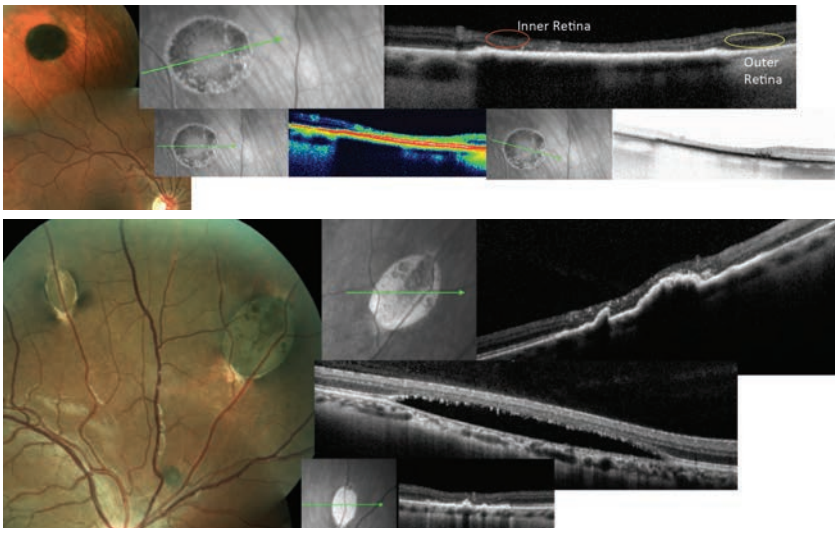


Fig. 5. The top images show typical CHRPE lesion with associated typical OCT findings: thickening and hyperreflectance of the RPE, atrophy of the outer retina with sinking or caving of the inner retina and reduced reflectance of the choroid. The bottom images show typical RPEH-FAP lesions, as well as the variable OCT presentations.

While antibody loads will decrease, they tend to persist at low levels throughout life. IgM antibodies appear within the first week of infection and rapidly increase. Afterwards, IgM decreases to a low level and will disappear after several months (*Table 1*).[3]

CASE 2: Smells Like Teen Spirit

A 14-year-old Caucasian female presented for an evaluation of retinal holes in both eyes. She was in good health and although her ocular history was remarkable for myopia, her medical history was unremarkable. Her mother added that there was no pertinent family history. Her visual acuities were 20/20 OD, OS. She had normal chair skill testing with full confrontation visual fields, full extraocular motilities and pupils that were equally round and reactive to light with no afferent pupillary defect.

The patient had normal anterior segment findings. IOPs were 15mm Hg OD, OS. Retinal findings are available (*Figures 3 and 4*).

1. The retinal lesions represent which finding?
a. Retinal holes.
b. Hypertrophic retinal pigment epithelium (RPE).
c. Choroidal nevi.
d. Choroidal metastasis.

2. What special ocular imaging/testing would be most helpful in the diagnosis?
a. Visual field.
b. Optical coherence tomographyimaging (OCT) over lesions.
c. OCT angiography.
d. Scleral depression.

3. What specific history should be further questioned in this case?
a. Family history of melanoma.
b. Family history of retinal detachment.
c. Family history of colorectal cancer
d. History of autoimmune disease.

4. What is the proper management of the retinal lesions?
a. Retinal laser.
b. Intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents.
c. Intravitreal steroids.
d. Observation.

5. What referral is necessary?
a. Neurologist.
b. Dermatologist.
c. Gastroenterologist.
d. Rheumatologist.

Case 2 Discussion

The bilateral lesions noted on fundus examination are the result of hypertrophy of the RPE. In the past, these lesions have been inaccurately defined as congenital hypertrophy of the retinal pigment epithelium (CHRPE). As these lesions are acquired, they are best referred to as RPE hamartomas associated with familial adenomatous polyposis (FAP), abbreviated as RPEH-FAP. [4]

FAP is an autosomal dominantly inherited condition in which patients develop multiple polyps in the colon by their teenage years. Left untreated, these patients have almost 100% chance of developing gastrointestinal malignancy.[4] When FAP combines with non-GI manifestations such as osteomas, dermoid tumors, cutaneous cysts and other neoplasms, it is known as Gardner syndrome.

It is important to recognize the features that distinguish CHRPE lesions from RPEH-FAP. CHRPE, which are mostly benign and have no systemic association, present as unilateral, solitary round lesions with variable pigmentation (*Figure 5*). They are flat without choroidal involvement or overlying serous detachments. On OCT, a thickening of the RPE will be observed. In areas of lacunae within the CHRPE, thinning of the RPE may exist. Atrophy of the outer retina with excavation of the inner retina over the lesion may also occur (*Figure 5*).[5]

RPEH-FAP lesions, on the other hand, are bilateral, multiple, oval or pisciform-shaped and have hypopigmented tails that point posteriorly. On OCT, these lesions may demonstrate choroidal elevation, excavation or both and have overlying shallow serous detachments (*Figure 5*).[6] While these RPE lesions themselves do not require treatment, clinicians must refer patients to a gastrointestinal specialist to examine for colon polyps. Early detection and intervention could prevent cancerous formation.[4]

Following the fundus examination, we questioned the patient's accompanying family members about the possibility of a family history of colorectal disease and cancer. The mother reported that both the patient's father and maternal grandmother have had a history of colon cancer, for which the patient's father was currently under treatment. The patient was referred to a pediatric gastroenterologist for further management.

CASE 3: Even Flow

A 29-year-old white male was referred for retinal detachment in his left eye. The patient complained of gradual vision loss in his left eye over the last two months. Medical, past ocular and family histories were all unremarkable. The patient's best-corrected visual acuities were 20/20 OD, 20/200 OS. Chair skills, confrontation visual fields and extraocular motilities were all normal. His pupils were equally round and reactive to light with no afferent pupillary defect. IOPs were 15mm Hg OD, 18mm Hg OS.

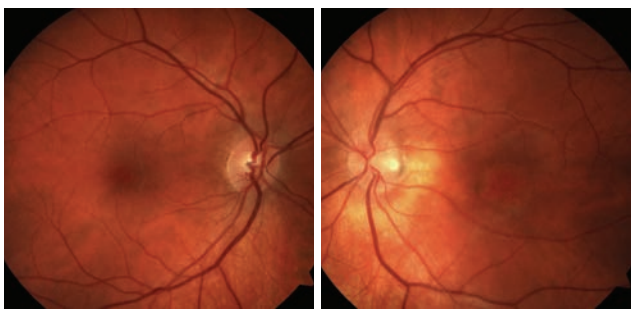


Fig. 6. Fundus photographs of the right and left eyes.

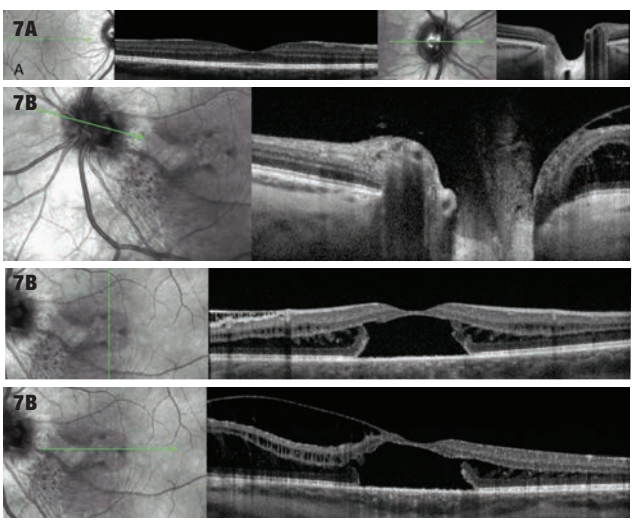


Fig. 7. (A) OCT cross sections through macula and optic nerve of right eye. (B) OCT cross sections through macula and optic nerve of the left eye.

Anterior segment findings were normal. Posterior segment findings and OCT findings are depicted (*Figures 6 and 7*).

1. What description is consistent with the macular OCT of the left eye?

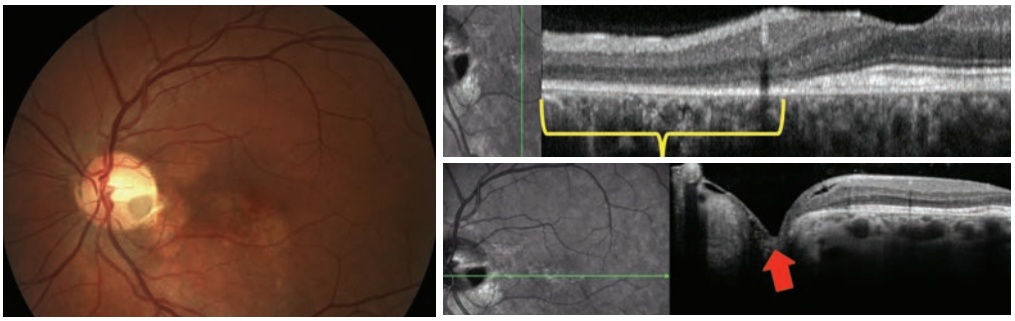


Fig. 8. This patient has an optic disc pit without intraretinal or subretinal fluid. Outer retinal disruption (section in yellow brackets) exists indicative of possible serous detachment in the past. Cavitation in the area of the pit is shown with the red arrow.

a. Choroidal elevation.
b. Cystoid macular edema.
c. Intraretinal fluid and localized serous retinal detachment.
d. Macular hole with vitreomacular traction.

2. The patient's optic nerve appearance is consistent with which of the following?
a. Glaucomatous cupping.
b. Optic disc pit.
c. Temporal disc pallor.
d. Optic nerve coloboma.

3. Which of the following is the most likely cause of patient's vision loss?
a. Rhegmatogenous retinal detachment.
b. Choroidal neovascular membrane.
c. Serous retinal detachment.
d. Cystoid macular edema.

4. Which of the following is the most correct diagnosis here?
a. Central serous chorioretinopathy.
b. Choroidal neovascular membrane.
c. Vitreomacular traction syndrome.
d. Optic nerve pit associated maculopathy.

5. Which of the following is the most appropriate treatment?
a. Observation.
b. Topical nonsteroidal anti-inflammatory eye drops.
c. Intravitreal anti-VEGF injection.
d. Surgical vitrectomy.

Case 3 Discussion

This patient has optic disc pit maculopathy. Optic nerve cavitation anomalies exist as a spectrum of conditions that include optic disc pits, morning glory syndrome and optic nerve colobomas.[7] With no signs of gender predilection, the prevalence of optic disc pit has been estimated to occur in one out of 10,000 people, and bilateral involvement is found in 10% to 15% of these cases.[8]

These anomalous disc formations may result in visual field defects, but can also lead to maculopathy when fluid accumulates within the layers of the retina (intraretinal fluid) or in the subretinal space (subretinal fluid or serous retinal detachment). The source of this fluid is still up for debate.[9]

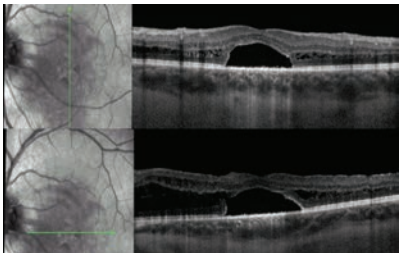


Fig. 9. Macular OCT one month post-op.

Central vision may be affected by the presence of this subretinal and intraretinal fluids. While the former is often not connected to the disc, the latter typically is.[7] OCT imaging of the macula, papillomacular bundle and the optic nerve is helpful in demonstrating the macular changes as well as possible connection of fluid to the optic nerve. It may also reveal anomalous nerve cavitations (*Figure 8*).

It is important to differentiate optic disc pits from cupping in glaucoma and to monitor these patients for maculopathy. In those with maculopathy, it is necessary to differentiate macular fluid from other causes of retinal edema, such as diabetic macular edema and central serous retinopathy. Patients with optic disc pit that do not show intraretinal fluid or serous retinal detachment do not require treatment. OCT is quite effective in detecting the presence of serous detachments and macular involvement.[10]

As progressive maculopathy can result in vision loss, surgical intervention may be advised.[11,12] This patient underwent pars plana vitrectomy with subthreshold laser temporal to the disc, plus gas injection. At one month post-op, the patient had objective visual improvement as well as structural improvement demonstrated on OCT (*Figure 9*).

CASE 4: Bring Me to Life

A 30-year-old Caucasian female presented to the clinic with recent-onset vision loss and sparkling lights in her left eye for two weeks. She reported taking Prozac (fluoxetine, Eli Lilly) for depression, but had no

additional medical conditions. She's worn lenses for myopic correction since age 13. Her best-corrected entering acuities were 20/20 OD and 20/60 OS. She had full confrontation visual fields and full extraocular motilities. Her pupils were normal with no afferent pupillary defect. IOPs measured 15mm Hg OD and 15mm Hg OS.

Anterior segment findings were normal OU. The posterior segment was normal in the right eye, while OS findings from fundus photography, fluorescein angiography (FA), indocyanine green angiography (ICGA), fundus autofluorescence (FAF) and OCT were suspicious (*Figure 10*).

1. The white lesions seen on fundus examination represent alterations to which retinal layer?
a. Nerve fiber layer.
b. Inner plexiform layer.
c. Outer nuclear layer.
d. RPE.

2. Which of the following factors has not been reported to be associated with this patient's diagnosis?
a. Age.
b. Sex.
c. Refractive error.
d. Medication.

3. All of the following can be associated with this condition except:
a. Optic nerve hyperemia.
b. Vitreous cells.
c. Bilateral involvement.
d. Serous retinal detachment.

4. Which of the following are typical visual field findings in this diagnosis?
a. No reduction in visual field.
b. Enlarged blind spot; temporal and paracentral scotomas.
c. Generalized depression.
d. Arcuate defects.

5. What is the most likely etiology of the patient's condition?
a. Infectious.
b. Degenerative.
c. Autoimmune.
d. Metastatic.

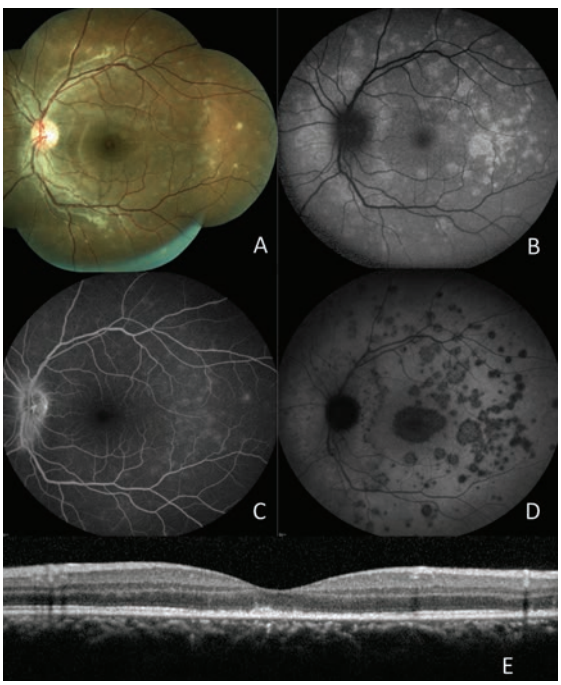


Fig. 10. (A) Fundus photograph; (B) FAF; (C) late FA; (D) late ICGA; (E) OCT.

Case 4 Discussion

This patient was diagnosed with multiple evanescent white dot syndrome (MEWDS). It is typically a unilateral condition, but bilateral cases have been reported.[13] MEWDS most commonly affects young, white, myopic females. Patients have frequently reported symptoms of paracentral and temporal scotomas, blurred vision and shimmering photopsias.[13]

MEWDS is an autoimmune inflammatory response that mainly affects the RPE and photoreceptors. Ocular examination reveals multiple white lesions in the fundus. Optic nerve hyperemia may also be present.[13] Anterior segment is typically normal, but anterior vitreous cells may be present.[13] FA reveals punctate areas of hyperfluorescence, often in a wreathlike pattern and shows hyperfluorescence of the optic nerve in late stages. ICGA, however, reveals early hypofluorescent lesions that remain hypofluorescent in late stages. Lesions seen with ICGA may be more numerous than those seen on examination and FA.[14,15]

The white spots on a fundus examination appear as hyperfluorescent lesions on FAF. The FAF may also reveal more extensive involvement not seen on fundus examination alone. OCT findings reveal alteration to the RPE and photoreceptors, often with disruption in the photoreceptor integrity line.[14,15]

MEWDS is diagnosed based on clinical presentation and diagnostic imaging.[14,15] While the condition is typically self-limiting, observing these patients for typical progression of the disease and confirming the diagnosis by monitoring gradual self-improvement over time is necessary.[14,15]

This condition falls under the group of posterior inflammatory "white dot syndromes," known to masquerade as other ocular inflammatory syndromes.[15] Some are more progressive than others, so an accurate diagnosis is essential for proper management. Cases with an abnormal clinical course may require systemic workup to determine specific infectious or inflammatory etiology in need of local or systemic therapy. Patients with white dot syndromes also possess a predilection for choroidal neovascularization, so long-term follow up is imperative.[15]

CASE 5: We Are All Made of Stars

A 30-year-old Caucasian male presented to the clinic for macular evaluation. He reported longstanding vision loss, which worsened progressively throughout his childhood and teenage years. Leading up to the visit, he had not noticed any recent changes to his vision. His medical history was positive for seasonal allergies, for which he used Claritin (loratadine, Merck). The patient reported no family history of systemic or eye disease.

Best-corrected visual acuities were 20/200 OD and 20/150 OS. He had normal chair skill testing with full confrontation visual fields, normal extraocular motilities and pupils that were equally round and reactive to light. His IOPs measured 15mm Hg OD and 16mm Hg OS. Anterior segment findings were also normal.

The patient's posterior segment findings include fundus photography, FAF and macular OCT findings (*Figure 11*).

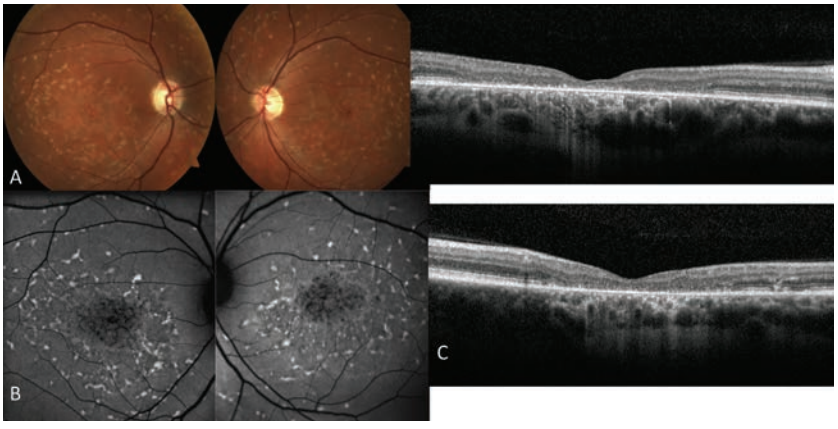


Fig. 11. (A) Fundus photograph; (B) FAF; (C) OCT.

1. Hyper-autofluorescent areas on the FAF correspond to:
a. RPE atrophy.
b. Buildup of lipofuscin.
c. Drusen.
d. Pigment epithelial detachments.

2. Hypo-autofluorescent areas on the FAF correspond to:
a. RPE atrophy.
b. Buildup of lipofuscin.
c. Drusen.
d. Pigment epithelial detachments.

3. OCT imaging reveals:
a. Atrophy of vitreoretinal interface and epiretinal membrane formation.
b. Retinal nerve fiber layer atrophy.
c. Retinal ganglion cell atrophy.
d. Atrophy of the outer retina, including RPE and photoreceptors.

4. The hyper-reflectance of the subfoveal choroid on the OCT is caused by:
a. Overlying nerve fiber layer atrophy.
b. The thickening of the choroidal vessels.
c. Atrophy of the overlying RPE.
d. The upward displacement of the choriocapillaris.

5. Abnormal findings are to be expected for each of the following diagnostic tests except:
a. Visual field testing.
b. Ophthalmodynamometry.
c. ERG.
d. Color vision.

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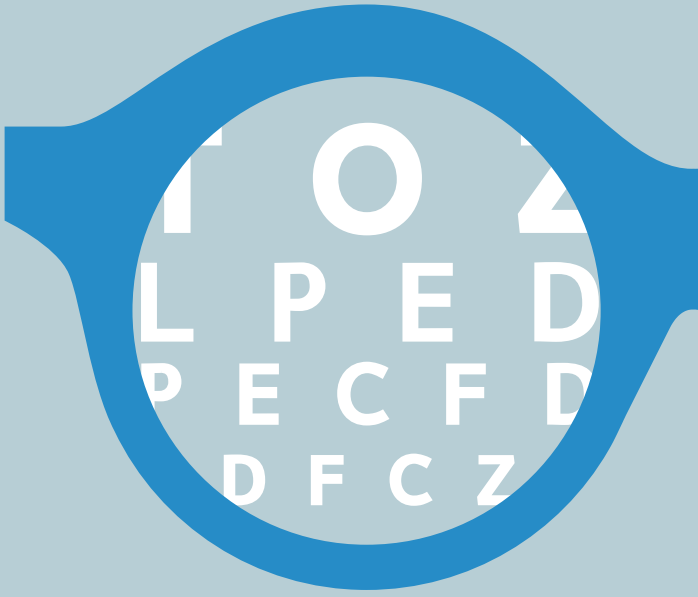

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Case 5 Discussion

The patient's diagnosis is Stargardt disease. The most commonly occurring inherited macular dystrophy, it is inherited in an autosomal recessive fashion.[16] The gene responsible, ABCA4, codes for a protein located on the photoreceptor's outer segment discs. This protein is involved in the transport of all-trans-retinal (atRAL) out of the outer segments so it can be properly metabolized by the RPE. Improper transportation results in accumulation of waste material and lipofuscin. In addition to Stargardt disease, the ABCA4 gene is implicated in many other conditions such as cone-rod dystrophies, retinitis pigmentosa and age-related macular degeneration.[16]

As there are many variations of gene mutation in ABCA4, there are also different phenotypical presentations of Stargardt.[16] A hallmark characteristic is yellow-white pisciform-shaped flecks at the level of the RPE. These lesions may be present in addition to foveal atrophy that is classically described as having a "beaten bronze" appearance, or the flecks may present in isolation. In the past, pisciform lesions in the absence of macular atrophy was termed fundus flavimaculatus; recently, most agree they are different phenoytypes of the same disease.[16]

Patients affected by Stargardt disease typically begin to notice vision loss in their teenage years, falling on the spectrum of mild loss to 20/200 or worse.[16] Initial fundus findings may be subtle, making the condition challenging to diagnose early. Patient symptoms may seem exaggerated when compared with fundus examination in early stages of the disease. FAF and OCT imaging, however, may reveal subtle alterations to the RPE and outer retina even before fundus alterations are visible.[16] For this reason, these tools are quite useful for early diagnosis. Patients may also develop red-green color defects, visual field defects and may have abnormal ERG findings.[16]

OCT in Stargardt disease can reveal various levels of RPE and photoreceptor atrophy. In this case, there was significant outer retinal atrophy, loss of visibility of the photoreceptor integrity line and external limiting membrane (ELM) throughout a large area of the macula. This directly correlates with vision loss in the patient. In certain stages of the disease, reports note a thickening of the ELM on OCT.[17] In addition, flecks may present as subretinal hyper-reflective deposits with various disruption of overlying photoreceptor layers.[18]

There are also various autofluorescent patterns found across the many different clinical manifestations of Stargardt disease. Flecks are hyper-autofluorescent, while areas of RPE atrophy will be hypo-autofluorescent. Bull's-eye autofluorescent patterns can also be found.[19]

Diagnosis of the condition is typically made based on fundus examination, FAF and OCT testing; however, a definitive diagnosis would require genetic testing to confirm the genetic mutation. No current treatment for Stargardt disease exists, but patients should be monitored for development of choroidal neovascular membranes.[20]

Taking steps to strengthen your diagnostic acumen on a regular basis is critical to your practice. This exercise can provide you with the tools you need to provide sound, thorough care to your patients. ■

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Dr. Haynes is a clinical optometric fellow at the Charles Retina Institute.

Answer Key

Case 1: (1) d; (2) a; (3) b; (4) c; (5) c.
Case 2: (1) b; (2) b; (3) c; (4) d; (5) c
Case 3: (1) c; (2) b; (3) c; (4) d; (5) d.
Case 4: (1) d; (2) d; (3) d; (4) b; (5) c.
Case 5: (1) b; (2) a; (3) d; (4) c; (5) b.

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10 Tips and Helpful Hints for Neuro-ophthalmic Disease

Diligent testing and a suspicious mind can go a long way. **By Kelly A. Malloy, OD**

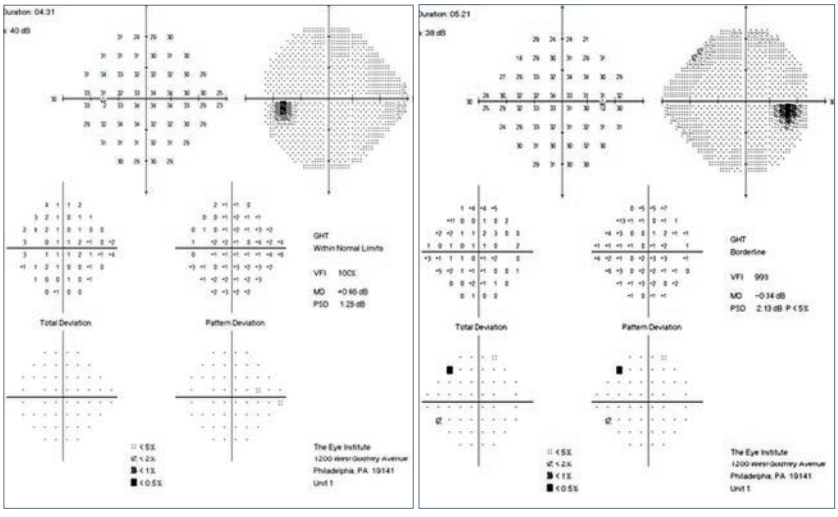
Neuro-ophthalmic disease is often daunting for eye care practitioners, considering it can present with abnormalities in any part of the examination, and with features overlapping with many other ocular and systemic conditions. ODs don't want to unnecessarily refer patients, but they also don't want to miss a potentially serious or life-threatening condition.

Some of the reasons for unnecessary referrals to neuro-ophthalmic disease specialists include uncorrected or inaccurately corrected refractive error, uncorrected presbyopia, dry eye disease (DED), meibomianitis and hypoplastic or anomalous optic discs.

Necessary referrals are often missed when patients are asymptomatic or present with an early or mild form of a condition. Issues can also be overlooked when the provider does not perform a complete assessment or fails to correlate the constellation of signs and symptoms with a neuro-ophthalmic process.

Navigating where patients fall on this imaginary, and often blurred, line between a common presentation and a medical emergency can be challenging. Every presentation is unique, and clinicians must use their history-taking skills and clinical acumen to determine the appropriate level of concern for each patient.

All ODs should be familiar with neuro-ophthalmic presentations, because patients don't know when they need a neurologist or a neuro-ophthalmic disease specialist—they



Case 1. This 51-year-old woman presented with a complaint of daily right-sided head and eye pain, as well as worse vision in the right eye. Her VFs were normal with no RAPD. All other aspects of her afferent and efferent visual function were intact. Despite wearing progressive lenses, her vision was reduced more so at near than at distance in the right eye compared with the left eye. Careful refraction found that she was over-minused by 1.00D in the right eye. With the new prescription trial-framed, she felt her eyes were more relaxed and her vision was comfortable and symmetric.

Table 1. Visual Function Tests

Afferent	Efferent
• Visual acuity • Visual fields • Swinging flashlight test to assess for an RAPD • Color vision testing • Assessment for red desaturation • Assessment for reduced brightness sense	• Pupil measurements in bright and dim illumination • Measurement of palpebral apertures • Ductions and versions • Cover testing in multiple positions of gaze • Exophthalmometry

rely on their trusted eye care provider to tell them when it's necessary.

Here are some helpful hints—based on years of experience—to consider when dealing with a potential neuro-ophthalmic disease case. While not a comprehensive review of all the responsibilities and presentations one may encounter, it includes some pitfalls to avoid.

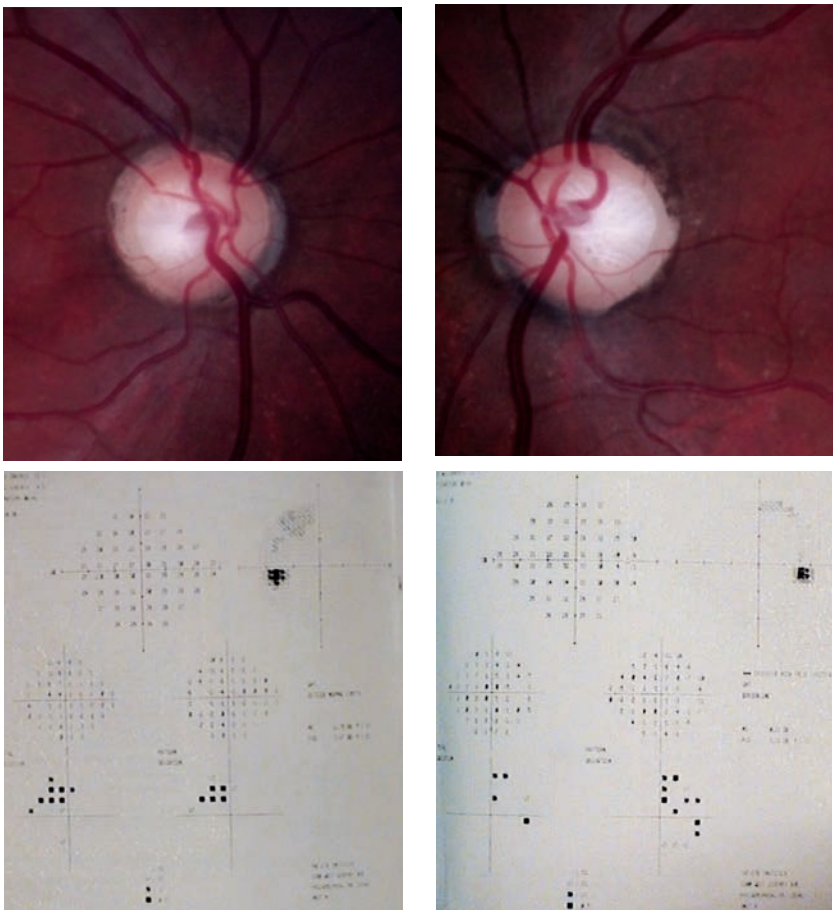
Everyone's a Suspect

Clinicians should always consider that every patient might have a neuro-ophthalmic disease process. While this is obvious for patients who present urgently with symptoms such as diplopia or sudden vision loss, clinicians should also be on the lookout for a neuro-ophthalmic disease process even in patients presenting for routine eye care without any specific complaints. It is important to fully assess the ocular health status in all patients. The combination of afferent and efferent visual function tests will help uncover an abnormality (*Table 1*).[1,2]

Not Always Cause for Alarm

Often, a routine exam can explain symptoms and help avoid unnecessary referrals. Some common, and often unspecific, signs and symptoms can be easily explained without the need for additional referral:

Frontal headaches and eye pain. Patients frequently present with headaches or eye pain that initially seem unexplained. However, careful consideration can minimize the need for neuroimaging or a neuro-ophthalmology consultation. A careful refraction is crucial, especially in younger patients, because seeing 20/20 doesn't necessarily mean the patient is properly corrected. Patients are often referred for eye pain and frontal headaches, and the etiology is uncorrected hyperopia or over-minused myopic patients—symptoms are due to over accommodation. In patients with such complaints, be sure to fully relax the eyes before refining the refraction.



Case 2. This 73-year-old man was being treated for glaucoma. However, his optic discs demonstrated diffuse neuroretinal rim pallor. In addition, his VF loss was only temporal and respected the vertical meridian, which is not consistent with glaucoma and more concerning for a sellar mass. Neuroimaging demonstrated a pituitary macroadenoma.

Basic refractive techniques, including initial maximum plus to maximum visual acuity and binocular balance, are essential, especially in pre- and early presbyopic patients. Always consider the potential for latent hyperopia in patients with unexplained eye pain and frontal headaches. Perform a cycloplegic refraction before considering additional workup or referral (*Case 1*).

Blur or transient vision loss. Patients complaining of nonspecific eye pain and blurry vision sometimes have DED, meibomianitis or both. Patients with these conditions may complain of what sounds like episodes of transient vision loss. Here, a detailed history is key. If the symptoms are related to DED, the patient typically reports that the episodes of vision loss last a few seconds and are relieved with blinking or use of a lubricating drop. A true episode of transient vision loss represents a transient ischemic attack and should be treated as a medical emergency—the patient should go to the hospital immediately for neuroimaging to rule out stroke.[3,4]

Visual field (VF) loss. Patients with significant DED may perform poorly on a formal VF due to ocular irritation and reduced blinking during the test. This often becomes more problematic as the test progresses, and they may do more poorly on the second eye tested. If it is truly a neuro-ophthalmic disease process, they should have a corresponding relative afferent pupillary defect (RAPD) in the eye that performed more poorly on the VF.[5,6] Prior to performing a VF, patients with DED should instill lubricating drops. Also, make sure the patient understands they are allowed to blink normally during the test.

Eyelid edema or ptosis. Heavy eyelids that appear droopy may be a true ptosis or an anterior segment issue. Feel the upper lid margin for fullness suggestive of meibomianitis, and ask the patient if they have a stringy discharge in the medial canthus upon awaking. Even without visually blocked glands on the lid margins, try to manually express the glands. If you are unable to express a clear secretion, consider meibomianitis as a diagnosis. Meibomianitis is more likely to be a bilateral process, whereas some of the most concerning neuro-ophthalmic causes of ptosis such as cranial nerve (CN) III palsy and Horner's syndrome tend to be unilateral. Myasthenia gravis can present with unilateral or bilateral ptosis. However, myasthenic ptosis is fatigable, so remember to test for worsening of the ptosis after two minutes of upgaze and test for improvement of the ptosis after two minutes of ice application. These would both be suggestive of myasthenia gravis.

Top 10 Neuro Tips

Some of the most common issues overlooked are mild papilledema, optic neuropathy, ocular misalignment, Horner's syndrome and nystagmus. Several neuro-ophthalmic processes are medical emergencies and, when suspected, patients should be sent directly to the emergency department for immediate testing and treatment (*Table 2*). These 10 tips can help you get to the bottom of your patient's concerns and manage their care properly:

1. Take a detailed history. Office staff should be prepared to triage phone calls about the need for urgent appointments. To avoid a delayed evaluation of a potentially emergent situation, clinicians must be aware of every call for an urgent or emergent appointment.

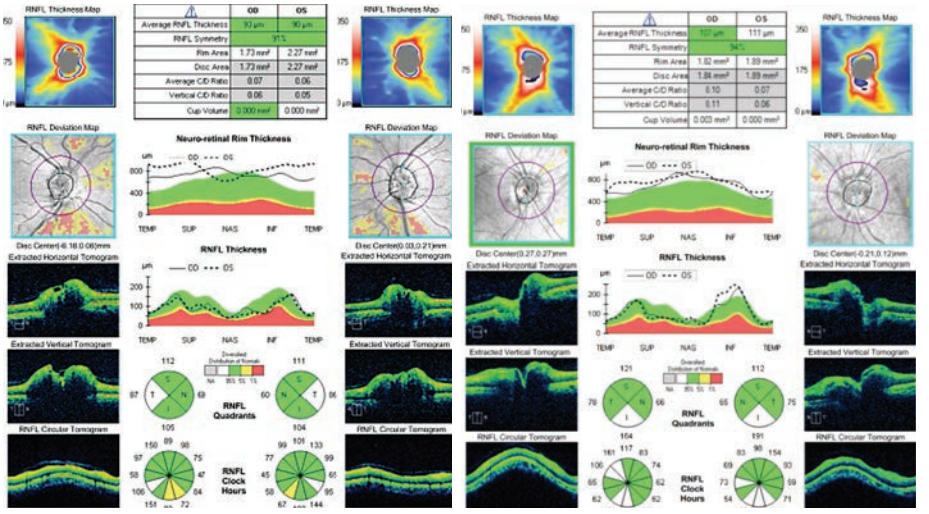
As the first step of any appointment, urgent or not, a detailed history should not be rushed. A few extra minutes in the beginning of the exam can provide a better sense of what is going on and avoids the risk of missing something urgent, emergent or even life-threatening.

Do not limit your questions to the eyes, as patients rarely think it is important to mention non-ocular signs and symptoms; also, do not ignore symptoms already being managed by another specialist. Perform a full review of systems and take a thorough medical, surgical, family and social history. Some important considerations include: alcohol, drug and tobacco use; poor nutrition and changes in appetite or weight; a history of cancer; pregnancy or recent birth (changes in the pituitary gland can occur in these patients); and symptoms of giant cell arteritis in patients over age 50.

For patients with recent hospitalizations, blood tests or imaging studies, staff should obtain the reports, as well as those from the patient's other doctors. Do not assume someone else is handling everything; use these reports to determine if the patient needs further testing, treatment or referral. Even if they do not yet have any visual symptoms, a thorough history may aid in the timely diagnosis of important systemic conditions such as giant cell arteritis, myasthenia gravis, multiple sclerosis or stroke, to name a few.

2. Check blood pressure. Abnormal or asymmetric (between arms) blood pressure, pulse or both could help you identify the etiology of the clinical presentation. You can make a difference in the patient's overall health if you discuss the importance of medication compliance, call and get them back to their primary care physician or cardiologist if overdue, or send them to the emergency department if warranted by extremely abnormal results.

3. Explain any reduced visual acuity. Even a visual acuity of 20/25 could signify an early neuro-ophthalmic disease process. Therefore, clinicians must explain



Cases 3 and 4. Both of these patients demonstrate optic disc elevation as evidenced by a thick neuroretinal rim OU. However, the patient on the right is more concerning for papilledema due to the inferior RNFL thickening, as opposed to the temporal RNFL thickening in the patient on the left. In papilledema, the area of the disc that swells first is the one with the most nerve fibers, which is inferior. Since the thickness of the neuroretinal rim often follows the ISNT rule, it would be unlikely to have only the temporal RNFL thicker than normal in papilledema.

reduced visual acuities by media opacities, macular changes or other processes. The degree of media opacity must match the level of visual acuity—a mild cataract, for example, should not cause significant vision loss. Additionally, symmetric lens changes and worse visual acuity in one eye need further explanation. Do not assume keratoconus is the cause of reduced vision in patients with scissor reflexes or high astigmatism on retinoscopy; if they do not have corneal striae, edema or scarring to account for the vision loss, another cause is at play. If no clinical finding justifies the level of visual acuity, further neuro-ophthalmic workup or referral is warranted.

4. Get a VF—today. If visual acuity is reduced even mildly or if the patient has visual complaints, even in the setting of 20/20 vision, be sure to get a formal VF that day to better assess the afferent visual system. A VF can be instrumental in determining if an afferent problem exists, can localize a problem to the suprasellar cistern or occipital lobe and can be crucial in determining the urgency of the condition. A wait could delay an important diagnosis and treatment.

A disease of the arcuate nerve fibers, glaucoma should manifest with nasal steps and proximal or distal arcuate defects. A glaucoma patient who has greater field loss temporally than nasally needs neuro-imaging to rule out a suprasellar mass (*Case 2*).

VFs can help clinicians anatomically localize any potential disease process. Clinicians should also match the VF to both the optic disc appearance and the degree of RAPD—if they do not match, consider that the VF may be inaccurate and repeat the test. Sometimes it is difficult to ascertain if there is indeed pallor of the neuroretinal rim, especially in patients who are highly myopic or those who are status-post cataract extraction with intraocular lens implants. All tests of afferent visual function are necessary to see if they support a diagnosis of optic neuropathy. Color vision testing can be helpful here. RAPD assessment will also be crucial; if an RAPD is present, it can localize a defect to the portion of the visual pathway shared with the light reflex pathway. An RAPD, which localizes anterior to the

You Are Not Alone

Work together with the patient's other doctors for the good of the patient. If you hear suspicious symptoms or see any abnormal findings, even if not eye-related, discuss them with the patient's primary care physician, neurologist, emergency department doctor or other specialists involved in the patient's care.

Don't be afraid to suggest a certain diagnosis that they have not yet considered, and be sure everyone is clear on who is taking the lead on ordering any necessary workup. Also, if you are referring the patient to a neurologist or neuro-ophthalmic disease specialist, communicate the reason for the referral. Supply them with the details of your examination so that, when they see the patient, they can assess for any interval change.

If you think the situation is urgent, give comanaging physicians the courtesy of determining if they want to see the patient before their next available appointment.

If you think the situation is emergent, consider sending the patient to the emergency department rather than making an outpatient referral. If you are not sure, make a call and ask the specialist's opinion. If you do send the patient to the hospital, call ahead and make them aware that the patient is on their way, and alert them of the concerning symptoms or findings.

Good communication develops strong relationships with other doctors in your community, which is helpful for both your patients and your practice.

lateral geniculate nucleus, should lead to suspicion of a problem with the optic nerve, chiasm or tract.

5. Assess for an RAPD. Always check pupil measurements yourself; do not delegate this task to a technician or student—and certainly do not dilate the patient before looking at the pupils. When assessing for an RAPD, be sure to use the proper endpoint—you are comparing the time to pupillary escape in each eye. An RAPD is present when asymmetry exists in this time to pupillary escape; the eye with the faster escape has the RAPD. Do not confuse the pupillary size to be a measure of an RAPD, as it is a measure of the efferent, not the afferent, visual system.

6. Measure pupils and eyelids. Measuring pupil sizes in both bright and dim illumination is necessary to assess for anisocoria. In addition, you must measure palpebral apertures to look for asymmetry. Do not approximate these measurements. When anisocoria is greater in dim illumination, or a pupil is smaller on the same side as the smaller palpebral aperture, consider Horner's syndrome. If it is acute or associated with pain, emergent workup is warranted to rule out a carotid dissection. Otherwise, perform in-office apraclonidine testing to confirm or rule out the presence of Horner's syndrome. Further referral and workup is warranted with a positive test—reversal of anisocoria within one hour after drop instillation.

If the pupils do not react well to a light stimulus, check the near reflex. A greater reaction to an accommodative target than to a light stimulus, or light-near dissociation, can be instrumental in making some important diagnoses, such as blind eye, tonic pupil, Argyll Robertson pupil, dorsal midbrain syndrome and aberrant regeneration of CN III.

7. Use optical coherence tomography (OCT). Any amount of papilledema is a medical emergency. OCT testing and assessing for a spontaneous venous pulsation can help distinguish between mild papilledema and hypoplastic or anomalous discs or optic disc drusen. In papilledema, the swelling tends to begin inferiorly and superiorly. If an OCT shows retinal nerve fiber layer (RNFL) thickening only inferiorly, consider early papilledema; an OCT with only nasal RNFL thickening in the setting of a small disc is more suggestive of an anomalous disc, which should never show progressive thickening (*Cases 3, 4 and 5*). If any question exists that the presentation is an anomalous disc, repeat the OCT in a month to rule out interval progression. An accurate OCT is crucial for these patients, as a poor image with missing data can make the RNFL measurements falsely thin, masking early thickening in papilledema.

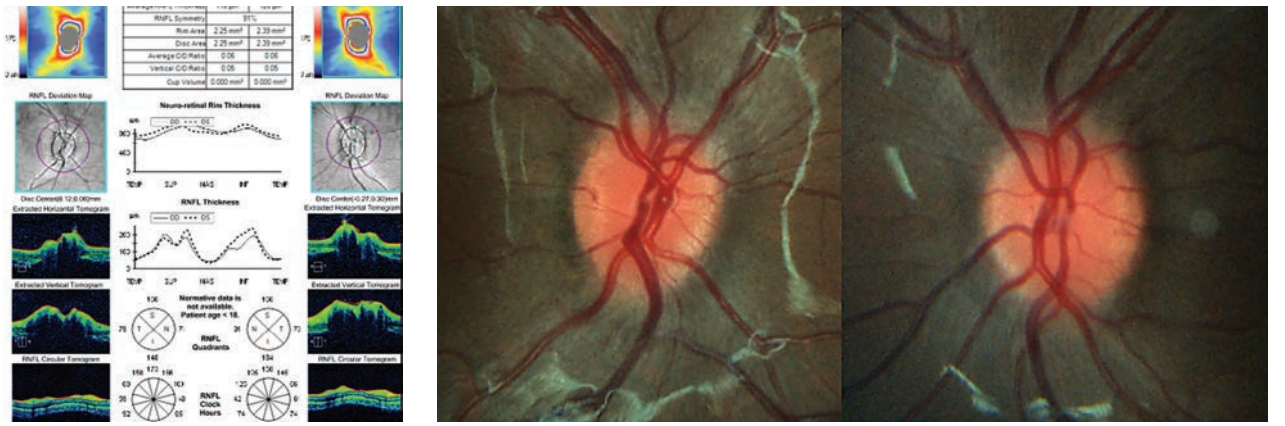
Table 2. Suspected Conditions That Need Immediate Referral to an Emergency Department

Condition	Signs and Symptoms	Required Testing
Giant cell arteritis	Patients over age 50, with possible headache, eye or other pain, scalp tenderness, temporal artery tenderness, diplopia, jaw claudication, fever, loss of appetite, weight loss, joint aches, arteritic anterior ischemic optic neuropathy, CN palsies, homonymous hemianopia or any other new onset neurologic deficit	STAT ESR, CRP, CBC, platelets
Aneurysm	CN III palsy complete or partial, with pain and/or pupil involvement	MRA, CTA and/or angiogram
Papilledema	Indistinct disc margins, obscuration of vessels at disc margin, peripapillary wrinkles (Paton's lines), axoplasmic stasis – with or without other symptoms	Brain MRI with contrast and MRV to R/O mass or venous sinus thrombosis
Pituitary apoplexy	Bitemporal visual field loss, junctional scotoma, incongruous homonymous hemianopia, headache, nausea, vomiting, fever	Brain CT/MRI
Carotid artery dissection	Painful Horner's syndrome: ptosis and miosis	MRA, CTA, angiogram
Acute stroke	Transient vision loss, branch retinal artery occlusion, central retinal artery occlusion, homonymous hemianopia, facial palsy, sudden weakness or numbness on one side of body, slurred speech, skew deviation, INO, gaze palsy	CT then MRI of brain, carotid, and hypercoagulable evaluation, thorough cardiac evaluation

8. Ensure appropriate workup and treatment. When comanaging papilledema patients, make sure they have had a thorough workup to determine the etiology of the papilledema. This includes magnetic resonance imaging (MRI) of the brain with and without contrast and magnetic resonance venography (MRV) to rule out venous sinus thrombosis. After these tests, patients without contraindication should have a lumbar puncture to measure the opening pressure and analyze the cerebrospinal fluid contents. If ultimately diagnosed with idiopathic intracranial hypertension, patients need to be educated that it could still lead to severe vision loss and they must be watched carefully. If vision loss is occurring despite weight loss attempts and Diamox (acetazolamide, Duramed) use, the patient may need surgical consultation for possible optic nerve sheath fenestration, ventriculoperitoneal shunt placement or both.

9. Perform cover testing in all gazes. Always accurately characterize patients' symptoms of blur and diplopia, as patients often confuse these two symptoms. Ask enough questions to ascertain if the patient is experiencing true diplopia. If the diplopia resolves by covering either eye, it is true binocular diplopia, which is more concerning. Perform cover testing not just in primary gaze but in all positions of gaze, to determine if the results are comitant. All too often, clinicians only do cover testing in primary gaze, even though it is essential to assess cover testing in multiple positions of gaze to determine the etiology of the ocular misalignment. Clinicians must be familiar with the patterns of CN III, IV and VI palsies to be able to identify them on the cover testing results.

Other concerning manifestations of diplopia exist, however, and ODs should remember more obscure



Case 5. This five-year-old asymptomatic child has elevated optic discs. Although age-matched normals do not exist to help with OCT interpretation, clinicians can still appreciate that the average RNFL thickness, as well as the inferior and superior RNFL thicknesses, are greater than normal in this child. That, in combination with the lack of a spontaneous venous pulsation in either eye, is concerning for mild papilledema. Even without the presence of Paton's lines or definite obscuration of the retinal vessels coursing over the optic disc margin, there is still concern for mild papilledema. Immediate neuroimaging revealed a malignant brain tumor.

causes such as a skew deviation, which could also warrant urgent or emergent evaluation.

10. Test for torsion. As a general rule, a skew deviation or other brainstem motility finding is concerning for multiple sclerosis or other demyelinating process in a patient under age 50 and is suggestive of stroke in a patient over age 50. Skew deviation is often overlooked as a potential cause of diplopia and other non-specific complaints of visual disturbance. It is a vertical misalignment of the eyes and should be considered a differential diagnosis in any complaint of vertical diplopia or measurement of vertical ocular misalignment. One of the best ways to assess for an acute skew deviation is to look for the presence of torsion. Although not always the case, we typically expect to find incyclotorsion of the higher eye and excyclotorsion of the lower eye. This contrasts with a CN IV palsy, in which we would see a small amount of excyclotorsion of the higher eye.

When a patient complains of vertical diplopia or they have vertical ocular misalignment, clinicians should consider performing the Park's three-step test and torsion testing. Although most tend to think of non-comitant deviations as more concerning, that's not always true.

Skew deviations can have a vertical misalignment that is fairly comitant, and the magnitude does not have to be large. It is common to see a skew deviation in combination with internuclear ophthalmoplegia or nystagmus, so look carefully for these in any patient with a vertical misalignment. Any new onset nystagmus needs further evaluation.

If you consider that every patient could have a neuro-ophthalmic disease process, you are less likely to overlook something serious. If you do not consider such a diagnosis, you will never make such a diagnosis. To keep neuro diagnoses on your list of differentials, make your history and exam more thorough. Look at all of the patient's test results in combination to see if they point to a problem with either the afferent or the efferent visual system. If you find a problem, or if you cannot explain your patient's symptoms, additional neuro-ophthalmic workup and referral is imperative. ■

Dr. Malloy is the director of the Neuro-ophthalmic Disease Service at Pennsylvania College of Optometry, Salus University.

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Electrodiagnostics in Today's Practice

More ODs are turning their attention to advanced testing. Here's what you need to know about this technology. **By Hua Bi, OD, PhD**

In the clinical setting, visual electrodiagnostic testing objectively detects characteristic visual function changes of a given disease—leading to timely diagnosis. It can also help clinicians assess disease severity, evaluate therapeutic effects and adjust the treatment plan accordingly. This primer helps you understand the technology behind and clinical applications of electrodiagnostic tests and what they can do to boost your patient care.

The Fundamentals

Visual electrodiagnostic testing encompasses the electroretinogram (ERG), visual evoked potential (VEP) and electro-oculogram (EOG) tests, which allow localization of functional deficits of the retina, optic nerve and post-chiasmatic components along the primary visual pathway. Specifically, full-field ERG (ffERG) represents a mass response of the entire retina for the rod and cone systems; multifocal ERG (mfERG) provides topographic mapping of the central retina function for the cone system; and pattern ERG (pERG) and ffERG photopic negative response evaluate ganglion cell function. VEP assesses functional integrity of the primary visual pathway at the cortical level, while EOG test results reflect retinal pigment epithelium (RPE) function.

A quick review of what these tests do and how they are performed can help you better understand their clinical utility:

ffERG. This is a mass retinal response elicited by stimulating dilated eyes with flashes of light from a Ganzfeld stimulator, routinely recorded with corneal electrodes. Clinicians can evaluate the cone and rod systems respectively and delineate photoreceptor and bipolar cell contributions. Patients are tested under dark-adaptation using dim and bright single flash stimuli for scotopic responses (dark-adapted 0.01, 3.0 and 10 responses) and under light-adaptation using bright single flash and 30Hz flicker stimuli for photopic responses (light-adapted 3.0 and 30Hz flicker responses).

The responses are evaluated based on the morphology as well as the measurable parameters of waveform components. For example, the a-wave component of the ffERG dark-adapted 3.0 response is the first negative deflection, followed by the

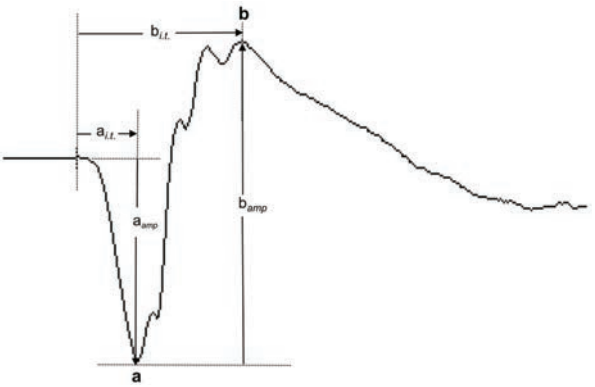
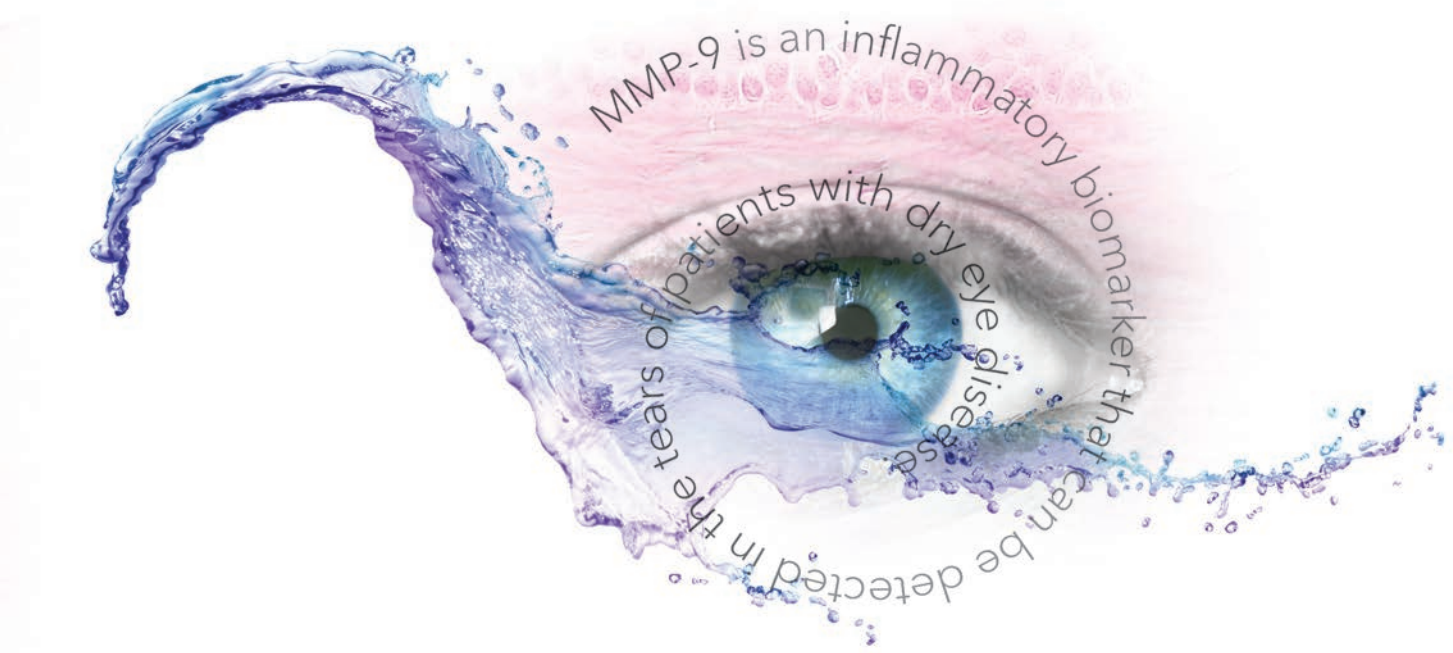


Fig. 1. This normal ffERG dark-adapted 3.0 response shows the a-wave and b-wave components and the measurement of their response amplitudes (a_{amp} and b_{amp}) and implicit times ($a_{i.t.}$ and $b_{i.t.}$).



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positive b-wave component (*Figure 1*). Both amplitude and implicit time are the parameters used to measure the ERG responses. The amplitude of the a-wave is measured from the baseline to the trough of the a-wave, whereas the b-wave is measured from the trough of the a-wave to the peak of the b-wave. Implicit time refers to the time needed for the response to reach maximum amplitude from stimulus onset. Photoreceptor function is reflected by the a-wave, and the b-wave reflects bipolar cell and Müller cell functions.[1] The oscillatory potentials (OPs) consist of low-amplitude and oscillating (high frequency) wavelets superimposed on the ascending phase of the b-wave. OPs reflect inner retinal currents involving neurons such as the amacrine cells.[2]

The photopic negative response (PhNR) is a slow negative component after the b-wave of the cone-driven ffERG, which has been shown to originate primarily from ganglion cell activities.[3]

mfERG. This test provides topographic mapping of the central retina function for the cone system with a diameter of 40° to 50°.[4,5] It simultaneously acquires ERG signals from a large number of discrete retinal locations, which allows detection of focal changes in retinal function. The signals are recorded with a corneal electrode on a dilated eye. Commonly used stimuli are 103 hexagonal elements, with the luminance of each hexagon independently modulated according to a pseudorandom m-sequence.

mfERG trace waveforms represent local responses extracted by correlating the recorded continuous ERG signal with the stimulus sequence. The first-order kernel trace reflects the retinal response to the flash. Each trace consists of an initial negative response (N1), a positive response (P1) and a second negative response (N2). The mfERG waveforms mainly reflect photoreceptor and bipolar cell activities. Amplitude and implicit time of the P1 component are most commonly used for analysis. Overall signal strength per unit area of retina is displayed on the three-dimensional plot (*Figure 2*).

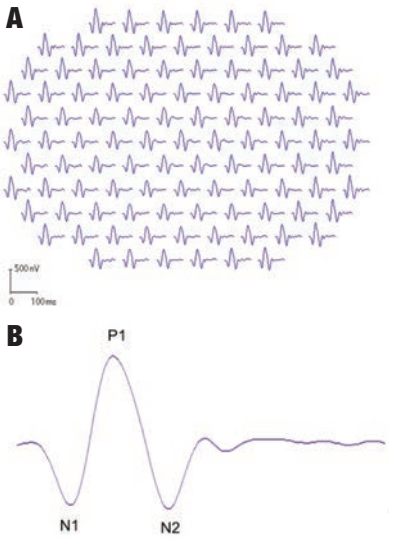
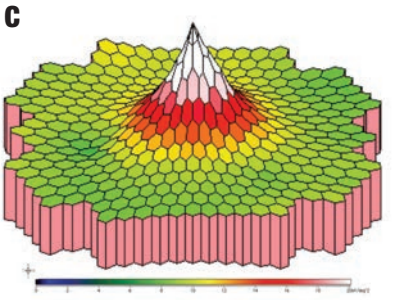


Fig. 2. These first-order mfERG responses (field view) show (A) a normal trace array with 103 elements (left eye), (B) the P1 component of an individual trace and (C) the three-dimensional P1 response density plot.



pERG. This is used primarily for functional evaluation of ganglion cells in the central retina.[6] It originates from the inner retinal layers, enabling an assessment of ganglion cell function. A DTL corneal electrode (Diagnosys) is used for the recording of an undilated eye's response to checkerboard pattern stimuli with spatial and temporal contrast modulation.

Checkerboard reversal patterns with low reversal rates (e.g., four reversals per second) elicit transient responses characterized by a negative-going (N35) component, a positive-going (P50) component and a subsequent negative-going (N95) component (*Figure 3*). The P50 component is partially driven by the ganglion cells, but also has origins distal to the ganglion cells. The N95 component reflects ganglion cell activities. Steady-state pERG responses are elicited by checkerboard patterns of high reversal rates (e.g., 16 reversals per second).

VEP. This is an electrophysiological signal recorded at the occipital

Anatomy Review: The Primary Visual Pathway

Within the retina, the photoreceptors transduce light into neural signals, and the visual information is transmitted to bipolar cells and then to ganglion cells.[1] An additional cell layer, the RPE underlying the retina is essential in supporting photoreceptor function. The duplex retina is uniquely structured with the rod and cone systems, respectively. While the rod system specializes under scotopic or dim light conditions with high sensitivity but low temporal resolution, the cone system specializes for photopic or bright light vision, color detection and fine spatial resolution. Subsequently, retinal ganglion cell axons become myelinated upon exiting through the lamina cribrosa region, and the majority project to the lateral geniculate nucleus (LGN). The axons of LGN neurons then travel along the optic radiations and carry the information to the visual cortex.

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lobe in response to checkerboard pattern (for pattern VEP) or flash visual stimulation (for flash VEP) presented in the central field of an undilated patient. It evaluates the integrity of the primary visual pathway serving the central visual field.[7] The active electrode is placed on the occipital scalp over the visual cortex. The midline occipital electrode can be used to assess prechiasmal function, whereas at least two additional lateral electrodes are recommended for assessing chiasmal and postchiasmal function.

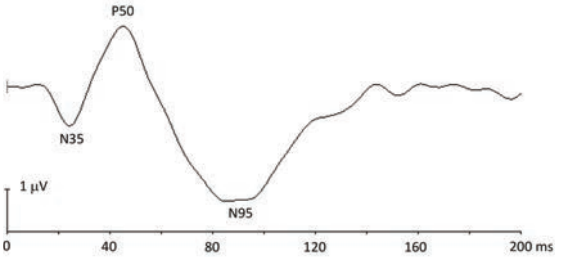

Fig. 3. This pERG response shows the N35, P50 and N95 components.

Table 1. Clinical Applications of Electrodiagnostic Testing in Retinal/Retinochoroidal and Optic Nerve Diseases

	Examples
Retinal/retinochoroidal diseases	
Hereditary	• Retinitis pigmentosa • Cone-rod dystrophy • Progressive cone dystrophy • Leber's congenital amaurosis • Congenital stationary night blindness • Stargardt disease • Best disease • Central areolar choroidal dystrophy • Pattern dystrophy • Dominant cystoid macular dystrophy • Doyne honeycomb retinal dystrophy • North Carolina macular dystrophy • Sorsby's fundus dystrophy • X-linked juvenile retinoschisis
Inflammatory and immune-related	• White dot syndromes - Birdshot chorioretinopathy - Acute posterior multifocal placoid pigment epitheliopathy - Diffuse unilateral subacute neuroretinitis - Multiple evanescent white dot syndrome - Multifocal choroiditis with panuveitis - Serpiginous choroiditis • Acute zonal occult outer retinopathy • Paraneoplastic retinopathies - Cancer-associated retinopathy - Melanoma-associated retinopathy
Toxic	• Systemic medications - Hydroxychloroquine • Intraocular foreign body
Nutritional deficiency	• Vitamin A deficiency
Vascular	• Diabetic retinopathy • Retinal vascular occlusions
Optic neuropathies	• Hereditary optic neuropathies - Autosomal dominant optic atrophy - Leber's hereditary optic neuropathy • Maldevelopment - Optic nerve hypoplasia • Primary open-angle glaucoma

Pattern-reversal VEP (pVEP) uses black and white checks reversed at a rate of two per second. Pattern onset/offset VEP uses alternating checkerboard (200ms) and diffuse gray (400ms) background as stimuli. pVEP is most commonly used due to its smaller variability across typical subjects compared with other types of VEP. The waveform components of pVEP include an initial negative peak (N75), a large positive peak (P100) and a second negative peak (N135) (*Figure 4*).

EOG. This evaluates RPE function under dark and light phases.[8] EOG uses defined eye movements to monitor the standing potential of the eye. Responses decrease with dark adaptation, reaching a minimal value at around 10 to 15 minutes. Responses then increase with light adaptation, reaching a maximal value at around seven to 12 minutes after light onset. Reduced Arden ratio—the light peak/dark trough ratio—indicates RPE dysfunction, given that the light rise process is regulated by Bestrophin-1 (encoded by BEST1 gene), a protein predominantly expressed in RPE cells.[9]

Clinical Applications

Visual electrodiagnostic testing can play a pivotal role in the management of many retinal/retinochoroidal and optic nerve diseases caused by maldevelopment, hereditary, toxic, metabolic, retinal vascular, degenerative, inflammatory and immune-related etiologies (*Table 1*).

Hereditary retinal diseases. These are often genetically heterogeneous, and the majority are caused by gene mutations that primarily affect outer retinal function. Thus, ffERG and mfERG abnormalities are among the main criteria for the diagnosis of many hereditary retinal diseases and for refining functional phenotypes.

Hereditary retinal diseases, such

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as retinitis pigmentosa and cone-rod dystrophy, could cause generalized deficits across the retina; those such as Stargardt disease could predominantly affect the macular area (*Figure 5*).[10,11] Particularly, patients with typical retinitis pigmentosa present with loss of rods followed by loss of cones, while patients with typical cone-rod dystrophy show loss of cones first followed by loss of rods. Therefore, assessment of both the rod and cone systems and topographic mapping of local dysfunction for the central retina are crucial for disease diagnosis.

ERG testing is especially valuable in determining the functional status of the retina in those with less prominent funduscopic abnormalities (*Figure 6*). Furthermore, careful monitoring is essential to determine whether the disease is stationary or progressive for both diagnostic and prognostic purposes.

Autoimmune retinopathy (AIR). This group of inflammatory-mediated retinopathies is characterized by vision loss, photoreceptor dysfunction and the presence of circulating autoimmune antiretinal antibodies. The spectrum of these retinopathies includes paraneoplastic AIR such as cancer-associated retinopathy (CAR) and melanoma-associated retinopathy (MAR), and retinopathies in the absence of malignancy—presumed non-paraneoplastic autoimmune retinopathies that have similar clinical and immunological features.

ERG is sensitive in detecting the retinal abnormalities, often showing significantly reduced responses early in the disease course, even when funduscopic appearance is normal.[12] Western blot and immunohistochemistry assays are also used to detect antiretinal antibodies in the diagnosis of AIR.[12]

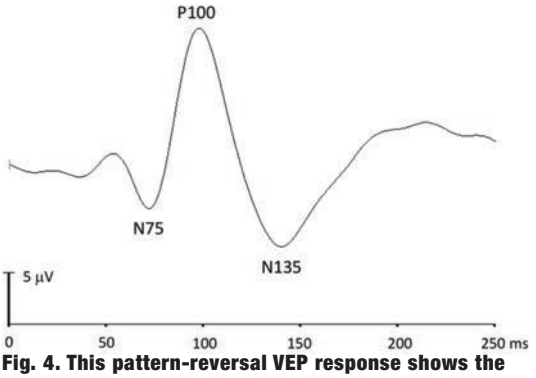

Fig. 4. This pattern-reversal VEP response shows the N75, P100 and N135 components.

CAR is commonly associated with lung cancer, but has also been reported in patients with breast, gynecologic, colon and other cancers.[13,14] In CAR, studies suggest that autoantibodies specific to tumor antigens cross-react with certain proteins (e.g., recoverin and α-enolase) existing in retinal cells and induce apoptotic cell death.[15,16]

In the early stages, severe electrophysiological response attenuation with rapid progression despite relatively less affected funduscopic manifestation is contributing evidence to the AIR diagnosis. Carcinoma always needs to be ruled out with careful medical workup. Considering retinal function abnormalities may precede the diagnosis of systematic cancer, ffERG and mfERG tests are pivotal in managing these cases.

MAR is a paraneoplastic retinopathy in patients with a positive history of malignant melanoma. It is more common in men than in women and commonly presents after the melanoma is diagnosed, often at the advanced stages of the disease.[17,18] ERG is quite sensitive in detecting retinal abnormalities associated with MAR.[19] Classic MAR patients show ffERG abnormality with reduced b-wave amplitude due to circulating antibodies reacting with bipolar cells.[20,21]

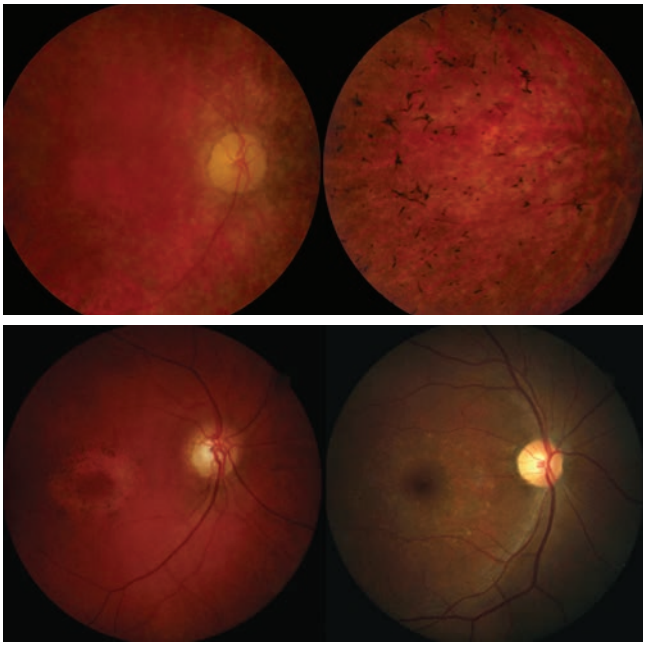

Fig. 5. The top panel shows fundus photos of retinitis pigmentosa. The bottom panel shows fundus images of cone-rod dystrophy (left) and Stargardt disease (right).

Plaquenil toxicity. Many systemic medications cause toxic retinopathy, one of which is Plaquenil (hydroxychloroquine, Sanofi-Aventis).[22-24] Widely used for treating conditions such as systemic lupus erythematosus and rheumatoid arthritis, Plaquenil is toxic to the retina and can cause photoreceptor losses and RPE disruptions.[25]

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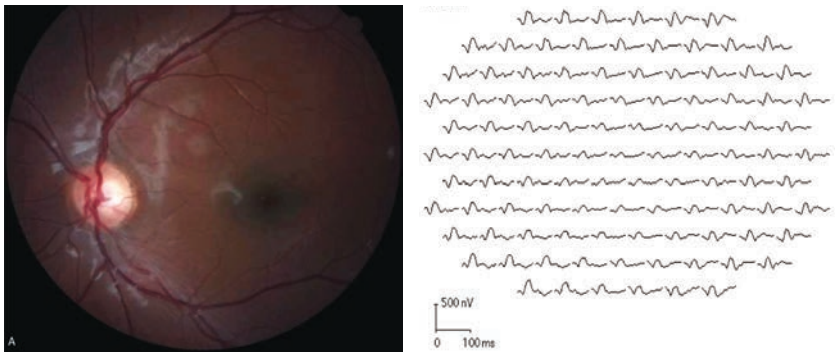

Fig. 6. Marked diffuse mfERG response attenuation was detected in both eyes of a 13-year-old Stargardt disease patient. This fundus image and corresponding mfERG show the results from the left eye.

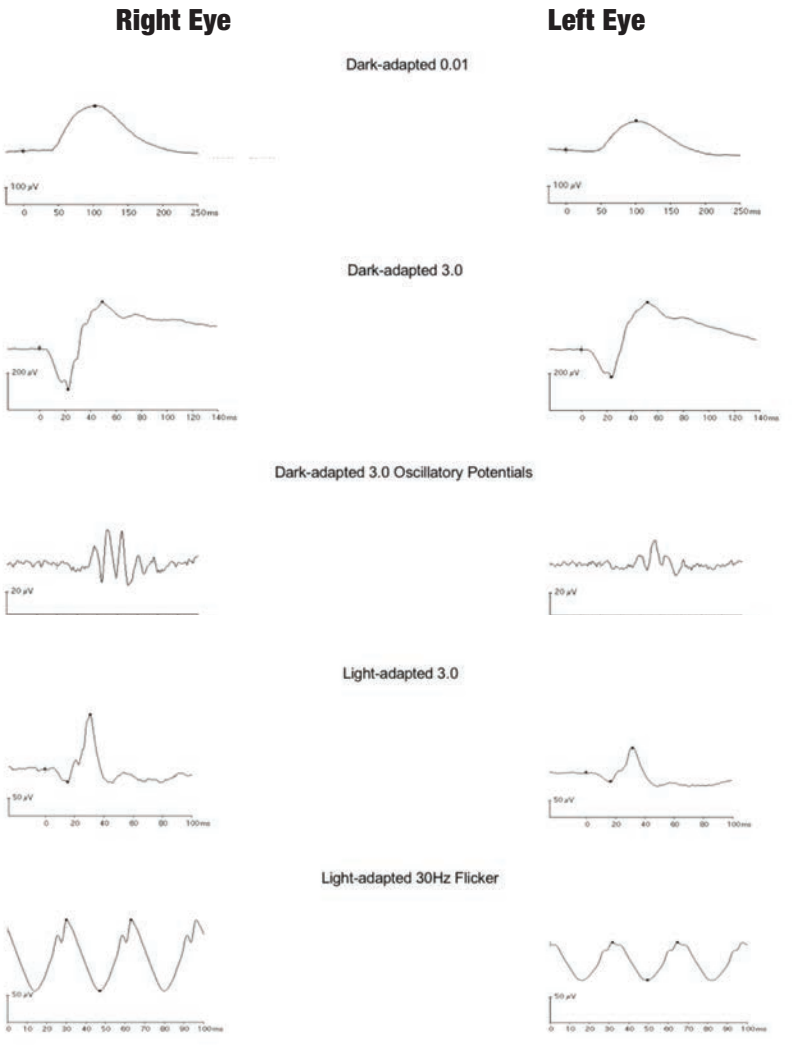

Fig. 7. Interocular comparison of ffERG responses of a patient with unilateral retinal siderosis shows marked amplitude reduction for both the scotopic and photopic responses in the left eye with an iron-containing intraocular foreign body.

Ophthalmology recommendations on screening for hydroxychloroquine toxicity, mfERG can provide objective corroboration for visual field testing.[26] Several remarkable features of mfERG testing are worth noting for their role in Plaquenil toxicity screening. First, mfERG detects dysfunction of the outer retina where the toxic damage occurs. Second, its sensitivity can be further enhanced by using ring ratio analysis to detect a localized parafoveal pattern of damage. Finally, it is excellent in detecting preexisting maculopathy to establish a baseline and identify those at higher risk for toxicity.

Toxic retinopathy resulting from intraocular foreign body. Retinal photoreceptors and RPE cells are especially susceptible to the released iron from metallic intraocular foreign body, as accumulation of intracellular iron in RPE and Müller cells results in retinal siderosis.[27] ERG provides quantitative and objective assessment of retinal dysfunction of the rod and cone systems to guide surgical management (*Figure 7*).

Optic nerve hypoplasia (ONH). This congenital anomaly is characterized by an underdeveloped optic nerve in one or both eyes. ONH is among the three leading causes of blindness in children in the United States, and its prevalence has increased over the past several decades.[28,29]

The pathogenesis of ONH is not fully understood, although young maternal age, primigravida and preterm birth are among the suggested risk factors.[30] pERG is useful to assess functional deficits associated with ONH, as a reduced N95 component in pERG has been found in patients with ONH (*Figure 8*).[31]

Primary open-angle glaucoma (POAG). Electrophysiological tests such as pERG could objectively assess dysfunction of ganglion cells.

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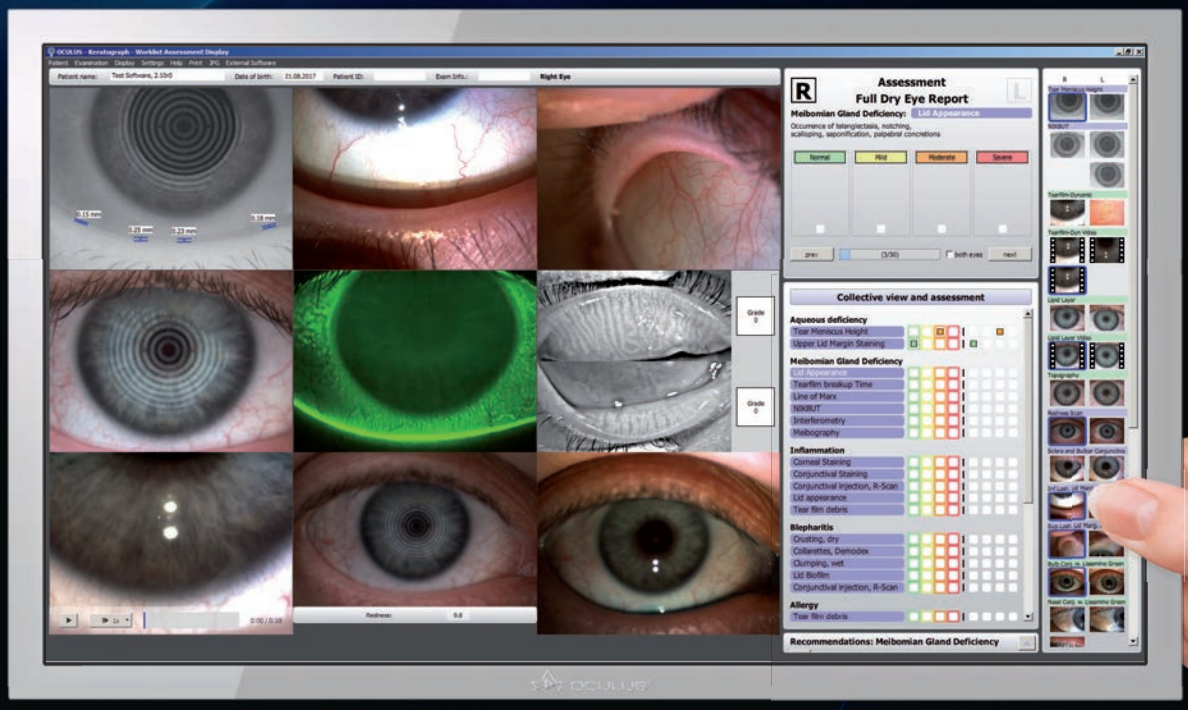

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Because of this distinctive feature, these tests may one day have a significant impact on the clinical management of glaucoma patients. Particularly, a recent study found pERG can provide early detection of ganglion cell dysfunction when visual field defects are minimal in early glaucoma.[32] Moreover, PhNR is capable of detecting a reversible aspect of glaucomatous dysfunction after IOP reduction.[33]

Retinal and optic nerve deficits in central nervous system disorders: multiple sclerosis (MS). In patients with MS or MS-associated optic neuritis, optic nerve axonal abnormalities are commonly reflected by prolongation of the pVEP P100 associated with demyelination, and also by attenuation of the P100 in a subset of MS patients.[34] In recent years, studies have reported abnormalities of non-myelinated axons and retinal neurons.[35] Furthermore, neuronal abnormalities not only involve ganglion cells, but can also predominantly occur in the inner and outer nuclear layers in a subset of patients.[36] Therefore, pVEP and mfERG can be valuable in detecting axonal and neuronal impairments.

The pathological disease processes affecting the primary visual pathway are reflected in their electrophysiological characteristics. Electrodiagnostics are effective in objectively evaluating visual function along hierarchical stages of the primary visual pathway, and the battery of tests must be interpreted in its totality. In conjunction with other clinical findings, these tests enable clinicians to diagnose many ocular diseases, monitor for disease progression and serve as a functional marker when assessing therapeutic efficacy. ■

Dr. Bi is an associate professor at Nova Southeastern University College of Optometry.

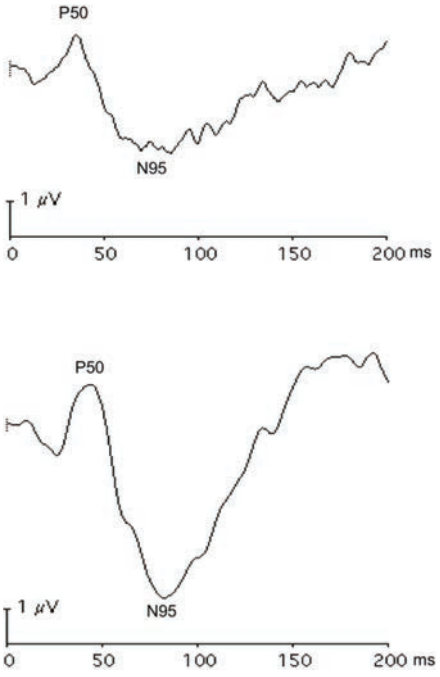

Fig. 8. These pattern ERG responses of the right (A) and left (B) eyes in a patient with bilateral asymmetric optic nerve hypoplasia show lower amplitude of the N95 component in the right eye with more severe optic nerve deficit.

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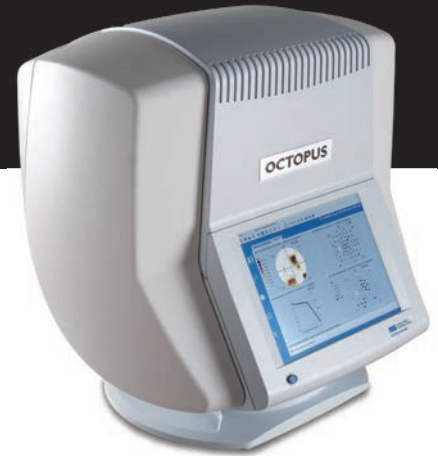

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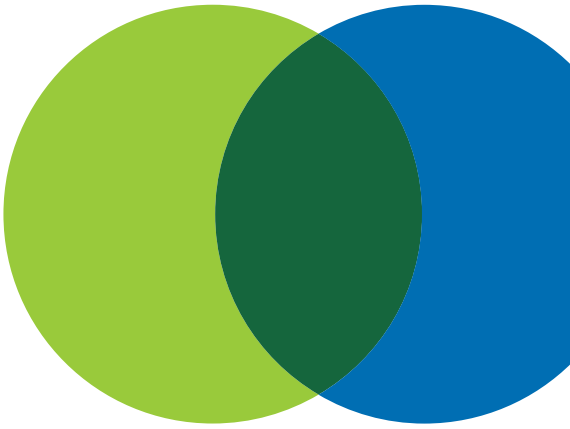

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THE WHEN AND WHY OF ORDERING BLOOD WORK

This is within most ODs' scope of practice—don't be afraid to use it.

By Blair Lonsberry, MS, OD, MEd

As optometry continues to expand its therapeutic privileges, the OD's role in the diagnosis, management and prevention of systemic health conditions is more important than ever—and an integral part in the diagnosis is lab testing. Regardless of whether the optometrist is ordering lab tests independently or is comanaging with a primary care provider (PCP), a working knowledge of what tests to order and what the results mean is crucial.

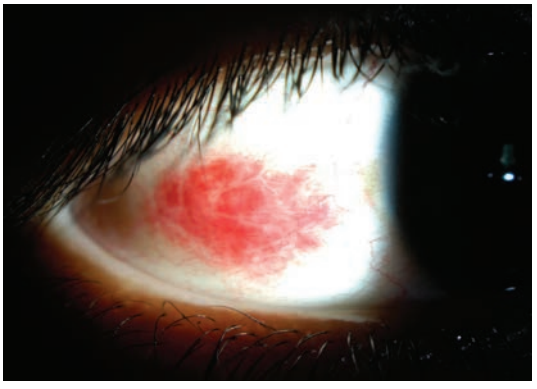

Fig. 1. Subconjunctival hemorrhage may be an indication of underlying anemia, which would require a CBC to hone in on the diagnosis.

Testing Basics

Most lab tests require a blood draw. Some require a urine sample, such as the nucleic acid amplification test (NAAT) for chlamydia and gonorrhea, or swab testing (oral and anal chlamydia/gonorrhea cannot be diagnosed from urine sample and require oral/anal swabs).[1] All of these require the patient get the testing done at an accredited facility. Some have specific pretest requirements such as fasting (e.g., fasting plasma glucose, lipid panel) or can be influenced by pretest conditions (e.g., patient hydration levels and excessive meat intake can alter kidney function tests), and it is important to educate the patient prior to sending them to the lab.[2]

Laboratory reports typically include when the sample was collected, when it was analyzed and the name of the person who requested the testing. The results will include a set of reference ranges for each test that are based on "normal" values for the average population and may be further classified by other variables such as race, age and gender. Any result that falls outside of the reference

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Goal Statement: Many ocular conditions have a systemic etiology diagnosed with lab testing. Whether ordering lab tests independently or comanaging with a primary care provider, optometrists must have a working knowledge of what tests to order and what the results mean. This article discusses the common lab tests necessary to diagnose systemic conditions such as diabetes, liver and kidney diseases, systemic inflammatory diseases, autoimmune disorders and infectious diseases.

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range is typically highlighted in some fashion (*Table 1*).[3,4]

A complete blood count with differential (CBC with diff) is probably one of the most common blood tests ordered and can often help to explain patient symptoms such as weakness, fatigue or bruising.[3,5] A CBC provides important information about the kinds and numbers of cells in the blood, especially red and white blood cells and platelets (*Table 2*).

A CBC with diff may be ordered for a variety of conditions that have associated ocular manifestations such as anemia, leukemia, infections or recurrent inflammation. Patients who have recurrent subconjunctival hemorrhages may have underlying anemia and would require a CBC to help rule it out (*Figure 1*).[4] A CBC may be ordered in a patient who presents with a bilateral uveitis to rule out the possibility of a systemic infection (e.g., elevated white blood count may be an indication of a bacterial infection). A CBC should also be ordered for patients who have unexplained retinal hemorrhages, cotton wool spots or both. Patients prescribed certain medications, such as oral steroids, blood thinners and diuretics, should have baseline testing and periodic monitoring.[3,4]

Lipid testing is often ordered as part of "routine" lab testing but is particularly important in patients with diabetes and for assessing risk for cardiovascular disease.[3,6] The American Association of Clinical Endocrinologists published 2017 guidelines for the management of dyslipidemia and prevention of cardiovascular disease, and have included a new "extreme risk" group.[6]

Patients who have arcus at a younger age (arcus juvenilis) or Hollenhorst plaques should have their cholesterol levels evaluated.[4] There are several specific components of cholesterol evaluation (*Table 3*):[3]

Total cholesterol. This measures all of the cholesterol in all the lipoprotein particles.

High-density lipoprotein cholesterol (HDL-C). This measures the cholesterol in HDL particles; often called "good cholesterol" because it removes excess cholesterol and carries it to the liver for removal.

Low-density lipoprotein cholesterol (LDL-C). This calculates the cholesterol in LDL particles; often called "bad cholesterol" because it deposits excess cholesterol in walls of blood vessels, which can contribute to atherosclerosis. Usually, the amount of LDL-C is calculated using the results of total cholesterol, HDL-C and triglycerides.

Triglycerides. This test measures all the triglycerides in all the lipoprotein particles; most is in the very low-density lipoproteins.[3]

Table 1. Kidney Function Test Results

Component	Your Value	Standard Range	Flag
Creatinine	1.35mg/dL	.06mg/dL to 1.3mg/dL	***H***
GFR Afr Amer(CKD-EPI)	70mg/dL	≥60mg/dL	
GFR nonAfr Amer(CKD-EPI)	61mg/dL	≥60mg/dL	

This common kidney function testing report includes the patient's creatinine and glomerular filtration rate (GFR). The creatinine levels are slightly elevated, highlighted by the "H" in the furthest column.

Case Example:
A 38-year-old white female presented for an updated spectacle correction. Her medical history was negative, she was taking birth control and a multivitamin, she denied any history of smoking and is an occasional alcohol consumer. Visual acuities (VAs) were correctable to 20/20 in both eyes, entrance skills were unremarkable, slit lamp was unremarkable and intraocular pressures (IOPs) were 16mm Hg OD and OS.

Upon dilated fundus exam, a cotton wool spot was noted in both eyes (*Figure 2*). Blood pressure measured 125/82mm Hg. She was referred to her PCP, who found her blood glucose and glycosylated hemoglobin test (A1c) values were normal and repeat blood pressure evaluation was within the normal range. A CBC with diff ultimately indicated low levels of hemoglobin and hematocrit, and she was diagnosed with anemia.

Here is a look at other common lab tests ODs can order, and how to interpret the results.

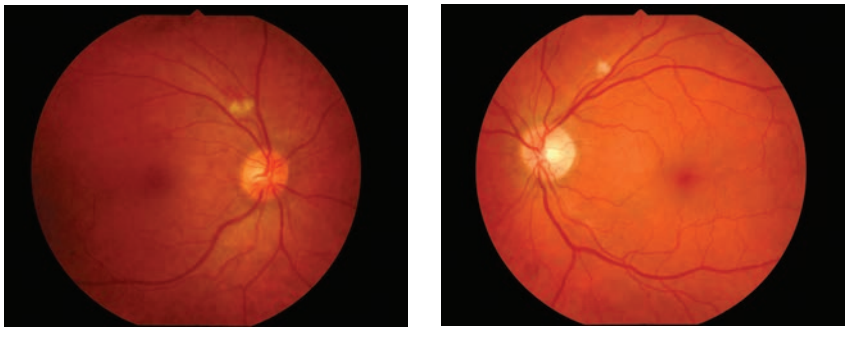

Fig. 2. This cotton wool spot superior to the optic nerve in the right and left eye of a 38-year-old asymptomatic female patient led us to order testing, which revealed a diagnosis of anemia.

Table 2. Representative CBC with Differential Report with Associated Standard Ranges

Component	Your Value	Standard Range	Flag
WCB	4.4x10(9)/L	4.0x10(9)/L to 10.5x10(9)/L	
RBC	5.12x10(12)/L	4.00x10(12)/L to 5.50x10(12)/L	
Hemoglobin	15.4gm/dL	13.0gm/dL to 17.0gm/dL	
Hematocrit	46.7%	37.0% to 50.0%	
MCV	91.2fL	82.0fL to 100.0fL	
MCH	30.1pg	28.0pg to 35.0pg	
MCHC	33.0	31.0 to 36.5	
RDW, CV	13.2%	11.0% to 14.5%	
RDW, SD	44.5fL	36.0fL to 50.0fL	
Platelet count	223x10(9)/L	140x10(9)/L to 375x10(9)/L	
MPV	9.6fL	-	

This is an example of a CBC with differential lab report. The components of a typical CBC with diff include:

Red blood cell count (RBC). This is the total number of RBCs in a specific volume of blood. This value does not give any indication of how the RBCs are functioning, just the total number. Low numbers could be an indication of blood loss, hemorrhaging and certain cancers. High levels could be an indication of time spent in high altitudes, pulmonary fibrosis or dehydration.[3,5]

Hemoglobin. This represents the amount of oxygen-carrying protein (hemoglobin) in a sample and reflects the number of RBCs present. This is used to screen patients for anemia and other conditions that can result in RBC breakdown. Low Hb levels may indicate anemia, blood loss or deficient levels of vitamin B6 or B12. High levels maybe an indication of sickle cell disease, high altitude or dehydration.[3,5]

Hematocrit. This value is related to the percentage of total blood volume comprised of RBCs. It is closely related to hemoglobin levels. Low levels can be an indication of anemia, blood loss, certain cancers or vitamin B6 or B12 deficiencies. Elevated levels can be an indication of high altitude or dehydration.[3,5]

Red blood cell indices. This includes mean corpuscular value (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC) and RBC distribution width (RDW). These indices provide information such as RBC size, weight and hemoglobin concentration and are helpful in classifying anemias. Low values could be an indication of iron deficiency, microcytic anemia or thalassemia. Elevated levels may indicate folic acid or vitamin B12 deficiency, alcohol abuse or macrocytic anemia.[3,5]

White blood cell count (WBC) and differential. This reflects the number of WBCs per volume of blood. The differential provides detailed information about the types of WBCs (e.g., neutrophils, eosinophils, lymphocytes) present, along with percentages. Low levels may be an indication of autoimmune diseases, liver or spleen disease, radiation sickness or bone marrow dysfunction. Elevated levels maybe present in infectious diseases (bacterial, viral or protozoan), inflammatory conditions, leukemia or severe emotional/physical stress.[3,5]

Platelet count. This value represents the number of platelets per volume of blood and is useful in diagnosing and managing blood clotting disorders and other diseases. Low values may be an indication of conditions such as leukemia, chemotherapy, hemolytic anemia or vitamin B12 or folate deficiency. Elevated levels maybe present in conditions such as rheumatoid arthritis or certain malignancies.[3,5]

Diabetes

This condition is a growing epidemic with significant physical, emotional and economic ramifications. Patients with diabetes should have yearly eye exams to assess for any ocular manifestations of the disease. However, with a large number of undiagnosed patients, it is not uncommon for an eye examination to be the first indication of a patient having diabetes.[4,7]

Diabetes is primarily diagnosed based on plasma glucose testing. Three main blood glucose tests exist (*Table 4*):

Fasting plasma glucose (FPG). This is the plasma glucose levels in the blood after the patient has refrained from eating or drinking anything but water for eight hours.[8]

Glycosylated hemoglobin test (A1c). This reflects the percentage of free glucose bound to hemoglobin in RBCs over a three-month time span. It is important to remember that A1c is an indirect measure of a patient's average blood glucose levels. Clinicians should consider additional tests when diagnosing diabetes. The National Health and Nutrition Examination Survey data determined that the accepted A1c cut-off of >6.5% (48mmol/mol) identifies one-third fewer cases of undiagnosed diabetes than a fasting glucose cut-off point of >126mg/dL (7.0mmol/L).[8]

Oral glucose tolerance test (OGTT). This tests the patient's blood glucose levels before and two hours after drinking 75g of anhydrous glucose dissolved in water. This test is often done at the same time as FPG. The FPG level is the pre-test glucose level and the OGTT value is two hours after drinking the glucose solution. An OGTT test of >200mg/dL (11.1mmol/L) is considered abnormal.[8]

All three tests are thought to be equally appropriate for diagnostic testing, but a combination of A1c with either a FPG or OGTT results

in a more accurate diagnosis.[8]
The 2017 American Diabetes Association Standards for Diabetes Care recommends clinicians consider testing for Type 2 diabetes in asymptomatic adults of any age who are overweight (BMI >25kg/m^2 or >23kg/m^2 in Asian Americans) and who have one or more additional risk factors for diabetes.[8] In addition, screening should begin for all patients at age 45 and, if tests are normal, repeated at a minimum of three-year intervals. Patients who are suspicious for diabetes or have been diagnosed can benefit from further recommended testing, including:[8]

- fasting lipid profile
- liver function tests
- urinary albumin-to-creatinine ratio
- serum creatinine and estimated glomerular filtration rate
- thyroid stimulating hormone (TSH) levels in Type 1 diabetes

Case Example:
A 56-year-old white patient presented as a walk-in because he broke his current pair of glasses. He was a long-haul truck driver from out of state and needed a new pair of glasses as quickly as possible. His medical history was negative and he wasn't taking any medications. He was a pack-a-day smoker and occasionally consumed alcohol. His uncorrected VAs were 20/50 OD and 20/40 OS. Entrance skills were unremarkable, manifest refraction revealed +2.25 –0.50 x 180 (20/20) OD and +2.00 DS (20/20) OS. Slit lamp results showed arcus 360 degrees in both eyes. IOPs were 16mm Hg and 17mm Hg. Blood pressure was 125/85mm Hg. Undilated fundus evaluation revealed scattered dot and blot hemes/exudate—prompting dilation (*Figure 3*).

I told the patient that I didn't remember him mentioning that he had diabetes. He said he doesn't, though his wife does. I educated him on the present signs of changes to the back of his eye consistent with diabetes and recommend he see his PCP as soon as possible, as blood glucose testing was not available at this office.

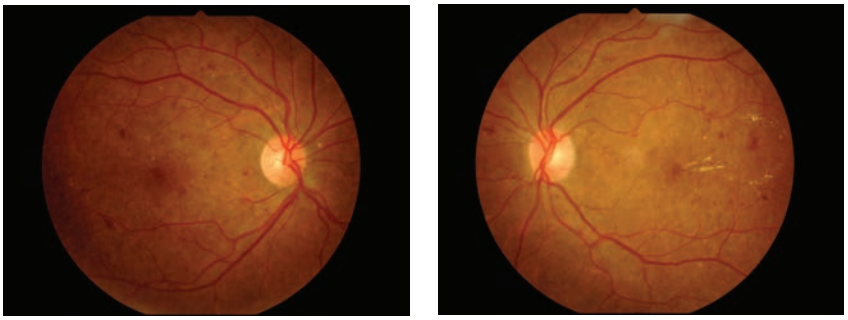

Fig. 3. This 56-year-old male's fundus images indicate the presence of hemorrhaging and exudates associated with diabetic retinopathy.

Two hours later, I received a call from an emergency room physician wanting to discuss the patient. The physician wanted to know why I told the patient he needed to have his blood pressure checked when it is normal. I clarified that I was concerned about his blood sugar, not blood pressure, as I had a strong suspicion that he has diabetes, given my findings of retinal hemorrhages and exudate. The physician wasn't convinced, but agreed to check his blood glucose levels.

The patient called me two days later and stated that the ER doctor confirmed that he had diabetes with a random plasma glucose level of 380mg/dL and an A1c value of 10.2.

Liver and Kidney Testing

These labs are ordered for a variety of systemic conditions such as diabetes, autoimmune diseases and sarcoidosis—many of which have ocular manifestations. Liver and kidney testing may also be requested in patients taking certain medications, including oral antivirals for prophylaxis of herpes simplex virus, herpes simplex zoster or both.

Kidney function tests. Three main tests are common to test kidney function (*Table 1*). Blood urea nitrogen (BUN) measures the amount of urea nitrogen in the blood, which becomes elevated in patients with kidney dysfunction. A markedly increased BUN is conclusive evidence of severe impaired glomerular function.[3,4] Creatinine is produced at a constant rate depending on a person's muscle mass and is removed from the body by the kidneys. Production of creatinine is constant as long as muscle mass remains constant and reflects renal function. Abnormal elevations as measured by serum creatinine testing indicate renal function impairment.[3] The glomerular filtration rate estimates the amount of blood that passes through the glomeruli each minute and provides a good indication of kidney function (and possible staging of kidney disease).[3]

Liver function tests. Alanine aminotransferase (ALT) is an enzyme found in the liver in high concentrations and relatively low concentrations in the heart, muscles and kidneys. Aspartate transaminase (AST) is an enzyme present in tissues of high metabolic activity and is released into the blood after cell damage. ALT and AST are both often elevated in liver disease and are an indirect measure of liver damage. These enzymes are normally inside liver cells, and their presence in the blood indicates liver cell damage.[3] ALT is a better indicator of liver

Table 3. A Representative Lipid Profile

Component	Your Value	Standard Range
Cholesterol	201mg/dL	≤239mg/dL
Triglyceride	99mg/dL	≤199mg/dL
HDL Chol	39mg/dL	≥40mg/dL
LDL Calc	142mg/dL	≤159mg/dL

Table 4. Criteria for Pre-diabetes and Diabetes Testing[8]

	Pre-diabetes	Diabetes
FPG	100mg/dL to 125mg/dL (5.6mmol/L to 6.9mmol/L)	>126mg/dL (7.0mmol/L)
OGTT	140mg/dL to 199mg/dL (7.8mmol/L to 11.0mmol/L)	>200mg/dL (11.1mmol/L)
A1c	5.7% to 6.4%	>6.5%

damage than AST, but both tests are typically ordered together. Bilirubin—the breakdown product of erythrocyte hemoglobin—is eliminated by the liver in the bile. Elevated levels can be an indication of liver damage and is seen in patients with cirrhosis and hepatitis.[3] Albumin, which maintains colloidal osmotic pressure in the vascular and extravascular spaces, is a source of nutrition and part of a complex buffer system. A decrease in albumin levels can indicate an acute inflammatory infectious process. Albumin can be used to determine nutritional status, acute illness, liver and kidney damage and other chronic diseases.[3]

Inflammatory Markers

Testing for systemic inflammatory markers is a crucial diagnostic component for potentially sight-threatening conditions such as ischemic optic neuropathy (ION). The two most common tests include erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).[3,9]

ESR. In response to acute and chronic inflammation, the amount of protein (mostly fibrinogen) increases in the plasma, encouraging the RBCs to stick together.[3,4] ESR measures the rate at which the RBCs settle out of solution during a specified period of time, which can be the result of some infections, collagen-vascular diseases, inflammatory diseases and tissue damage from myocardial infarction.[3] While the presence of elevated ESR is nonspecific and doesn't indicate where the inflammation is coming from, it can be used when monitoring for decreased levels. ESR is indicated in patients suspected of having temporal arteritis and uveitis-related systemic disease.[3,9]

CRP. This an acute phase protein that is more sensitive than the ESR, responds more quickly to the presence of inflammation and disappears faster on resolution. Standard CRP is indicated to assess a patient for systemic inflammation, while high-sensitivity CRP is primarily used for cardiac disease assessment.[3,4]

An ESR, CRP or both should be ordered for patients with suspected ION to help rule out the arteritic form (temporal or giant cell arteritis), especially in patients who are experiencing scalp tenderness, jaw claudication with a unilateral swollen optic nerve or both.[4]

Autoimmune Disorders

This is a varied group of conditions in which the body's innate immune system sets up a chronic inflammatory response to its own tissues.[9] Because ocular manifestations—such as dry eye, episcleritis, scleritis, uveitis, retinal hemorrhages, ION and retinal vasculitis—may be the first sign of an underlying autoimmune disease, optometrists must be aware of the possible underlying systemic cause and appropriate lab testing.[9] Some of the most common autoimmune disorders that optometrists come in contact with include rheumatoid arthritis (RA), systemic lupus erythmatosus (SLE), HLA-B27-associated conditions, Sjögren's syndrome, Graves' disease and multiple sclerosis. Several lab tests (*Table 5*) can help uncover autoimmune dysfunction, including:

Rheumatoid factor (RF) and anticyclic citrullinated antibodies (anti-CCP). These two markers, used to help diagnose RA, have similar sensitivity (67% for anti-CCP vs. 69% for RF), although anti-CCP is more sensitive (95% vs. 85% for RF) in its predictive value for development of erosive disease.[9,10] However, in the early stages of RA, patients may test negative with both. As there are characteristic joint changes seen in RA, patients who test negative but have symptoms should be referred for radiographic studies of hands and feet (primary tissues and joints affected in RA) for baseline and to aid in diagnosis. Other rheumatic diseases may also test positive for RF, and as patients age they may test positive for RF but not have RA.[10,11]

Antinuclear antibodies (ANA). The presence of ANA in serum supports an SLE diagnosis and related autoimmune diseases, while its absence in a patient with suspected SLE makes the diagnosis much less likely.[12] ANA results are typically presented as an endpoint titer and the staining pattern (or patterns) produced by the patient's serum. Nuclear staining patterns are loosely associated with the underlying autoimmune disease and include: homogeneous, speckled, centromere and nucleolar.[12] ANA is primarily a screening test for lupus, but is not diagnostic of the condition, as other diseases will test positive for ANA, including scleroderma (95%), mixed connective tissue disease (100%), Sjögren's syndrome (60%), RA

(50%) and juvenile idiopathic arthritis (15% to 40%).[13]

Confirmatory testing is required to diagnose lupus in conjunction with presenting signs and symptoms. Antidouble-stranded DNA (anti-dsDNA) and anti-Smith (anti-Sm) antibodies are highly specific for SLE, but anti-Sm antibodies lack sensitivity. Anti-dsDNA and anti-Sm antibodies are seen in approximately 70% and 30% of patients with SLE, respectively.[13] The Sjögren's syndrome antibody Anti-Ro/SSA and anti-La/SSB antibodies are present in approximately 30% and 20% of patients with SLE, respectively; however, both antibodies are more commonly associated with Sjögren's syndrome.[11,13]

Human leukocyte antigen (HLA). The HLA encodes proteins on the surface of leukocytes that have a critical role in immunity, including antigen processing and presentation to T-helper cell and self-recognition by immune cells.[14] Research strongly links HLA-B27 to several spondyloarthropathies, including ankylosing spondylitis, psoriatic arthritis, reactive arthritis and the inflammatory bowel diseases (Crohn's and ulcerative colitis).[14] HLA-B27 is also strongly associated with acute anterior uveitis and recurrent acute anterior uveitis in particular, with as much as 80% of patients with recurrent acute anterior uveitis being HLA-B27.[14,15] However, regardless of the strong association, research suggests HLA testing has limited usefulness in diagnostic testing in uveitis patients. Only 1% of patients who test positive for HLA-B27 are likely to develop uveitis, and patients who present with uveitis secondary to an HLA-B27 condition are likely to have other signs and symptoms indicative of the disease process.[14,15] If a patient does test positive for HLA-B27, they could have one of several conditions, so it doesn't necessarily help in the diagnostic process.

Thyroid testing. In hyperthyroidism, or Graves' disease, an overactive thyroid gland secretes excessive T_3 and T_4, which results in low thyroid-stimulating hormones.[16,17] Because T_3 and T_4 are decreased in hypothyroidism, the pituitary secretes more TSH.[16,17] Ultrasensitive serum TSH studies have the highest sensitivity and specificity to screen for both hypo- and hyperthyroidism. An additional test can detect the presence of free serum T_4 and T_3.[16]

In Graves' disease, the most common cause of hyperthyroidism, patients will experience heart palpitations, heat intolerance, weight loss, skin rash and ophthalmopathy (25% to 85%).[17] The prevalence of distinct ocular abnormalities are: eyelid retraction, 92%; exophthalmos,

Autoimmune Masquerader

A condition with characteristics similar to an autoimmune disease is sarcoidosis, the exact etiology and pathogenesis of which remain unknown, although multiple causes are possible.[1]

Sarcoidosis is one of the main causes of inflammatory eye disease, and one study found ocular involvement was the presenting symptom in approximately 20% to 30% of patients.[2] The researchers noted uveitis, typically bilateral, granulomatous and chronic, in 30% to 70%, with conjunctival nodules seen in 40%.[2]

Sarcoidosis suspects require an extensive work-up, including a complete physical exam, laboratory testing (including CBC, ESR/CRP, kidney and liver assays, and angiotensin converting enzyme [ACE]) and pulmonary radiographic studies.[1] Even though ACE level is elevated in 75% of untreated sarcoidosis patients, the test has limited utility due to poor sensitivity and insufficient specificity (almost a 10% rate of false positive results).[1] Pulmonary radiographic studies often start with a chest radiograph, followed by high-resolution computed tomography. Bilateral hilar adenopathy is a classic finding in sarcoidosis. If the diagnosis of sarcoidosis is unclear, additional scans such as positron emission tomography or gallium-69 radioactive tracer may help.[1]

Case Example:

A 28-year-old black male was referred for glaucomatocyclitic crisis. His medical history was unremarkable and he was taking no medications, although he does have a positive family history of sarcoidosis (sister). VAs were 20/40 OD, OS (no improvement on pinhole), and entrance skills were unremarkable. Slit lamp revealed 3+ conjunctival injection in both eyes, 4+ cell OD (plasmoid aqueous) and 3+ cell OS and mutton fat keratic precipitates OU. IOPs were 28mm Hg OD and 14mm Hg OS. Although fundus evaluation was challenging, 2+ vitreal cells were noted OD and 1+ OS. The fundus appeared unremarkable OU.

The patient was diagnosed with bilateral granulomatous uveitis with elevated IOP secondary to the uveitis OD. The patient was started on Pred Forte 1% (Allergan) every hour OU, homatropine 5% BID OU and timolol 0.5% BID OD. The patient was referred to his PCP for a uveitis work-up with an emphasis on sarcoidosis testing.

On follow up three days later, he showed improvement in his anterior chamber reaction (3+ OD and 2+ OS) and reported that he saw his PCP and was told there wasn't any conclusive evidence of sarcoidosis, as his ACE testing was 48nmol/mL/min (typical adult levels between 8nmol/mL/min and 53nmol/mL/min) and they found no granulomas on his chest x-rays.[3]

We treated the patient over the next six months with topical steroids and oral steroids; however, whenever it appeared his inflammation had cleared and we began tapering, he would flare up. We referred the patient back to his PCP and requested repeat lab testing and advanced imaging. The patient received a gallium-69 scan, which confirmed sarcoid granulomas in his lungs.

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62%; extraocular muscle dysfunction, 43%; ocular pain, 30%; increased lacrimation, 23%; and optic neuropathy, 6%.[17]

Clinicians should consider TSH, free T_4/T_3, orbital imaging or a combination of all three in any patient exhibiting signs of Graves' disease.[16,17] Thyroid testing should also be considered for a patient presenting with superior limbic keratoconjunctivitis, which is associated with thyroid dysfunction.[18] Patients typically present with a gritty or foreign body sensation, superior redness or injection and hyperemia. Slit lamp results demonstrate superior palpebral or bulbar conjunctival inflammation with the development of pannus in the superior one-third of the cornea.[18]

Table 5. Lab Testing for Autoimmune Disorders

Autoimmune Disorder	Lab Testing
Rheumatoid arthritis	• RF • Anti-CCP • Radiographic testing of hands/feet • ANA • CBC
Systemic lupus erythematosus	• CBC (leukopenia, anemia, thrombocytopenia) • ESR/CRP • ANA • Urinalysis (hematuria, proteinuria) • Antiphospholipid antibodies • Anti-dsDNA • Anti-Sm antibodies • Ro/SSA • La/SSB
Ankylosing spondylitis Psoriatic arthritis Reactive arthritis Inflammatory bowel diseases	• HLA-B27
Graves' disease	• TSH • Free T_4/T_3 • Orbital imaging for suspected orbital changes
Sarcoidosis	• CBC • ESR/CRP • Liver testing • Kidney testing • ACE • Pulmonary radiographic studies

Case Example:
A 33-year-old Hispanic female presented to the clinic complaining about an acute-onset, painful red right eye. It started a couple of days ago with a "nodule" and redness, and the pain seemed to be going through her entire eye. She tried a "take the red out" eye drop that helped. Her medical history is positive for RA, diagnosed three years previously. She stated that she didn't have a PCP at the time and had been taking Celebrex (celecoxib, Pfizer) for her joint pain. Upon further questioning, she reported she occasionally gets a rash when out in the sun for too long, and her mother also had RA.

Entering uncorrected VAs were 20/40 OD (no improvement on pinhole) and 20/20 OS. Entrance skills were unremarkable, although she did experience eye pain on eye movement. Blood pressure was 125/80mm Hg. Slit lamp exam revealed an elevated, sectoral 3+ redness on the nasal palpebral conjunctiva (the deep scleral vessels appeared to be engorged), 2+ cells and trace flare (*Figure 4*). OS was unremarkable. IOPs were 18mm Hg OD, OS, and dilated fundus exam revealed cotton wool spots in both eyes.

We diagnosed the patient with a non-necrotizing scleritis. Although typical first-line therapy is oral nonsteroidal anti-inflammatory drugs (NSAIDs), she was already taking Celebrex, which didn't seem to be managing the condition.[15] Upon further questioning, she was unable to tell us who diagnosed her with RA or what tests were done. With the diagnosis of scleritis and the presence of cotton wool spots in both of her eyes, we began to question her diagnosis of RA and made an urgent referral to rheumatology for SLE assessment.

The report from rheumatology confirmed a diagnosis of lupus. Her lab results were positive for ANA, and then confirmed with anti-dsDNA as well as anti-Sm. The rheumatologist was concerned she was going through a "flare up," given her active scleritis, and additional lab testing that indicated that she had abnormal kidney, liver and cardiac enzymes. Because of these concerns, the rheumatologist bypassed the second-line therapy of oral steroids and went directly to systemic immunosuppressive therapy (oral cyclophosphamide).[19] She was also taken off Celebrex and started on hydroxychloroquine 400mg per day to help manage her joint pain and skin rashes.

Infectious Disease

A variety of ocular conditions can manifest secondary to a systemic infectious disease, including hyperacute conjunctivitis, keratitis, uveitis and retinal vasculitis. The main underlying infectious diseases that can manifest ocular complications include chlamydia, gonorrhea, syphilis, herpes, tuberculosis (TB) and Lyme disease. Most of these conditions have their own diagnostic lab testing:

Nucleic acid amplification test (NAAT). This is the diagnostic test of choice for chlamydial and gonorrhea infection of the genitourinary tract and uses vaginal swabs for women or first-catch urine for men. Conjunctivitis (typically hyperacute) is the most common ocular manifestation of these conditions.[1,20,21]

Syphilis testing. There are two types of serologic tests for syphilis: nontreponemal and treponemal-specific. The use of only one test is insufficient for diagnosis since serologic testing (especially nontreponemal tests) can be associated with false positive results.[24] Ocular syphilis can involve almost any eye structure, but posterior uveitis and panuveitis are the most common and present with diminished VA. Additional findings can include interstitial keratitis, anterior uveitis, optic neuropathy and retinal vasculitis.[23]

Nontreponemal tests are nonspecific and have traditionally been used for initial syphilis screening due to their relatively low cost, ease of performance and ability to be quantified for the purpose of following response to therapy.[22] The tests include rapid plasma reagin (RPR), venereal disease research laboratory and the toluidine red unheated serum test.[22]

Treponemal tests are more complex and expensive to perform than nontreponemal tests and have traditionally been used as confirmatory tests for syphilis when the nontreponemal tests are reactive. However, newer versions of these tests are automated, enhancing simplicity and facilitating ease of use and enabling their use in initial testing.[22] These test include: fluorescent treponemal antibody absorption, microhemagglutination test for antibodies to *Treponema pallidum*, *T. pallidum* particle agglutination assay, *T. pallidum* enzyme immunoassay and chemiluminescence immunoassay.[22]

Lyme testing. Lyme disease is the most common tick-borne disease in the United States, Canada and Europe.[24] A variety of ocular manifestations have been associated with Lyme disease, including conjunctivitis (most common), keratitis, iridocyclitis, retinal vasculitis, choroiditis, optic neuropathy and uveitis. Serologic testing should be performed in patients who meet *all* of the following criteria:[24]

- A recent history of having resided in or traveled to an area endemic for Lyme disease.
- A risk factor for exposure to ticks.
- Symptoms consistent with early disseminated disease or late Lyme disease (e.g., meningitis, radiculopathy, mononeuritis, cranial nerve palsy, arthritis, carditis).

Serologic testing for anti-*Borrelia burgdorferi* antibodies, a two-tier conditional strategy, is recommended to support the diagnosis of Lyme disease. The traditional two-tiered testing algorithm uses a sensitive enzyme immunoassay, such as a whole cell-based enzyme-linked immunosorbent assay followed by a more specific Western blot test. Separate IgM and IgG blots are typically performed.[24]

Tuberculosis. This disease should be suspected in patients with relevant clinical manifestations (cough of more than two to three weeks' duration, lymphadenopathy, fevers, night sweats, weight loss) and relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, past or present residence in or travel to an area where TB is endemic).[25] Ocular manifestations of TB include choroiditis, chorioretinitis, choroidal granuloma, optic neuritis, orbital cellulitis, scleritis, necrotizing scleritis, posterior scleritis, interstitial keratitis and anterior chamber granuloma.[26]

Patients suspected of being exposed to TB should have a tuberculin skin test or interferon-gamma release assay, as they are designed for diagnosis of TB infection; a positive result supports (but cannot be used to establish) a diagnosis of active TB disease, and a negative result does not rule out active TB disease.

A chest x-ray is required, and if suspicion of TB is present, three sputum samples are recommended for smear analysis, culturing and nucleic acid amplification testing.[25]

Optometry plays a crucial role as part of the health care team in the management of patients' ocular and systemic health. Optometrists must be familiar with the specific lab testing that is often required in the diagnosis and management of systemic conditions with associated ophthalmic manifestations.

Dr. Lonsberry is a professor of optometry at Pacific University College of Optometry.

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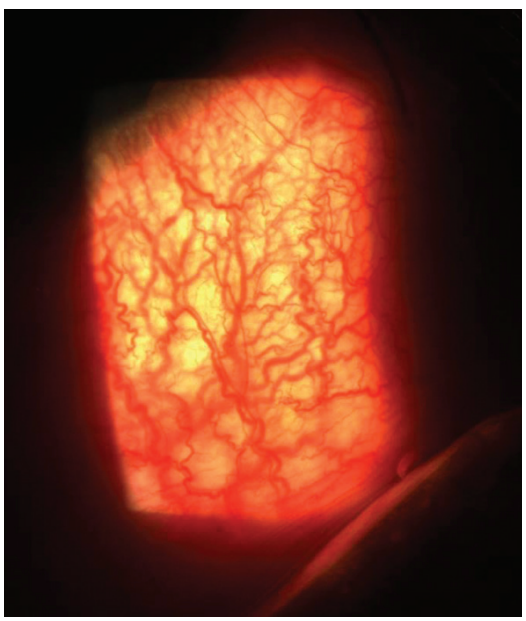

Fig. 4. This 33-year-old Hispanic female presented with scleritis. This image highlights inflammation of the deeper scleral vessels.

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OSC QUIZ

You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form and return it with the $35 fee to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. To be eligible, please return the card within one year of publication.

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

1. Which lab test is the most specific for liver damage?
a. Alanine aminotransferase.
b. Aspartate aminotransferase.
c. Creatinine.
d. Blood urea nitrogen.

2. The primary tissues affected in rheumatoid arthritis are:
a. Hands and feet.
b. Knees and elbows.
c. Hips and ankles.
d. Lower back.

3. Which of the following is not a characteristic finding in a patient diagnosed with rheumatoid arthritis?
a. Rheumatoid factor.
b. Antinuclear antibodies.
c. Joint x-rays or radiographic testing.
d. Anticyclic citrullinated antibodies.

4. What percent of patients with recurrent anterior uveitis are likely to be HLA-B27 positive?
a. 10%.
b. 30%.
c. 50%.
d. 80%.

5. What lab test is considered a confirmatory test for a patient suspected of having a syphilis infection?
a. Fluorescent treponemal antibody absorption.
b. Rapid plasmin reagin.
c. Angiotensin converting enzyme.
d. Nucleic acid amplification test.

6. All of the following statements about tuberculosis are true, except:
a. Three sputum samples are recommended for analysis in patients suspected of active TB.
b. A positive tuberculin skin test confirms the diagnosis that a patient has been exposed to tuberculosis and has either an active or latent infection.
c. Chest x-rays can help diagnose an active infection.
d. Interferon-gamma release assays can be used to help in the diagnosis of TB.

7. What lab test would be indicated for a patient you suspect for systemic lupus erythematosus and is used as a first-line screening test?
a. Chest x-rays.
b. Antinuclear antibodies.
c. Angiotensin converting enzyme.
d. Rheumatoid factor.

8. What is typically elevated in a patient with active sarcoidosis?
a. Antinuclear antibodies.
b. Antidouble-stranded DNA antibodies.
c. Angiotensin converting enzyme.
d. Fluorescent treponemal antibody absorption.

9. What lab test is typically elevated in a patient with active systemic lupus erythematosus and is used as a confirmatory test in diagnosis?
a. Antinuclear antibodies.
b. Antidouble-stranded DNA antibodies.
c. Angiotensin converting enzyme.
d. Anticyclic citrullinated antibodies.

10. Which of the following is not an HLA-B27-related condition?
a. Reactive arthritis.
b. Inflammatory bowel disease (Crohn's).
c. Rheumatoid arthritis.
d. Psoriatic arthritis.

11. What lab test would be the most beneficial in diagnosing a patient with ankylosing spondilitis?
a. Antinuclear antibodies.
b. Chest x-rays.
c. HLA-B27.
d. Rheumatoid factor.

12. A patient who presents with a panuveitis and an interstitial keratitis should be screened with which lab test?
a. Antidouble-stranded DNA antibodies.
b. Rheumatoid factor.
c. Antinuclear antibodies.
d. Rapid plasmin reagin.

13. What lab test should be ordered immediately if you suspect a patient is suffering from giant cell arteritis?
a. Complete blood count.
b. White blood count.
c. Antinuclear antibodies.
d. C-reactive protein.

14. Which of the following is not typically ordered in a patient suspected with Sjögren's syndrome?
a. Sjögren's syndrome antibodies.
b. Rheumatoid factor.
c. Antinuclear antibodies.

OSC QUIZ

d. Rapid plasmin reagin.

15. What test is primarily used in the diagnosis of a patient suffering from either chlamydia or gonorrhea?
a. Rapid plasmin reagin.
b. Fluorescent treponemal antibody absorption.
c. Nucleic acid amplification test.
d. Angiotensin converting enzyme.

16. What is the most prevalent eye complication associated with Graves' disease?
a. Exophthalmos.
b. Optic neuropathy.
c. Extraocular muscle dysfunction.
d. Lid retraction.

17. What lab test can give an indication of a patient's nutritional status (i.e., low levels are found in patient's with poor nutritional status)?
a. Blood urea nitrogen.
b. Albumin.
c. Creatinine.
d. Bilirubin

18. What lab test is indicative of kidney function because it is typically produced in normal levels based on a patient's body mass?
a. Blood urea nitrogen.
b. Albumin.
c. Creatinine.
d. Glomerular filtration rate.

19. Which of the following is a diagnostic criteria for a patient suspected of having diabetes?
a. Fasting plasma glucose >126mg/dL.
b. Glycosylated hemoglobin >6.5%.
c. Oral glucose tolerance test >200mg/dL.
d. All of these are diagnostic for diabetes.

20. Which of the following is considered the "good cholesterol"?
a. High-density lipoprotein cholesterol.
b. Low-density lipoprotein cholesterol.
c. Triglycerides.
d. Very low-density lipoproteins.

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19. Ⓐ Ⓑ Ⓒ Ⓓ
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 understanding of the common laboratory tests ODs can order. ① ② ③ ④ ⑤
22. Better understand the results of various lab tests and how they impact a patient's diagnosis. ① ② ③ ④ ⑤
23. Increase my understanding of a complete blood count test. ① ② ③ ④ ⑤
24. Become familiar with testing for diabetes, as well as liver and kidney conditions. ① ② ③ ④ ⑤
25. Increase my knowledge of lab testing for autoimmune diseases. ① ② ③ ④ ⑤
26. Improve my knowledge of testing for inflammatory and infectious diseases. 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
30. Additional comments on this course:

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

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Scleral Battle: Oblate vs. Prolate

Some doctors prefer to use one scleral lens design over the other. Here's why.

Edited by Joseph P. Shovlin, OD

Q Several scleral lens experts have recommended I use oblate designs instead of prolate designs when I can't attain good visual acuity with any over-refraction or flexure control in a patient with irregular corneas. Why?

A "The terms oblate and prolate describe the shape of the eye as either more flat or more steep in the center, respectively," Keith Parker, president and CEO of Advanced Vision Technologies, says. "In my opinion, however, regarding scleral lenses, the terms reflect nothing more than marketing hype. There is no difference between them except for a shallower sagittal depth" in an oblate design. And that's the key to a successful fitting for some patients.

Consider the Consequences

"Many [prolate] scleral lens designs traditionally generate increased sagittal depth to fit deeper eyes such as those with advanced keratoconus by using increasingly steep base curves," says Jason Jedlicka, OD, clinical associate professor and chief of the Cornea and Contact Lens Service at the University of Indiana. "While this accommodates the lens fitting, as lens base curves become steeper the tear layer behind the lens becomes increasingly convex, adding a high degree of plus power to the system."

When you compensate for the higher plus power with a higher minus power, it in turn impacts the quality of vision, Dr. Jedlicka says. "Whether due to image minification, optics set further from the corneal plane or a slight increase in higher-order aberrations, visual acuity can be reduced in high minus power scleral lenses in some instances."

Thus, "with a higher sagittal depth, the tear layer may create more distortion due to the increased prismatic effect of the optical path," Mr. Parker says.

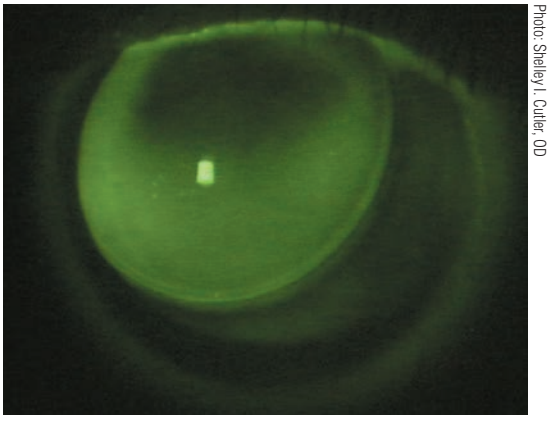

Photo: Shelley J. Cutler, OD

By incorporating flatter base curves, oblate scleral lenses can improve quality of vision.

Solving the Problem

Oblate designs, however, incorporate flatter base curves—often leading to better quality of vision.

"Flatter base curves allow lens powers to be significantly lower, often close to plano, as the vision is corrected with the tear layer under the lens," Dr. Jedlicka says. "Because of the reduced lens power and since the lens center can be set closer to the corneal plane, the impact of minification is reduced." As a result of this and the potential for a larger optic zone (which can be achieved due to the lower lens power), he says higher-order aberrations can also be mildly reduced.

"Flatter base curves and lower minus powers always give better vision and less distortion," says Chris Sindt, OD, clinical professor of ophthalmology and visual sciences at the University of Iowa. "For example, if I could go from a 55.00D base curve to a 45.00D base curve, I could take a -10.00D power to plano, but the converse is true, too. If the patient were aphakic, I could go from a +10.00 to +20.00, which could lead to worse vision." According to Mr. Parker, "a flatter base curve on high minus patients may help with the overall power needed for the final Rx."

According to Mr. Parker, a flatter base curve is also often used in conjunction with the shallower sagittal depth, which also helps to improve vision. "Remember, the base curve does not dictate the sagittal depth; it is the curves beyond the base curve that have the most significant effect on sagittal depth," he says.

At the end of the day, Dr. Sindt says, these cases "entirely depend on the disease and cornea below the lens." One lens design may work better than the other, and knowing how the design will affect the optics is key to choosing the right one. ■

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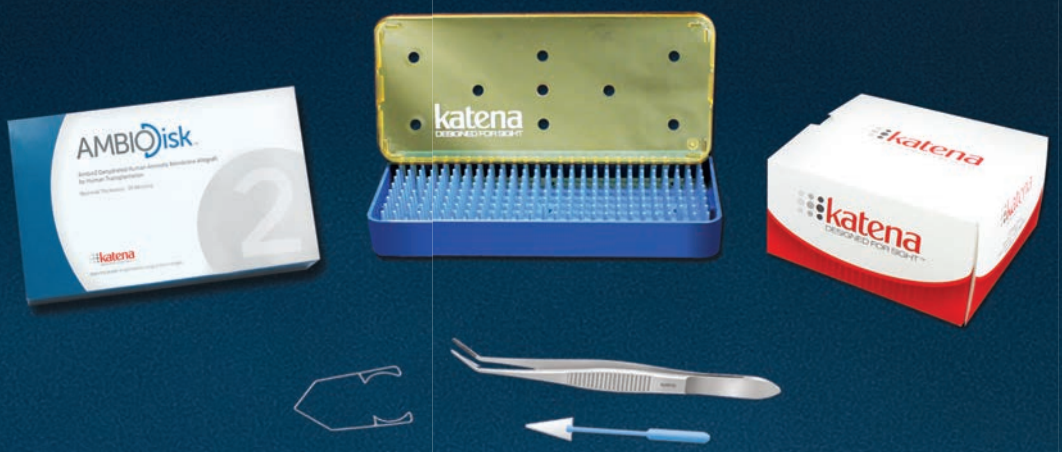

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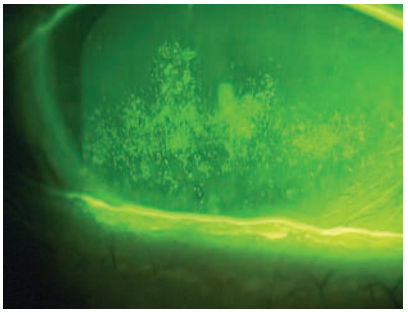
KB-Adv-011518-Rev0

HA: No Laughing Matter

Most doctors don't realize the extent to which we have or will have access to hyaluronic acid for dry eye in the United States. **By Paul M. Karpecki, OD**

Hyaluronic acid (HA) eye drops (i.e., sodium hyaluronate and hyaluronan) are the most commonly used dry eye therapies in Europe and Asia.[1] Even our Canadian neighbors have an over-the-counter option with Hylo (sodium hyaluronate, CandorVision). While taking a closer look at a therapy you don't have may seem frivolous, it makes plenty of sense once you see the various products we *can* access and the future role of HA in the United States. This successful treatment option might be more accessible than you think.



Patients with signs and symptoms of dry eye disease, such as the confluent superficial punctate keratopathy seen here, often benefit from therapies containing hyaluronic acid.

What's Special About HA

HA is a high molecular weight naturally occurring polysaccharide known as a gycosaminoglycan. It is found in the human body in connective tissue, synovial fluid and in the aqueous humor and vitreous of the eye.[2,3] HA has three primary characteristics that make it useful:

An ability to bind or retain water. HA has a high concentration of hydroxyl groups that result in hydrogen binding.[4] Studies show it can bind up to 1,000 times its weight in water, making it highly valuable as a wetting agent.[5] The unique structure involves coiled chains that allow water to be trapped within them.[6]

Viscoelasticity. This allows for shear stress and has been shown to stabilize the tear film and reduce symptoms of DED.[7]

Wound healing. Research shows HA clinically aids in epithelial cell proliferation, epithelial healing and epithelial migration.[8-12] Studies also demonstrate HA's ability to increase healing of corneal abrasions and alkali chemical injuries in rabbits.[13]

How You Can Use It

Research shows HA is quite effective in improving the symptoms of dry eye disease (DED), visual functioning in dry eye and even post-surgical patients.[14] Thus, manufacturers include it in several products on the market today, and researchers are exploring its effectiveness for any number of therapies:

Artificial tears. Viscosity-enhancing agents are critically important to artificial tear products, and different products use various agents, such as HA, polyacrylic acid, carboxymethyl cellulose, dextran, HP-guar, hydroxypropyl methyl cellulose, polyvinyl alcohol, polyvinylpyrrolidone and polyethylene glycol.[15]

A key characteristic of a high-quality HA therapy is the molecular weight, which is related to the length of the chain of each molecule. High molecular weight HA will be more cohesive and low molecular weight HA will be more dispersive. Examples of drops containing long-chain HA include Oasis Tears, Oasis Tears PF and Oasis Plus (Oasis Medical).

The concentration of HA does not necessarily reflect the molecular weight, as it is possible to have a high concentration of low molecular weight HA in a formulation.

Research shows Blink Contacts (Johnson & Johnson Vision), which contains 0.15% HA, increases tear break-up time and improves comfort and lens tolerance in contact lens-wearing patients.[16,17] Other studies of HA also noted increased tear break-up time for both evaporative and aqueous deficient DED.[18]

Contact lenses. Although most research focuses on the use of HA in dry eye and corneal wound healing, some studies are specific to contact lens wear. One study of 3 o'clock and 9 o'clock staining in patients wearing rigid gas permeable (RGP) lenses showed a statistical improvement in those treated with 0.1% sodium hyaluronate after two weeks compared with those treated with artificial tears.[19] HA can also decrease protein adhesion to contact lenses and can increase hydrophilicity.[20]

Researchers have also been working on hydrogel contact lenses that can release HA at a controlled rate.[21,22] Studies show daily disposable and extended wear contact lenses infused with HA—either by the soaking method or

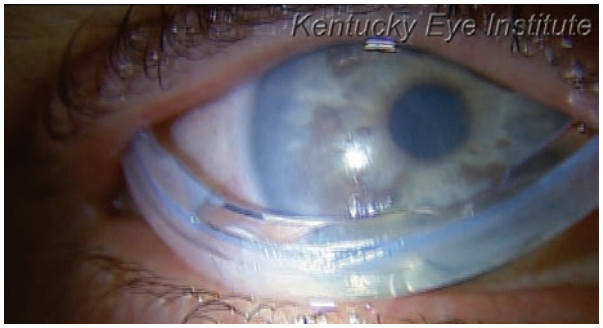
direct entrapment—allow for slow release of HA throughout the day and have proven better wettability and even improved symptoms of dry eye.[21,22]

The addition of HA to some contact lens solutions such as BioTrue (Bausch + Lomb) could be part of the reason why they work so well for some patients.

Cryopreserve amniotic membrane. New research has also uncovered reasons why cryopreserved amniotic membrane is so effective in corneal wound healing, and one key is long-chain hydrocarbon (HC) HA. In a study that compared cryopreserved with dehydrated amniotic membrane, data shows that cryopreservation better maintains the structural and biological signaling molecules of fetal tissues. The analysis found the HC-HA and pentraxin 3—a protein in the complement pathway of the immune system critical to inflammation treatment—present in cryopreserved formats were absent in the dehydrated tissue.[23]

Crosslinked HA. Finally, exciting technologies on the horizon for eye care include crosslinked HA. This compound resists degradation and adheres to the ocular surface for extended periods, without blurring.[24] A recent study demonstrates that crosslinked HA hydrogel accelerated the time to corneal wound closure compared with a non-crosslinked HA solution in companion animals.[25] This investigation involved 30 dogs and 30 cats with spontaneous acute corneal ulcers being treated with either crosslinked HA or a non-crosslinked HA solution (n=15 per group for each species), three times daily until the ulcer healed.[25] The investigators then used 25 dogs with persistent non-healing corneal ulcers treated twice daily until the ulcer healed. In all cases the crosslinked HA showed significantly accelerated healing time.[25]

A poster at the 2017 American Society of Cataract and Refractive Surgery meeting showed how this therapy translated to humans.[26] Researchers randomized 39 patients



Cryopreserved amniotic membranes, such as the Prokera (Bio-Tissue) seen here mid-insertion, provide HC-HA to the ocular surface and better preserve the structural and biological signaling molecules compared with dehydrated membranes.[23]

Paul Karpecki, OD, FAAO

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undergoing bilateral photorefractive keratectomy (PRK) with greater than 8mm epithelial defects to three groups: those receiving a crosslinked HA and a bandage contact lens, patients with crosslinked HA alone and those with a bandage lens and artificial tears. The most effective treatment was the crosslinked HA alone with more than 83% of the patients healing within three days compared with an average of 53.8% with today's current therapy choice of bandage contact lens post-PRK.[26]

HA has numerous current applications in eye care, and US optometrists can take advantage of its clinical benefits with some artificial tear products, contact lens solutions and amniotic membrane therapies. Given the promising research on HA, clinicians should hopefully have many more exciting innovations to look forward to in the near future. ■

Note: Dr. Karpecki is a consultant for many companies mentioned here.

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Acting on an Epidemic

Here's how to approach the clinical diagnosis and management of EKC.

By Breanne B. McGhee OD, and Richard Mangan, OD

A 43-year-old Caucasian male presented to clinic with an irritated, red and teary right eye ongoing for three days. The patient visited a local urgent care clinic with the same concerns two days prior and was prescribed topical gentamicin TID with no improvement. He also indicated flu-like symptoms weeks prior to the onset of his ocular symptoms, which had since resolved.

His entering distance aided visual acuities were 20/20 OD, OS. A slit lamp biomicroscopy revealed 1+ diffuse bulbar conjunctival injection, thick purulent discharge and negative corneal findings with unremarkable sodium fluorescein staining. The diagnosis of acute conjunctivitis was made and the patient was prescribed topical prednisolone acetate QID OD until his follow-up in a week. He was instructed to discontinue the previously prescribed topical antibiotic.

However, the patient returned two days later with complaints of eye pain, decreased vision, light sensitivity and foreign body sensation in the right eye; all of which had progressively worsened since his initial examination. His visual acuities were measured at 20/40 OD with no improvement to pinhole and stable in the left eye. Anterior segment findings were 3+ bulbar conjunctival injection, mild chemosis, trace follicles in the inferior palpebral conjunctiva, diffuse lid erythema and swelling, and multiple small central corneal infiltrates in the right eye (*Figure 1*). We also observed positive palpable adenopathy on the ipsilateral preauricular aspect of the face, but no evidence of pseudomembranous formation. Based on the clinical presentation and ocular findings, we diagnosed the patient with epidemic keratoconjunctivitis (EKC).

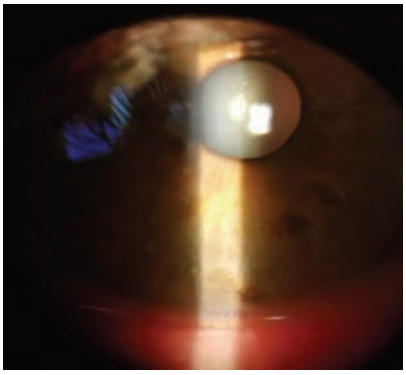

Fig. 1. Multiple small central corneal infiltrates in our patient's right eye.

Our Approach

To sterilize the anterior ocular surface from the adenovirus, several drops of Betadine (povidone-iodine 5%, Purdue Pharma) sterile ophthalmic prep solution were instilled into the eye and he was asked to roll the eye around to provide the Betadine access to all conjunctival areas. Betadine drops were also applied to the lash and lid margins and cleaned with cotton swabs under sanitary gloved conditions. A thorough saline solution lavage followed. One drop of a topical anesthetic, Proparacaine (proparacaine hydrochloride ophthalmic solution 0.5%, Bausch + Lomb) and Acular (ketorolac tromethamine 0.5%, Allergan) were used before and after Betadine rinse for ocular comfort. Following treatment, we instructed the patient to continue with the topical prednisolone acetate ophthalmic suspension every three hours in the right eye for two days and apply topical artificial lubricants every hour or two.

Two days later, he exhibited significant improvement in ocular signs and symptoms (*Figure 2*). The topical steroid was tapered down. The patient remained stable at the five-day follow-up visit with absent ocular signs.

Discussion

Adenoviral infections are common viral conditions that often affect several bodily systems, including the eye. Adenoviruses are non-enveloped, double-stranded DNA genome with multiple serotypes which enter the host nucleus and shed their DNA, resulting in inflammatory response cascades and subsequent infection. The severity and duration of the virus depends on the serotype involved and level of immunocompromise. Upon viral shedding, clinical manifestations become observable for up to 16 days during this incubation period.

The EKC type of ocular adenovirus presentation is highly contagious, and commonly transmits through airborne droplets and hand contact with ocular secretions.[1,2] Because of the non-enveloped genetic structure of adenoviruses, they are often impervious to disinfectants and able to survive for weeks on common fomites such as tables, countertop surfaces, desks,

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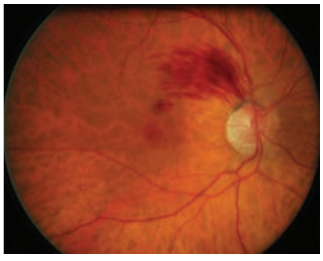

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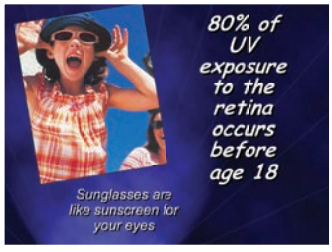

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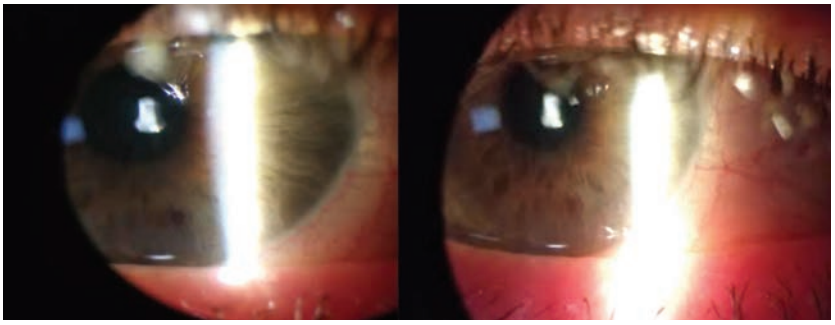

Fig. 2. These images show our patient's significant improvement in ocular signs and symptoms after two days of treatment.

doorknobs and towels. Outbreaks are common in winter and summer months and within daycares, schools, military training facilities and professional medical office settings, including optometry.[1,2]

The presentation of EKC is patient dependent with variable severity, intensity and duration. Mild-to-moderate cases have typical symptoms of conjunctival injection, foreign body sensation, tearing, subepithelial corneal infiltrates and lid edema.

Confirming the diagnosis can be tricky and challenging because ocular symptoms and signs of EKC share similar clinical features as other conditions with allergic, bacterial, inflammatory, contact-related and herpetic etiologies. Therefore, EKC should be a diagnosis of exclusion. An extensive case history and careful ocular evaluation are warranted to rule out other potential masqueraders.[2] AdenoPlus (Quidel) immunoassay is a highly specific and sensitive in office detection method that uses conjunctival scrapings and provides "yes or no" results within approximately 10 minutes confirming the presence of an adenovirus.[3]

Other diagnostic tests include polymerase chain reaction and cell culture combined with confirmatory immunofluorescence; however, these are not readily available.[3]

Therapies

Currently, the FDA has approved no treatments for adenoviruses.[1,2] However, some off-label inexpensive medicine approaches are effective in its management. Because of the highly contagious nature of the condition, hygiene is stressed aggressively to prevent an epidemic. Milder cases respond well to topical artificial tears and cool compresses alone without the intervention of pharmaceutical therapy.[1] Topical corticosteroids are effective in the reduction of corneal epithelial infiltrates and ocular relief; however, they are believed to prolong the viral shedding process.[1,2] Potential side effects of steroids should be assessed, especially if long-term use is required. In cases that do not respond well to corticosteroids or other approaches, topical tacrolimus 0.03% is a safe alternative option.[4] Topical and oral nonsteriodal anti-inflammatory drugs can also aid in ocular pain management and relief.[5]

Several studies show success with Betadine against adenoviruses.[1,6]

Zirgan (ganciclovir 0.15%, Bausch + Lomb) has also revealed promising results against adenoviruses.[7,8] Other available options include cyclosporin A and cidofovir; however, studies did not find significant reductions or alterations in the infection disease process.[9]

Prognosis

Although EKC is a self-limiting and short-lasting condition, some untreated or more severe cases may lead to devastating or permanent effects such as conjunctival or corneal scarring, pseudomembranes, irregular astigmatism, nummular keratitis, periorbital involvement and secondary bacterial infections, all of which become more complex to treat and require more aggressive treatment approaches.[2]

Adenoviruses are common conditions that medical professionals encounter often. While there are no approved treatment protocols, it is important to make patients aware of the effectiveness of off-label, safe, inexpensive therapies which are underused. Once the diagnosis of an acute EKC presents, aim to eliminate the presence of the viral infection by initiating maximal therapy early in the disease course. Delaying the application of these treatment methods may result in an ineffective or minimal therapeutic response. ■

Dr. McGhee practices at Chiasson Eye Center and Bond Wroten Eye Clinic in Louisiana.

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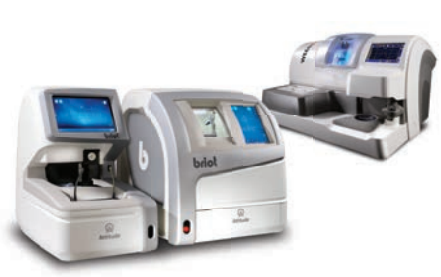

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A Rare Syndrome in Your Chair

Some patients with flu-like symptoms and subretinal inflammation may actually have APMPPE, a disease of the eye with neurological and other systemic manifestations.

By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

During flu season, everyone does their best to steer clear of symptomatic patients. But every once in a while, they need our help. Acute posterior multifocal placoid pigment epitheliopathy (APMPPE), for example, is an immune-mediated chorioretinal disease that usually affects young adults—about 40% of whom report influenza-like symptoms prior to the onset of visual symptoms.[1] While uncommon, this white-dot syndrome may end up in your chair. Here's what you need to know.

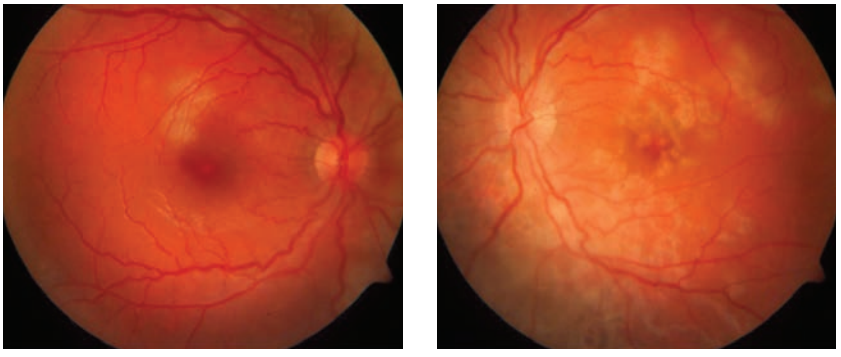

Fig. 1. A 27-year-old female presented one week after a flu-like illness with floaters and blurred vision. Multifocal cream-colored flat placoid lesions were found in the posterior pole of each eye.

Clinical Features and Course

APMPPE usually involves both eyes, and patients typically present with blur, metamorphopsia or scotomas with characteristic fundus findings. The mean age of onset is 27 years and it affects males and females in equal numbers.[2]

Although the exact cause of APMPPE is unknown, researchers suspect a virus may be to blame. For one, the condition can subside without treatment and may recur at any time, similar to other conditions with a viral etiology. Viruses may stay dormant for extended periods of time, then for reasons yet unknown may unexplainably become reactivated.[1,5]

In addition, prior to the onset of APMPPE, patients may present with virus-like symptoms such as nausea, fever, swollen lymph glands and vomiting. Moderate to severe headaches may also occur and, rarely, patients may have neurological signs such as aphasia (temporary loss of speech), limb weakness or both.[1,2] In the early stages of the disease, patients may complain of blotchy scotomata, photopsia, metamorphopsia and photophobia. Later stages are marked by moderate decreases in vision. Less commonly, the impaired vision may be severe.[3]

APMPPE may present with anterior segment findings that include episcleritis, non-granulomatous uveitis and perilimbal anterior stromal corneal infiltrates. The vitreous may have a mild cellular reaction that usually accompanies multifocal yellowish-white flat placoid lesions located mainly in the posterior pole involving the retinal pigment epithelium (RPE) (*Figure 1*).[4] These lesions do not extend beyond the equator and tend to fade over a two-week period, where they are replaced by varying degrees of RPE atrophy and hyperpigmentation.[1,4]

Other findings may include papillitis, retinal periphlebitis, central retinal vein occlusion, optic nerve neovascularization and subhyaloid hemorrhage.

Intravenous fluorescein angiography (IVFA) will show a classic "block early, stain late" pattern.[1-4] The early phase of IVFA will show the acute lesions as hypofluorescent, suggesting nonperfusion or infarction of the RPE, choroid or both (*Figure 2*). The lesions then become hyperfluoresent in the late phase of the study (*Figures 2 and 3*).[1,2]

Optical coherence tomography findings in APMPPE include subretinal fluid with a hyperreflective line anterior to the RPE.[1]

Systemic Concerns

A review of systems is important in cases of suspected APMPPE, as

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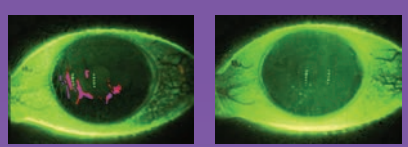

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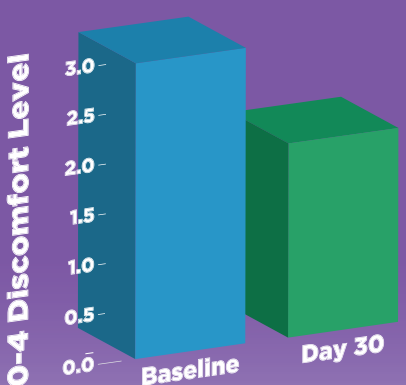

systemic associations of APMPPE may involve the skin (erythema nodosum), kidneys (nephritis with urine casts), muscles, thyroid gland (thyroiditis) and blood vessels (vasculitis).

It has also been associated with multiple complications in the central nervous system (CNS), including cerebral vasculitis, peripheral neuropathy, headaches, aseptic meningitis, meningoencephalitis, sixth cranial nerve palsy, transient hearing loss and cavernous sinus thrombosis.[1,3] Symptoms of severe headache or meningeal symptoms warrant neuroimaging and further neurologic workup. Research has associated fatalities due to cerebral vasculitis with the disease.[1]

APMPPE-like lesions can be present in patients with sarcoidosis, syphilis and tuberculosis; therefore, clinicians should test patients to exclude those conditions.[4]

Differential Diagnosis

The white-dot syndromes are a group of multifocal inflammatory conditions involving both the retina and the choroid. They are characterized by the appearance of white dots in the fundus. Several of these conditions can simulate APMPPE (*Table 1*).

The most similar of the white-dot syndromes to APMPPE is serpiginous choroiditis. The chorioretinal lesions in serpiginous choroiditis are localized to the posterior pole, but produce a more profound choroidal atrophy than in APMPPE. Serpiginous choroiditis also resolves more slowly than APMPPE, and patients have a poorer visual prognosis, with more frequent recurrences of inflammation.[3,4]

Table 1. Differential Diagnosis of APMPPE

Type	Condition
Other white-dot syndromes	• Serpiginous choroiditis • Multifocal choroiditis and panuveitis • Punctate inner choroidopathy • Birdshot chorioretinopathy • Multiple evanescent white-dot syndrome
Infectious conditions	• Syphilis • Tuberculosis • Fungal disease • Toxoplasma retinochoroiditis • Pneumocystis choroiditis • Viral retinitis
Neoplastic disease	• Choroidal metastases • Lymphoma

Management

Most cases of APMPPE resolve within a few weeks. However, in some cases where central macular involvement is profound, visual acuity does not significantly improve.[2]

Because APMPPE is generally self-limiting, there is no rationale for treatment if no neurological complication is encountered. The outcome for the visual system without treatment is characteristically good, and a gradual improvement in visual acuity occurs over weeks to months, with most eyes achieving a visual acuity of 20/30 or better.

If CNS vasculitis is present, systemic corticosteroid treatment is recommended. Steroid therapy may also be indicated for extensive disease that involves the fovea. This may offer a theoretical advantage in shortening the disease course or modifying its effects on central vision.[1,6]

APMPPE is an immune-mediated disease characterized by discrete areas of subretinal inflammation. It may be associated with a number of systemic conditions and thus warrants a detailed systemic workup. Although the disease is self-limiting with a relatively good prognosis, patients with extensive macular involvement may be treated with systemic steroids in an effort to preserve visual acuity to the greatest extent possible. ■

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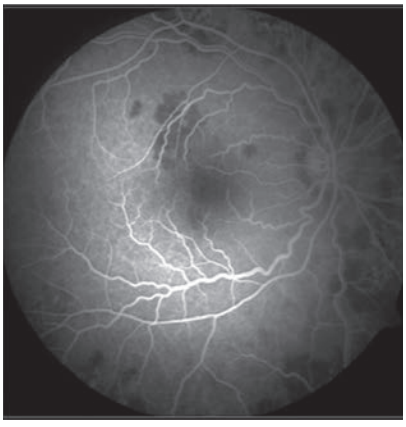

Fig. 2. Early phase of the IVFA shows the acute lesions are hypofluorescent.

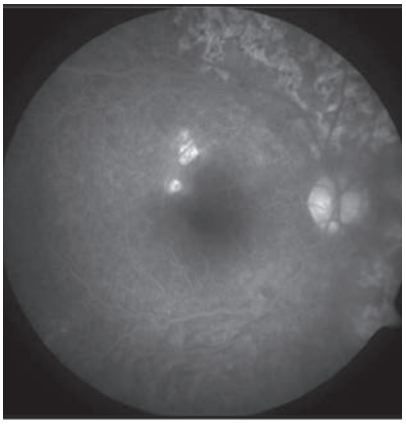

Fig. 3. In the late phase of the study, the lesions become hyperfluorescent.

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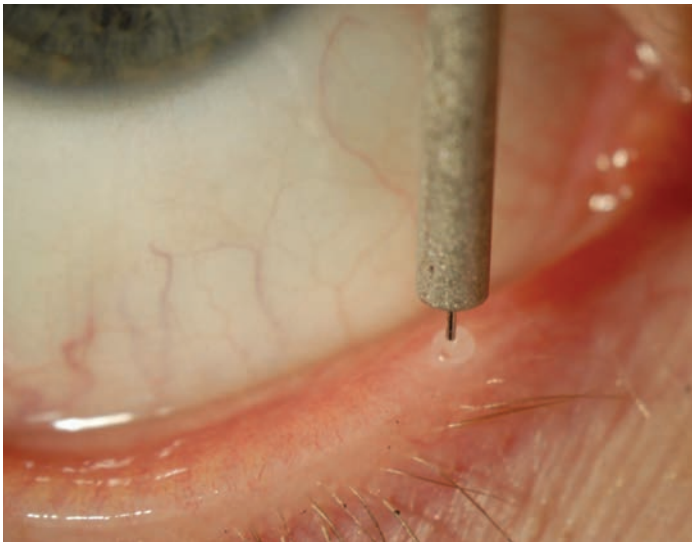
Give Plugs a Chance

Today's punctal occlusion options can serve more patients than ever.

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

In nearly 40 years of implanting punctal plugs for dry eye disease (DED) and other ocular surface disorders, attitudes toward the devices have wavered from extreme popularity to widespread rejection. When we last covered punctal occlusion 10 years ago, the literature's assessment—and our own—was mixed. While we ultimately advocated for the judicious use of punctal plugs, it was predominantly for those patients with moderate to severe DED who did not adequately respond to lubrication therapy and topical immunomodulators, such as Restasis (cyclosporin A 0.05%, Allergan).



Punctal plugs like this one can be used in a broader range of patients today than a decade ago.

We echoed the report of the Dysfunctional Tear Syndrome Study Group, which concluded that "punctal plugs could result in retention of pro-inflammatory tear components on the ocular surface and may enhance damage to the ocular surface, accelerate the disease process, and produce greater patient discomfort," and to "treat the inflammatory condition before blockage of tear drainage with punctal plugs."[1] Moreover, our opinions reflected those of the most comprehensive assessment of DED and management at that time, the report of the Tear Film and Ocular Surface Society's International Dry Eye Workshop (DEWS).[2]

As David Sackett, heralded as the father of evidence-based medicine, once famously said: "Half of what you'll learn in medical school will be shown to be either dead wrong or out of date within five years of your graduation; the trouble is that nobody can tell you which half." While we like to believe that our formed opinions are spot-on and shatter-resistant, time and additional study often prove us to be incorrect. It can be quite a humbling experience to read your own work years later and realize that you flatly disagree with it.

New Findings

In the recent TFOS DEWS II report, authors readdressed the issue of inflammation and punctal occlusion. "The use of punctal occlusion in the presence of ocular surface inflammation is controversial because, theoretically, occlusion of tear outflow could prolong the presence of pro-inflammatory cytokines on the ocular surface," it reads. "However, a recent study shows that punctal occlusion in 29 individuals with moderate DED for three weeks resulted in reduced corneal fluorescein staining and symptom scores, without elevation of cytokine or matrix metalloproteinase-9 levels, questioning whether cytokine levels would necessarily elevate with punctal occlusion over short periods of use."[3]

The referenced study, in which more than 400 tear proteins were analyzed from subjects with moderate DED, also found that some diagnostic features were predictive of greater success with punctal occlusion.[4,5] In general, patients with a lower Schirmer's test score at baseline were found to have a more beneficial tear protein response than patients with higher scores prior to plug insertion.[5]

Another recent study also examined subjective and objective measures before and after punctal plug insertion in 45 patients with

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aqueous-deficient dry eye disease.[6] Researchers found that the introduction of punctal plugs resulted in a statistically significant improvement in: (1) mean symptom score (as measured by the Ocular Surface Disease Index); (2) mean tear volume (as measured via the Schirmer's test); (3) mean fluorescein tear breakup time; (4) pattern of corneal fluorescein staining; (5) corneal sensitivity; and (6) conjunctival impression cytology features.[6] There was also a decrease in the dependence on artificial tear use among subjects.[6] So clearly, newer evidence suggests that punctal occlusion may provide distinct benefit to patients with DED, both with regard to clinical signs and symptoms as well as tear chemistry composition. The Management and Therapy Subcommittee of TFOS DEWS II stipulated that punctal occlusion may be indicated in "any condition that would benefit from aqueous retention on the ocular surface" (*Table 1*).[3]

Table 1. TFOS DEWS II Indications for Punctal Occlusion Therapy[3]

- Symptomatic contact lens wear
- Dry eye related to refractive surgery
- Aqueous-deficient dry eye secondary to systemic disease (e.g., Sjögren's syndrome)
- Dry eye associated with a rapid tear break-up time
- Systemic medications that reduce tear production (e.g., antihistamines, antidepressants)
- Lid palsy or lid closure abnormalities
- Superior limbic keratoconjunctivitis
- Any corneal irregularities or scarring that affects tear stability
- Toxic epitheliopathy

Upgraded Gear

One aspect of punctal occlusion that was really not addressed thoroughly in the DEWS II report was that of plug design and material, particularly with regard to absorbable intracanalicular plugs. The earliest absorbable or "temporary" plugs were composed exclusively of collagen. These can last anywhere from two to 10 days after implantation, depending on the size of the plug and the individual patient.[7] Such plugs are ideal for managing temporary surface issues and are useful diagnostically as they help determine whether punctal occlusion and long-term plugs will be well-tolerated.

Long-term absorbable plugs are composed of synthetic polymers that dissolve more slowly than collagen, lasting from two to six months. Materials used in the manufacture of these plugs include glycolic acid/ trimethylene carbonate copolymer, PCL (Δ-caprolactone/L-lactide copolymer) and polydioxanone.[7] While many of these products are commercially available in the United States, the peer-reviewed literature is all but devoid of research involving these devices. The primary advantage of this design appears to be the complete lack of cap irritation that may be experienced with silicone punctal plugs. Additionally, since the material dissolves completely over several months, the presumed likelihood of secondary infection and inflammation is low.

Another aspect of punctal occlusion that is often overlooked is the use of perforated punctal plugs, sometimes referred to as "partial occluders" or "flow controllers." Their design includes an open inner channel that's 0.2mm to 0.3mm narrower than the shaft width of the plug.[8] The primary indication for use of a perforated plug is epiphora, whether encountered after insertion of a conventional plug or associated with acquired punctal stenosis.

Clinical studies show that the use of these devices eliminates epiphora in 84% of patients.[9-11] Factors that may limit success include the presence of unmanaged blepharitis and increased patient age, which may be associated with more severe horizontal lid laxity and poor lid-globe apposition.[10]

Practicing optometrists may still be reluctant to employ punctal plugs for patients with ocular surface disease, except in severe cases where all other treatments have failed. However, our current understanding indicates that this philosophy is outdated. Along with many new and exciting therapies that we have in 2018, tear conservation remains an important step in managing our dry eye patients. Punctal plugs deserve a second chance. ■

Disclosure: Dr. Kabat is a consultant/advisor for Lacrivera and Ocusoft.

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Looking Through the Cracks

A patient's fundus shows signs of an underlying disease.

By Eric Dillinger, OD, and Mark T. Dunbar, OD

A 58-year-old Hispanic female presented to the emergency room with a chief complaint of a red right eye and associated foreign body sensation beginning one day earlier. She denied any recent illness, allergies or pain.

Her medical history was significant for high cholesterol, for which she takes simvastatin 20mg and a multivitamin. She's also smoked half a pack of cigarettes per day for 30 years, and her mother has wet age-related macular degeneration bilaterally.

On examination, her vision acuity, with habitual correction, was 20/40-2 OD and 20/100+1 OS with no improvement with pinhole. Her confrontation visual fields were full-to-careful finger counting, and motility testing was normal. Intraocular pressure (IOP) was measured at 14mm Hg OD, 15mm Hg OS via Tonopen (Reichert). Her pupils were round and reactive to light with no afferent pupillary defect. The anterior segment exam was positive for 1+ follicles and papillae. There was no preauricular node and her corneas were clear.

A dilated fundus exam shows changes (*Figure 1*). An OCT was also obtained and is available for review (*Figure 2*).

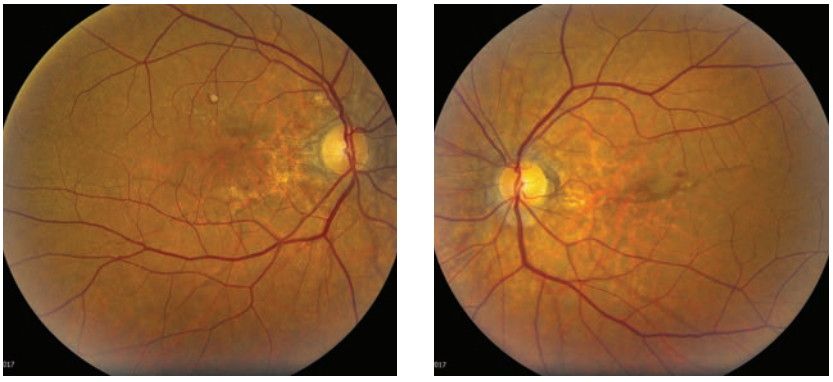

Fig. 1. Although our patient's initial complaint was a red eye, can you identify any other pathology based on her fundus photographs?

Take the Retina Quiz

1. What do the fundus changes in the posterior pole of both eyes represent?
a. Laquer cracks.
b. Choroidal rupture.
c. Drusen and geographic atrophy.
d. Angioid streaks.

2. What best describes the fundus photo and OCT of the left eye?
a. Subretinal fluid and hemorrhage due to choroidal neovascularization.
b. Intraretinal fluid and outer retinal atrophy due to AMD.
c. Atrophy and subretinal fluid associated with pathological myopia.
d. Choroidal rupture through the macula with associated subretinal fluid.

3. What is the correct diagnosis for this patient?
a. Ehler-Danlos syndromes.
b. Pseudoxanthoma elasticum.
c. Pathologic myopia.
d. AMD with choroidal neovascularization.

4. How should she be treated?
a. Observation.
b. Refer for intravitreal injection.
c. Refer for cardiac workup.
d. Refer for genetic testing.

For answers, see page 106.

Diagnosis

Our patient was diagnosed with mild viral conjunctivitis and recommended to use artificial tears pallitively as needed. Of particular interest were the changes seen in the macula and posterior pole of both eyes.

We saw obvious peripapillary atrophy surrounding each optic nerve as well as subtle radial cracks or defects in Bruch's membrane extending linearly from the optic nerve in various directions. These cracks or defects in Bruch's membrane represent angioid streaks. The streaks extend into the macula in both eyes. What's more, subretinal hemorrhages were seen in both maculas, with obvious elevation and subretinal fluid in the left eye. Despite the hemorrhage in the right eye, the macula appeared flat.

Based on these changes, we were certain our patient had developed

choroidal neovascularization (CNV) in both eyes as a result of the angioid streaks. An OCT confirmed an active CNV in the left eye with subretinal fluid. The macula in the right eye was flat with disruption of the inner-outer segment junction and minimal cystic changes. On further questioning, our patient reported being followed by a retinal specialist in the community for CNV in both eyes and had multiple intravitreal injections in both eyes to control the bleeding.

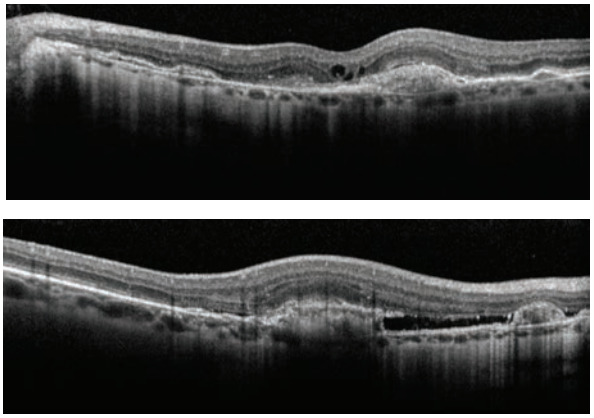

Fig. 2. Can you identify our patient's diagnosis based on the OCT images of our 58-year-old patient's right (at top) and left eyes?

Discussion

Angioid streaks are typically bilateral and rarely seen outside of the posterior pole. They emanate from the optic nerve radially and taper as they get further from the optic disc. The angioid streaks appear as crack-like breaks in Bruch's membrane and retinal pigment epithelium (RPE) atrophy. They can have varying colors depending on the tint of the fundus. In patients with a lighter fundus, the streaks will appear red, while those with a darker fundus will show medium to dark brown streaks.[1,2]

Angioid streaks often have a systemic association.[3] The most common are grouped into the mnemonic PEPSI:

- ***P***seudoxanthoma elasticum (PXE)
- ***E***hler-Danlos syndromes
- ***P***aget's disease of the bone
- ***S***ickle cell disease and other hemoglobinopathies
- ***I***diopathic causes

Interestingly, angioid streaks do have as high as 25% association with optic disc drusen.[3]

Systemically, many of these conditions associated with angioid streaks can be life-threatening. Pseudoxanthoma elasticum and Ehler-Danlos can have cardiac complications, whereas sickle cell anemia can cause organ failure or stroke due to clotting.[2] Because of the seriousness of these conditions, it is important to recognize the clinical findings and refer these patients as indicated.

The most common systemic association with angioid streaks is PXE, which is a hereditary disorder that affects the elastic tissues of the skin, eyes and blood vessels. Pseudoxanthoma elasticum usually affects the skin first, resulting in a classic "plucked-chicken" skin appearance on the lateral side of the neck, axillae and flexure creases. Eighty percent of clinical cases show mutations in the ABCC6 gene leading to abnormal MRP6 protein, which allows for genetic testing confirmation, if warranted.[2] Patients with PXE often have a characteristic *peau d'orange*, or "orange peel" appearance to the retina and RPE.[2] This dimpling of the RPE can be seen on the fundus photographs temporally in the right eye of our patient.

Ehler-Danlos syndromes are a hereditary group of connective tissue disorders that result in hypermobility, skin hyperextensibility and tissue fragility. Individuals with Ehler-Danlos syndromes can develop several ocular complications such as keratoconus, high myopia, retinal detachment, lens dislocation and angioid streaks.

Angioid streaks are typically asymptomatic, unless they involve the macula. Severe visual impairment is seen in 70% of cases with angioid streaks due to CNV.[2] Researchers estimate that CNV occurs in 72% to 86% of all patients with angioid streaks, and becomes bilateral 50% of the time within 18 months of the initial CNV presentation.[1,3]

OCT and fluorescein angiography are excellent ancillary tests to help assess the stability of the outer retina and any development of CNV as well as identify any breaks in Bruch's membrane or RPE abnormalities.

Upon further questioning, our patient revealed that she was diagnosed with PXE many years ago. The patient was initiated on a treatment plan of intravitreal injections of Avastin (bevacizumab, Genentech) in both eyes with re-assessment every four weeks with OCT to monitor the subretinal fluid and hemorrhages. ■

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

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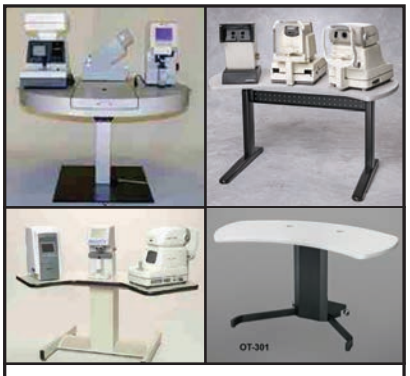

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Diagnostic **Quiz**

Growing Pains

By Andrew S. Gurwood, OD

History

A 55-year-old Caucasian male presented to our office urgently with a chief complaint of a foreign body sensation in his right eye for a duration of two hours.

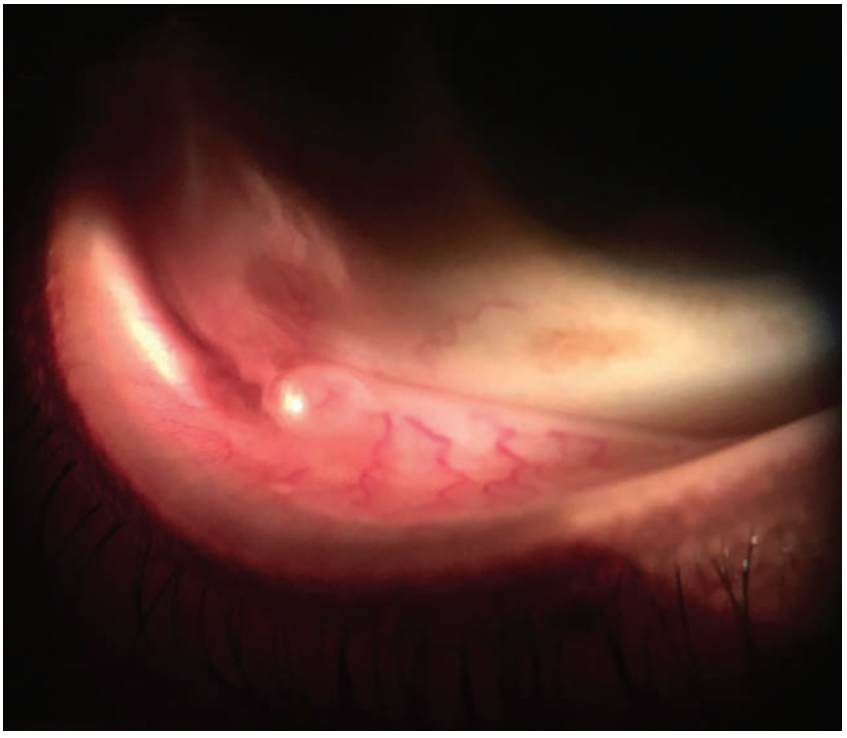
The patient explained that, before calling the office, he tried cold compresses and artificial tears, but could not find relief. He denied toxic exposure, trauma of any kind and any allergies.

His history was positive for systemic hypertension and hypercholesterolemia, for which he was medicated and compliant.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OU at distance and near. His external examination was normal, although there seemed to be mild excessive lacrimation in the left eye without conjunctival injection.

We observed no evidence of afferent pupil defect. The pertinent biomicroscopic examination of the anterior segment is demonstrated in the photograph. Goldmann applanation tonometry measured 15mm Hg OU. The dilated fundus findings were normal peripherally and centrally with normal nerves and maculae.



A 55-year-old patient presented urgently with a foreign body sensation in his right eye. Can this image help explain his issue?

Your Diagnosis

Does the case presented require any additional tests, history or information? What steps would you take to manage this patient? Based on the information provided, what would be your diagnosis? What is the most likely prognosis? To find the answers, please visit us online at www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 97):* 1) d; 2) b; 3) b; 4) b.

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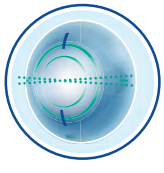

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Moisturizing agents are gradually released throughout the day for initial and all-day comfort.[2,4,5]

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References: **1.** Alcon data on file, 2012. **2.** Wolffsohn JS, Hunt OA, Chowdhury A. Objective clinical performance of 'comfort-enhanced' daily disposable soft contact lenses. *Contact Lens & Anterior Eye.* 2010;33(2):88-92. **3.** Laboratory study release profile; Alcon data on file, 2007. **4.** Winterton L, Lally J, Sentell K, Chapoy L. The elution of poly (vinyl alcohol) from a contact lens: The realization of a time release moisturizing agent/artificial tear. *J Biomed Mater Res B Appl Biomater.* 2007;80B:424-32. **5.** Laboratory study release profile; Alcon data on file, 2007.

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

Alcon A Novartis Division