Review of Systems: Managing Migraine Headache, **PAGE 98**

REVIEW® OF OPTOMETRY

September 15, 2017

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

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VOL. 154 NO. 9 ■ SEPTEMBER 15, 2017

IN THE NEWS

More than **85% of adolescent (ages 12 to 17) contact lens wearers have at least one habit that increases their chances of an eye infection**, according to a recent CDC Morbidity and Mortality Weekly Report. The most common risky habits in this age group were not visiting an eye doctor annually and wearing contact lenses while sleeping, napping or swimming. Patients ages 18 and older also reported ignoring proper lens and lens case replacement schedules.

A new study confirms the **additive effect of waiting five minutes between drop instillation** for dilation drops. Although clinicians recommend patients wait five minutes between instilling different drops, the only human study to explore concurrent drop instillation found a 10-minute wait did not increase the combined effect. The results of this new study show **waiting five minutes yielded a 5.6% relative pupil surface gain** when instilling one drop of 10% phenylephrine and one drop of 0.5% tropicamide.

Denion E, Charlot F, Béraud G. A 5-minute interval between two dilating eye drops increases their effect. Optom Vis Sci. 2017;94(8):838–44.

Contact lenses coated with the mucin MUC5AC extracted from the stomachs of pigs **no longer cause tissue damage** to porcine eyes, according to a new study. Researchers found the mucin absorbs into the contact lenses, creating a lubricating barrier between the contact lens and the cornea. They speculate lubricating drops or soaking contact lenses overnight in a mucin solution will create the protective effects.

Winkeljann B, Boettcher K, Balzer BN, Lieleg O. Mucin coatings prevent tissue damage at the cornea-contact lens interface. Advanced Materials Interfaces. 2017;1700186. [Epub].

In the Pipeline: Line-field OCT

This new modality may soon provide clinicians aberration-free retinal images at the cellular level.

By Rebecca Hepp, Managing Editor

A newly developed optical coherence tomography (OCT) technique, line-field (LF) OCT, may simplify how clinicians image ocular cellular processes, according to new research.[1]

The new technique uses linear illumination with rapid frame rates to allow digital correct of aberrations over the entire 3D volume of the retina, according to lead author Laurin Ginner, a PhD student studying under Rainer Leitgeb, PhD.[2] The result is *in vivo*, aberration-free retinal images at the cellular level without expensive hardware adaptive optics (AO).[1,2] While researchers already know combining AO with OCT can provide high isotropic resolution in 3D, "complexities of adaptive optics together with its costs make the commercialization of such technology difficult," the study says.[1]

"Line field-OCT holds substantial promise in the diagnosis and management of retinal disease," says Carolyn Majcher, OD, assistant clinical professor at the Rosenberg School of Optometry. "The cellular resolution provided by line-field OCT is far superior to conventional OCT such that even individual photoreceptors, capillaries and nerve fibers can be distinguished and evaluated.[2] Visualizing fine cellular details permits imaging of cellular physiologic and metabolic processes such as photoreceptor disc shedding and axonal transport."[1,3]

The researchers are also able to refocus, realign and digitally process the image data to help provide

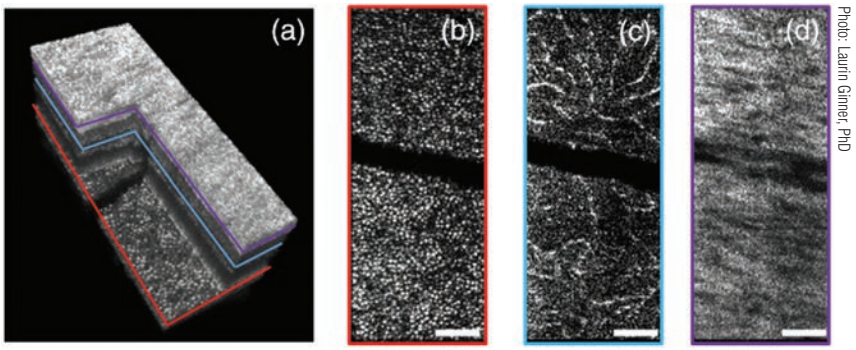

Photo: Laurin Ginner, PhD

This 3D volume of the retina is corrected for defocus and higher order of phase error (a). The photoreceptor layer is red (b), the outer plexiform layer is blue (c) and the nerve fiber layer is violet (d). The white scale bar denotes 100µm.[1]

the best results for proper diagnosis and treatment management.[1]

"This sort of 'functional OCT imaging' of the retina will aid clinicians in earlier detection of retinal and optic nerve diseases," says Dr. Majcher.[1] "Instead of waiting for cells to die off so that conventional thinning of the retina and nerve fiber layer can be detected, this technology will likely have the ability to detect pre-death cellular dysfunction. Prompt disease diagnosis and intervention before irreversible cell death occurs will result in greater visual preservation."

The researchers further applied the correction technique to OCT-angiography and found it improved vessel definition blurred by motion artifacts and defocus.[1]

"This will prove useful in the diagnosis of retinal vascular diseases and neovascular disorders such as diabetic retinopathy, retinal vein occlusion, age-related macular degeneration, and many more," says Dr. Majcher.

"The eye is the 'window' into the brain. Our hope is that the higher resolution will help to improve diagnostic accuracy in general," said Dr. Leitgeb in a press release.[2]

1. Ginner L, Kumar A, Fechtig D, et al. Noniterative digital aberration correction for cellular resolution retinal optical coherence tomography in vivo. Optica. 2017;4(8):924-31.
2. Medical University of Vienna. New OCT technique provides better 3-D imaging of the cellular structure of the eye. ScienceDaily. www.sciencedaily.com/releases/2017/08/170807082203.htm. Accessed August 8, 2017.
3. Kocaoglu O, Liu Z, Zhang F, et al. Photoreceptor disc shedding in the living human eye. Biomed Opt Express. 2016;7(11):4554-68.

Should Patients Laser Away Floaters?

Patients who experience reduced visual quality due to floaters may have a treatment option, YAG vitreolysis, according to new research.[1]

"It is an interesting and exciting area to think about because we all have numerous patients that have floaters," says Nate Lighthizer, OD, associate professor and chief of Specialty Care Clinics at the Oklahoma College of Optometry. "We see these patients on a day-in-and-day-out basis and often it's been frustrating because we historically haven't been able to give them many great treatment options."

Researchers randomly assigned 52 patients to either YAG laser vitreolysis or a sham laser treatment. Six months post-procedure, 54% of the YAG group reported an improvement in visual disturbances compared with only 9% of controls. In addition, 53% of the YAG group reported significant or even complete resolution of symptoms.[1]

The researchers also found the YAG group reported improved general vision, peripheral vision, role difficulties and dependency compared with pre-procedure.[1]

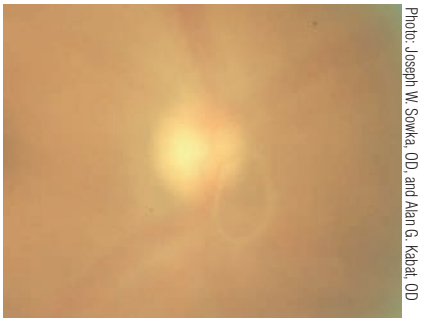

Photo: Joseph W. Sowka, OD, and Alan G. Kabat, OD

YAG vitreolysis may be an effective treatment option for a Weiss ring, but some worry about adverse effects.

"This study is one more step to provide more evidence that it's a safe, effective procedure, and patients show reduction in their symptoms, which is ultimately what we want for these symptomatic and often frustrated patients," says Dr. Lighthizer. "It is nice to possibly have a treatment option in between observation and vitrectomy surgery, and one that is better than doing nothing and much less invasive than the higher risk surgical option."

One reason for such positive study results is the careful patient selection process, said Jennifer Lim, MD, in a commentary.[2] The researchers carefully screened for patients with symptomatic Weiss rings of at least six month's duration and at least 3mm distant from the retina and 5mm from the posterior lens capsule.[2] In addition, the procedure used energies just high enough for photodisruption of the Weiss ring. Together, these measures mitigated complications, Dr. Lim said.[2]

While promising, the results don't necessarily confirm YAG vitreolysis is safe.[2] Dr. Lim recommends opacities close to the lens and underlying vital structures not be treated with the YAG laser—at least until more studies can confirm these initial results.[1,2]

"The clinical data and scientific studies are telling us that we need to pick our patients appropriately," adds Dr. Lighthizer. "Not all types of floaters will do equally well with this procedure, either. So we still have a lot to learn clinically about the procedure, but it is very exciting to finally have a treatment to offer our patients other than high-risk, invasive surgery."

1. Shah CP, Heier JS. YAG Laser vitreolysis vs sham YAG vitreolysis for symptomatic vitreous floaters. JAMA Ophthalmology. July 20, 2017. [Epub].
2. Lim JI. YAG laser vitreolysis—is it as clear as it seems? JAMA Ophthalmol. July 20, 2017. [Epub].

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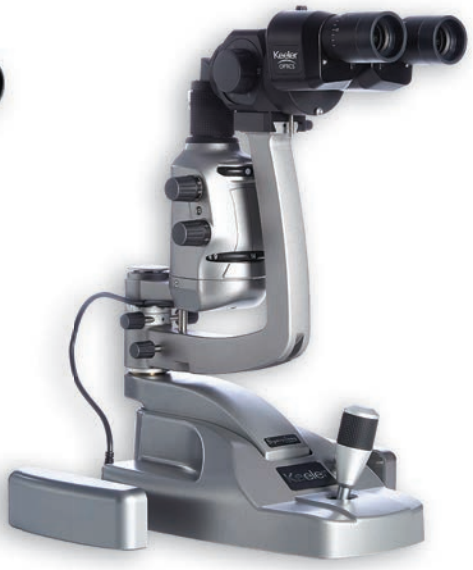

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Eye Docs Most Likely to Prescribe Branded Meds

Eye care practitioners prescribe more brand medications by volume than any other provider group, according to a recent analysis. In total, researchers found $2.4 billion in annual Medicare Part D prescription costs by optometrists and ophthalmologists in 2013, and the prescribers turned to branded medications 79% of the time.[1] Glaucoma medications made up half of the ophthalmic drugs prescribed, at a cost of $1.2 billion, with dry eye medications coming in second, accounting for $371 million.[1,2] These two categories, in addition to ocular inflammation and infection medications, made up 96% of drugs prescribed.[1]

The researchers conclude a switch to generics could reduce costs by $882 million a year.[1] However, allowing Medicare to negotiate prices is the only long-term solution to high costs, according to the study authors. "If the cost of generic medications increase, such as what occurred in 2014 when the price of generic prednisolone acetate and generic phenylephrine soared, changing providers' prescription patterns would not help to reduce costs," says senior author Maria Woodward, MD.[1]

In on the Fight

While policy change is one solution to lower costs, Alan Kabat, OD, professor at Southern College of Optometry, says another option is keeping the doctor in charge.

"Optometrists are already under immense pressure to prescribe generic medications from much of their patient base due to cost concerns, and also from most third-party insurers, both governmental and commercial," says Dr. Kabat. Even when branded medications are covered, they are typically in a higher tier, he says, requiring greater copay for the beneficiary. "Alternatively, insurance companies may require a prior authorization for a branded medication, with the doctor affirming that the patient has tried and failed with alternative treatment options, generics or legacy drugs."

If health care were merely a dollars-and-cents matter, this would be an easy decision, says Dr. Kabat. But it is far more complex. "Physicians are trained to provide the best treatment option in any scenario. The drug or procedure that offers, for example, the greatest efficacy, the easiest dosing regimen or the least potential for complications is often not the least expensive. In effect, the current approach forces practitioners to choose between what is best and what is cheapest."

"Lawmakers must recognize that physicians are the ultimate authority for determining which treatment option is most appropriate for each patient."

In the end, he says, negotiating for better drug pricing directly with companies as a means to lower Medicare costs would be much more palatable to all parties involved than mandating which medications a doctor can prescribe.

1. University of Michigan. A switch to generic eye drugs could save Medicare millions. www.uofmhealth.org/news/archive/201707/switch-generic-eye-drugs-could-save-medicare-millions. Accessed August 18, 2017.
2. Newman-Casey PA, Woodward MA, Niziol LM, et al. Brand medications and medicare part D. Ophthalmology. 2017 July. [Epub ahead of print].

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INDICATIONS AND USAGE

ZYLET® (loteprednol etabonate 0.5% and tobramycin 0.3% ophthalmic suspension) is a topical anti-infective and corticosteroid combination for steroid-responsive inflammatory ocular conditions for which a corticosteroid is indicated and where superficial bacterial ocular infection or a risk of bacterial ocular infection exists.

Ocular steroids are indicated in inflammatory conditions of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe such as allergic conjunctivitis, acne rosacea, superficial punctate keratitis, herpes zoster keratitis, iritis, cyclitis, and where the inherent risk of steroid use in certain infective conjunctivitides is accepted to obtain a diminution in edema and inflammation. They are also indicated in chronic anterior uveitis and corneal injury from chemical, radiation or thermal burns, or penetration of foreign bodies.

The use of a combination drug with an anti-infective component is indicated where the risk of superficial ocular infection is high or where there is an expectation that potentially dangerous numbers of bacteria will be present in the eye.

The particular anti-infective drug in this product (tobramycin) is active against the following common bacterial eye pathogens: Staphylococci, including *S. aureus* and *S. epidermidis* (coagulase-positive and coagulase-negative), including penicillin-resistant strains. Streptococci, including some of the Group A-beta-hemolytic species, some nonhemolytic species, and some *Streptococcus pneumoniae, Pseudomonas aeruginosa, Escherichia coli, Klebsiella pneumoniae, Enterobacter aerogenes, Proteus mirabilis, Morganella morganii,* most *Proteus vulgaris* strains, *Haemophilus influenzae,* and *H. aegyptius, Moraxella lacunata, Acinetobacter calcoaceticus* and some *Neisseria* species.

IMPORTANT SAFETY INFORMATION

- ZYLET® 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 infections of the eye and fungal diseases of ocular structures.

IMPORTANT SAFETY INFORMATION (continued)

- Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, and 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.
- Use of corticosteroids may result in posterior subcapsular cataract formation.
- 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 a slit lamp biomicroscopy and, where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infections. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.
- Employment of 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.
- Most common adverse reactions reported in patients were injection and superficial punctate keratitis, increased intraocular pressure, and burning and stinging upon instillation.

Please see Brief Summary of full Prescribing Information for ZYLET® on adjacent page.

BAUSCH+LOMB

Zylet®
loteprednol etabonate 0.5% and
tobramycin 0.3% ophthalmic suspension

BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use Zylet safely and effectively. See full prescribing information for Zylet.

Zylet® (loteprednol etabonate 0.5% and tobramycin 0.3% ophthalmic suspension)

Initial U.S. Approval: 2004

DOSAGE AND ADMINISTRATION

2.1 Recommended Dosing

Apply one or two drops of Zylet into the conjunctival sac of the affected eye every four to six hours. During the initial 24 to 48 hours, the dosing may be increased, to every one to two hours. Frequency should be decreased gradually as warranted by improvement in clinical signs. Care should be taken not to discontinue therapy prematurely.

2.2 Prescription Guideline

Not more than 20 mL should be prescribed initially and the prescription should not be refilled without further evaluation [*see Warnings and Precautions (5.3)*].

CONTRAINDICATIONS

4.1 Nonbacterial Etiology

Zylet, as with other steroid anti-infective ophthalmic combination drugs, 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

5.1 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.

5.2 Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

5.3 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 a slit lamp biomicroscopy and, where appropriate, fluorescein staining.

5.4 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. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.

5.5 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).

5.6 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.

5.7 Aminoglycoside Hypersensitivity

Sensitivity to topically applied aminoglycosides may occur in some patients. If hypersensitivity develops with this product, discontinue use and institute appropriate therapy.

ADVERSE REACTIONS

Adverse reactions have occurred with steroid/anti-infective combination drugs which can be attributed to the steroid component, the anti-infective component, or the combination.

Zylet:

In a 42 day safety study comparing Zylet to placebo, ocular adverse reactions included injection (approximately 20%) and superficial punctate keratitis (approximately 15%). Increased intraocular pressure was reported in 10% (Zylet) and 4% (placebo) of subjects. Nine percent (9%) of Zylet subjects reported burning and stinging upon instillation.

Ocular reactions reported with an incidence less than 4% include vision disorders, discharge, itching, lacrimation disorder, photophobia, corneal deposits, ocular discomfort, eyelid disorder, and other unspecified eye disorders.

The incidence of non-ocular reactions reported in approximately 14% of subjects was headache; all other non-ocular reactions had an incidence of less than 5%.

Loteprednol etabonate ophthalmic suspension 0.2% - 0.5%:

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.

In a summation of controlled, randomized studies of individuals treated for 28 days or longer with loteprednol etabonate, the incidence of significant elevation of intraocular pressure (≥10 mm Hg) was 2% (15/901) among patients receiving loteprednol etabonate, 7% (11/164) among patients receiving 1% prednisolone acetate and 0.5% (3/583) among patients receiving placebo.

Tobramycin ophthalmic solution 0.3%:

The most frequent adverse reactions to topical tobramycin are hypersensitivity and localized ocular toxicity, including lid itching and swelling and conjunctival erythema. These reactions occur in less than 4% of patients. Similar reactions may occur with the topical use of other aminoglycoside antibiotics.

Secondary Infection:

The development of secondary infection has occurred after use of combinations containing steroids and antimicrobials. Fungal infections of the cornea are particularly prone to develop coincidentally with long-term applications of steroids.

The possibility of fungal invasion must be considered in any persistent corneal ulceration where steroid treatment has been used.

Secondary bacterial ocular infection following suppression of host responses also occurs.

USE IN SPECIFIC POPULATIONS

8.1 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 fixtures) 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 at 0.5 mg/kg/day (6 times the maximum daily 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.

Reproductive studies have been performed in rats and rabbits with tobramycin at doses up to 100 mg/kg/day parenterally and have revealed no evidence of impaired fertility or harm to the fetus. There are no adequate and well controlled studies in pregnant women. Zylet should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

8.3 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 that appear in human milk could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. Caution should be exercised when Zylet is administered to a nursing woman.

8.4 Pediatric Use

Two trials were conducted to evaluate the safety and efficacy of Zylet® (loteprednol etabonate and tobramycin ophthalmic suspension) in pediatric subjects age zero to six years; one was in subjects with lid inflammation and the other was in subjects with blepharoconjunctivitis.

In the lid inflammation trial, Zylet with warm compresses did not demonstrate efficacy compared to vehicle with warm compresses. Patients received warm compress lid treatment plus Zylet or vehicle for 14 days. The majority of patients in both treatment groups showed reduced lid inflammation.

In the blepharoconjunctivitis trial, Zylet did not demonstrate efficacy compared to vehicle, loteprednol etabonate ophthalmic suspension, or tobramycin ophthalmic solution. There was no difference between treatment groups in mean change from baseline blepharoconjunctivitis score at Day 15.

There were no differences in safety assessments between the treatment groups in either trial.

8.5 Geriatric Use

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

NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate or tobramycin.

Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma TK assay, a chromosome aberration test in human lymphocytes, or in an *in vivo* mouse micronucleus assay.

Oral treatment of male and female rats at 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (500 and 250 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender. No impairment of fertility was noted in studies of subcutaneous tobramycin in rats at 100 mg/kg/day (1700 times the maximum daily clinical dose).

PATIENT COUNSELING INFORMATION

This product is sterile when packaged. Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the suspension. If pain develops, redness, itching or inflammation becomes aggravated, the patient should be advised to consult a physician. As with all ophthalmic preparations containing benzalkonium chloride, patients should be advised not to wear soft contact lenses when using Zylet.

MANUFACTURER INFORMATION

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

Based on 9007706-9004406 Revised 08/2016 ZYL.0053.USA.16

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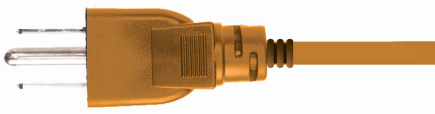

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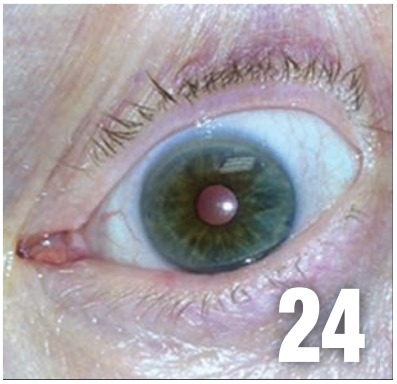

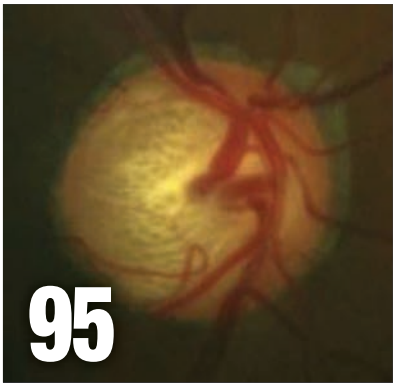

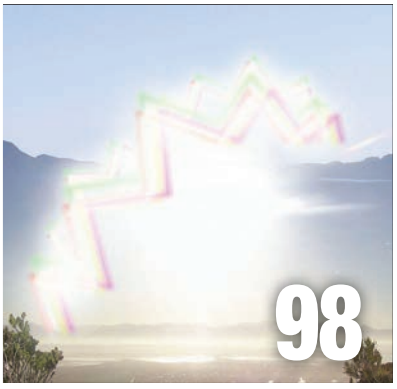

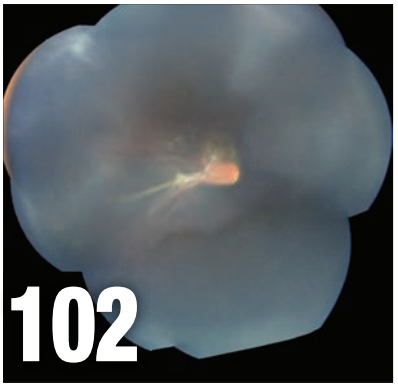

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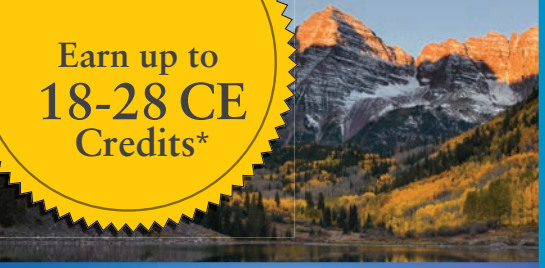

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Outlook

By Jack Persico, Editor-in-Chief

Rise of the Machines

Four decades on, optometry still hasn't bested its fears of automation. And that's a good thing.

If you practiced optometry in March 1978, when our first technology issue appeared, your exam room might have seemed as empty as a racquetball court compared to today. Then, optometrists used a few simple tools—most of them operated manually—that required expertise and careful consideration by the doctor. Now, gadgets are so pervasive that many have long since spilled out of the exam room into the pretest area, with use delegated to a tech.

Is there any room left, in your budget and in your brain, for more? This 40th installment of our series highlights technical advances spanning everything from the most fundamental areas, like refraction, to specialized tools such as dark adaptometry, corneal hysteresis, ERG and all manner of OCT. The latter category moves quickly—no sooner had we prepared a CE article on cutting-edge OCT technology than news broke about *another* iteration, called line-field OCT, which may allow imaging at the cellular level. Don't miss our news story on page 3 about that.

If you're leery of such tech-heavy care, you're not alone. Anxiety about the relationship between doctors, patients and devices has been present for decades. The cover of the March 1978 issue was an illustration of an OD cowering beneath a huge, intimidating computer under the headline, "Automation: Will it Click With Today's OD?" Inside, a reader survey on attitudes toward new technology opened with this provocative sentence: "It killed the cobbler and the weaver, and some ODs think they may be next." *It*, of course, was automation. "The idea that patients may some day be able to click, buzz and whirr their way into an accurate prescription at their local department store worries many ODs," the author continued, expressing a fear that still pervades optometry, as online refraction seems potentially able to deliver what the department store couldn't.

The tech boogeyman of '78 was the autorefractor, a recent introduction at the time. Opinions were decidedly mixed. Just 44% of readers favored automating such a core responsibility. While 61% expected it to improve efficiency, only 34% said it would improve quality of care. In the article, ODs worried about the erosion of the doctor-patient relationship ("The patient feels rushed; he feels the doctor doesn't care because he turns him over to an assistant who doesn't care either"), overreliance on devices ("I'm afraid of the quickie exam, where there is no case analysis of a patient's needs") and accuracy ("When you can prescribe the results obtained by an automated refractor, maybe I'll consider one. Otherwise, they are very expensive retinoscopes.").

Many readers of that first technology issue could conceivably have left their office and found a theater showing *Star Wars*, released 10 months prior but still a smash hit. It became a cultural event because it tapped into our man-vs.-machine dread, letting us exorcise our demons as we watched an intrepid human take down a technological terror. Four decades later, it's a story we still need to hear: technology alone is never enough. We at RO love tech, but keep that in mind as you read this month's coverage. ■

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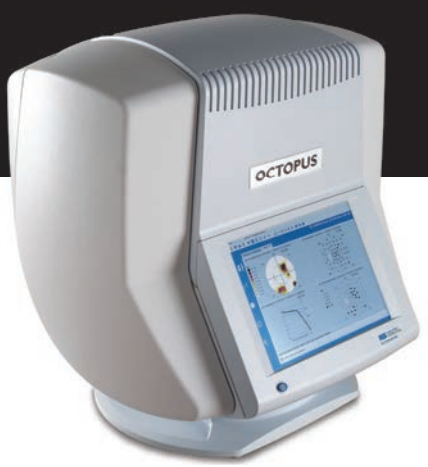

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Through **My Eyes**

By Paul M. Karpecki, OD, Chief Clinical Editor

Crank Up Your Clinic

It may be time to upgrade your tools to help you stay ahead of online competition—and ocular disease progression.

Last month, I observed a significant peripheral malignant melanoma in a patient who presented without complaints and 20/20 vision. No online testing or online optical using dated prescriptions would have caught that—and the consequences could have been life-threatening. But what *did* help me catch that was the technology I have at my fingertips these days. While we are all leery of innovations such as online refraction and dispensing services, some advances do more good than harm when used properly. Here is a look at the technological advances transforming your office.

Handheld Instruments

Technology is getting smaller and more portable. One of the more extreme examples is the recently introduced handheld full-field flash ERG + VEP (RETeval), which fits in the palm of your hand and holds its charge for at least 70 patient exams, according to the company. In the near future, the Eyekinetix (Konan Medical) may help you measure a relative afferent pupillary defect in less than 30 seconds with accuracy far beyond what's possible with a swinging penlight.

Refraction

Automated systems are beginning to stack up. I recently conducted a study evaluating the Perfectus (VMax) refracting system, which uses point spread function (PSF). Preliminary results suggest the device is as accurate as a doctor's refraction in 97% of cases—and even more with difficult refractions such as in patients with keratoconus. These technologies have changed how patients view the optometric refraction by providing increased efficiency, accuracy, EHR compatibility and innovations such as wavefront and PSF.

Early Diagnoses

We all know the sooner a diagnosis is made, the better the prognosis. We can now detect diseases much earlier than ever before with the help of newer technology.

Age-related macular degeneration (AMD). Dark adaptation has over 90% sensitivity and specificity for AMD. It's not merely a predictor; if you fail this six-minute test, you have AMD. In addition, the Pharmanex Biophotonic scanner (NuSkin) is designed to help you accurately measure the level of carotenoids or antioxidants in patients' soft tissue, such as the palm of their hand.

Glaucoma. New technologies are modernizing your glaucoma examination, including OCT, progression analysis and corneal hysteresis measurements. Longitudinal studies show that patients with a corneal hysteresis number less than 10 have three to five times greater progression of visual field loss.[1] For me, it is often the deciding factor for my initiation of treatment in patients with early or inconclusive signs of glaucoma.

Diabetic macular edema (DME). In diagnosing DME—a disease complication we know can result in irreversible damage to the macula and permanent loss of vision without proper treatment—OCT can help distinguish a subtle epiretinal membrane to pinpoint why a patient's vision is compromised.[2]

Dry eye disease (DED). Research shows that a clinician who used symptoms alone to determine DED would be wrong more than 40% of the time.[3] Point-of-care testing is essential in helping to determine who does or does not have DED. For example, an osmolarity reading between 280mOsmol/L and 295mOsmol/L and within 5mOsmol/L between eyes in a patient that has not applied eye drops in the last two hours has a 3% chance or less of having DED.[4]

Optometry is embracing smaller, more specific and point-of-care testing to help improve our diagnostics. Let's use our innovations to distinguish our profession from online and in-pharmacy efforts, increase our efficiency and accuracy of diagnosis and ensure patients understand their ocular, and often systemic, health depends on us. ■

1. Medeiros FA, Meira-Freitas D, Lisboa R, et al. Corneal hysteresis as a risk factor for glaucoma progression: a prospective longitudinal study. Ophthalmology. 2013;120(8):1533-40.
2. Pershing S, Enns EA, Matesic B, et al. Cost-effectiveness of treatment of diabetic macular edema. Ann Intern Med. 2014;160(1):18-29.
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4. Nolfi J, Caffery B. Randomized comparison of in vivo performance of two point-of-care tear film osmometers. Ophthalmol. 2017 May 22;11:945-50.

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Keep Your Enemies Close

Finally! I can justify snooping online at work—and you can, too.

By Montgomery Vickers, OD

I guess by now you've heard that certain online glasses sellers—who shall remain nameless—are opening brick and mortar stores. What does this mean for private practice optometry? For me, I am relieved I no longer have to spend sleepless nights working on software algorithms that would allow my patients to order their glasses through my website.

OK, I admit, I never spent a single sleepless night working on software algorithms that would allow my patients to order their glasses through my website. In fact, I don't actually know what a "software algorithm" is. When I searched the Internet for an explanation, my well-trained computer just said, "Doc, you really don't want to know, but here is how you should spell it in your column."

Thanks, Sweet Pea.

Top 10

We do, however, like to spend a lot of our limited brainpower following online "competitors" to make decisions about our own practices. Here are the top 10 reasons you should follow online competitors:

10. We can see what they charge for stuff we never heard of.

9. We like it when we feel totally outmanned and intimidated.

8. We can change to keep up with the latest and greatest trends like tinting lenses in house.

7. When a patient says they are going to order whatever they need online, we can either match the price or, even better, hate their guts.

6. We can come up with verbal ammunition that we can use on our online-buying patients so they will hate *our* guts.

5. We can quickly and clearly recognize untapped areas of service for our patients and then more efficiently procrastinate in implementing them in our offices.

4. Turns out we can order our contact lenses cheaper than our labs ever want to sell them for.

3. We can misspell a couple of words in our searches and learn all there is to know about boll weevils.

2. We can compare the layouts of our websites and realize that nobody ever actually buys from us because they saw a picture of our family.

1. If we don't spend our time following online competitors, we will unfortunately have time to call our moms.

Keeping Up with the Joneses

So, obviously, it is critically important to your success to follow and, in your own way, emulate and reproduce what you see others doing online. When I saw that online competitors were opening brick and mortar stores, I felt proud that I am way ahead of them—I had the vision and foresight to have a brick and mortar office clear back in 1979. Tough luck for them.

Doctors, you cannot—I repeat, *cannot*—exactly compete with what these tech savvy, cash flush companies are doing. Yes, you will lose a few sales along the way. You probably won't have a logo designed by a Wall Street advertising firm. You won't have your company go public with its stock.

The good news? By the time they crush your business, your kids will be through college. Keep smiling! ■

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Headbanger's Ball

A patient experiences vertical double vision following a recent fall, but the neuroimaging is normal. What's causing his diplopia? **Edited by Paul C. Ajamian, OD**

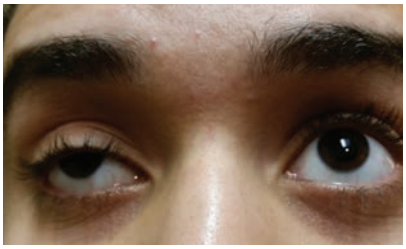
Q I saw a patient this morning who had fallen a week ago, hitting directly on the back of his head. One day later, he presented to the ER with vertical diplopia. They ordered a computed tomography (CT) scan, which was normal. What's at the root of the symptoms?

A One day after the patient fell, he noticed the double vision, which went away with closing either eye, says Dennis Mathews, OD, of Eye Specialty Group in Memphis, Tenn. "With the exception of a two to three prism diopter left hypertropia, distance and near, his eye exam was normal this morning. My first impression: The left superior oblique muscle is the culprit," says Dr. Mathews.

Rationale and Differential

The fourth cranial nerve, also called the trochlear nerve, originates in the dorsal midbrain. "The nerve is long and thin and courses along the tentorum, petrosal ridge and the sphenoid ridge. It is highly sensitive to closed head trauma with small hemorrhages possible," says Dr. Mathews.

Traumatic fourth nerve palsies may be bilateral in a minority of cases, but are usually unilateral, explains Dr. Mathews. "Skew deviations may look like fourth nerve palsies, but these lesions do not show a torsional component, may be comitant early and show other brainstem or cerebellar signs," he says. Those include lower brainstem signs, such as internuclear ophthalmoplegia, and coordinated motor defects if the cerebellum is involved.



Besides trauma, myasthenia gravis should be ruled out in everyone with acquired vertical diplopia.

The differential includes a cavernous sinus lesion, which was ruled out by motility exam, as these normally are associated with oculomotor nerve palsy, abducens nerve and Horner's pupil. Other possible causes are tumor, infection, aneurysm, diabetes and multiple sclerosis. With cranial nerve palsies, practitioners should always consider myasthenia gravis when evaluating acquired vertical diplopia in adults and children.

"In this case, with the history that was obtained, traumatic trochlear nerve palsy is the correct diagnosis. The anterior medullary velum is a common place for a traumatic lesion to present. This area is located under the fourth ventricle in the caudal dorsal membrane."

Pinpointing the Nerve

Using the Park three-step test can allow practitioners to differentiate the nerve responsible for causing the vertical defect, says Dr. Mathews. "After the side of hypertropia is identified, the patient is asked to look to both sides with the hyper deviation being compared. If the hyper deviation increases when looking toward the opposite side, a superior muscle is involved; otherwise, an inferior muscle is involved."

The four possible muscles causing our patient's problem are the left superior oblique, the left inferior rectus, the right superior rectus and the right inferior oblique. "In this patient's case, the three-step test showed the left superior oblique was involved. While the brain CT scan was normal, these images are superior to magnetic resonance imaging (MRI) only in revealing acute blood or bony defects, but not soft tissue disease," says Dr. Mathews. He adds that an MRI would be appropriate to discover conditions such as multiple sclerosis, a tumor in the cavernous sinus or brain stroke. Magnetic resonance angiography would be used to discover an aneurysm.

Treatment

Dr. Mathews recommends patching to get the patient through the next month. One unobtrusive way to cover the involved eye is with a few pieces of scotch tape on the back surface of the patient's glasses.

Dr. Mathews says that, even though a general timeline doesn't exist to consider bloodwork, imaging, prism and, ultimately, surgery if the patient doesn't improve after one month, consider referral to a neuro-optometrist, neuro-ophthalmologist or neurologist for a second opinion. ■

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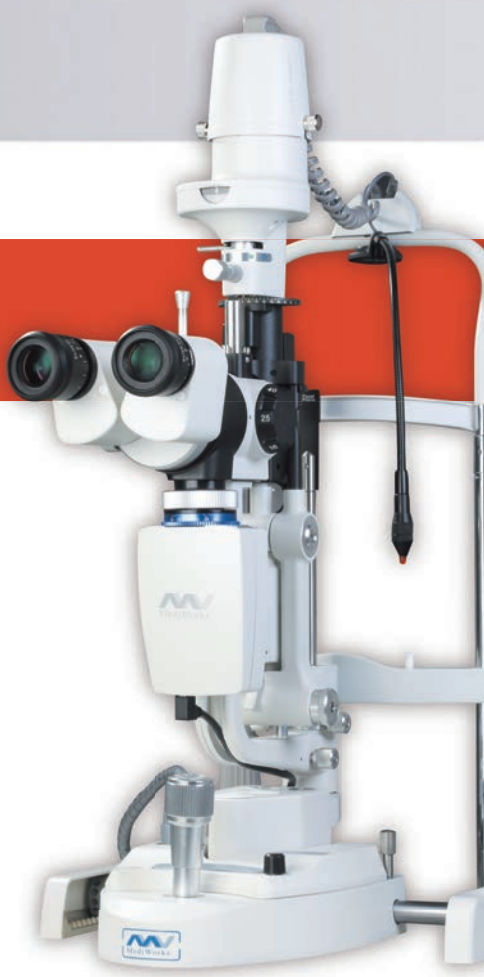

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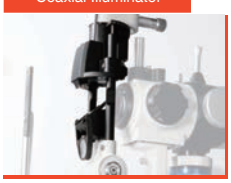

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Combining Images, Not Codes

With new OCT technology comes new coding do's and don'ts.

By John Rumpakis, OD, MBA, Clinical Coding Editor

OCT is quickly becoming an integral part of optometric practice. From a coding standpoint, OCT was first introduced in 1999 with code 92135, *scanning computerized ophthalmic diagnostic imaging (e.g., scanning laser) with interpretation and report, unilateral*—a broad, non-anatomically specific code representing all ocular OCT procedures. In January 2011, 92135 was replaced with three new codes with anatomically-specific application and rule sets:

- **92132:** *scanning computerized ophthalmic diagnostic imaging, anterior segment, with interpretation and report, unilateral or bilateral.*
- **92133:** *scanning computerized ophthalmic diagnostic imaging, posterior segment, with interpretation and report, unilateral or bilateral; optic nerve.*
- **92134:** *scanning computerized ophthalmic diagnostic imaging, posterior segment, with interpretation and report, unilateral or bilateral; retina.*

With these new codes came new edits within the National Correct Coding Initiative (CCI) identifying areas of conflict when performing these tests on the same day as other commonly performed procedures or with each other.

Coding Implications

Fundus photography with interpretation and report—92250—and either 92133 or 92134 cannot be performed on the same date of service on the same patient. The 2017 CMS policy manual states:

Fundus photography (CPT code 92250) and scanning ophthalmic computerized diagnostic imaging (e.g., CPT codes 92132, 92133, 92134) are generally mutually exclusive of one another in that a provider would use one technique or the other to evaluate fundal disease. However, there are a limited number of clinical conditions where both techniques are medically reasonable and necessary on the ipsilateral eye. In these situations, both CPT codes may be reported appending modifier 59 to CPT code 92250.

This language is problematic for those who view the -59 modifier as a "magic trick" for getting around this rule set. The clinical conditions that warrant both techniques are few and far between, and billing these two on the same day should be very rare.

OCT-A

New technology, such as OCT-A, often tempts clinicians to get creative with coding to embellish their reimbursements. But OCT-A should be reported with CPT 92134 only, without additional codes such as 92499 (unlisted ophthalmic procedure). The broad definition of 92134 easily encompasses the application of angiography, and no additional code should be used.

The better and more integrated the technology, the more integral the provider's clinical acumen. For example, clinicians must assess the patient's clinical presentation and which structure/image is the most clinically applicable when deciding on the proper code—not simply bill both the fundus photograph and the OCT, or bill only the fundus photography for a higher reimbursement.

The November 2014 CPT Assistant clarifies coding OCT procedures with a clinical example: *Our office performs fundus photography examinations using a scanning laser which produces a fundus photograph. Is it appropriate to report CPT code 92135 for this method of examination of the fundus?*

CPT's answer drives home the point: "It is important to note that if the only necessary service provided is generating a fundus photograph without the need to quantify the nerve fiber layer thickness and to analyze the data via a computer, then reporting code 92250 is appropriate, even if the photograph was taken with a scanning laser." The reverse is true as well; if an instrument captures both images simultaneously, the clinician must determine which is most clinically relevant prior to the point of capture.

As OCT technology progresses and combined imaging becomes more common, the burden of proper test application, interpretation and subsequent coding of those tests will increase for the clinician. Stay tuned as policies continue to change. ■

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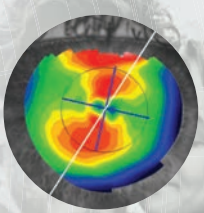

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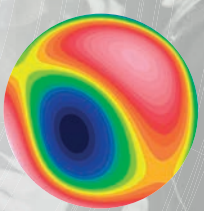

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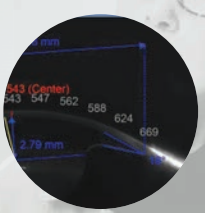

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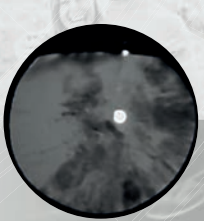

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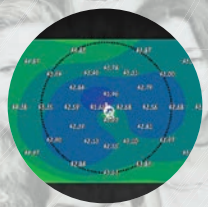

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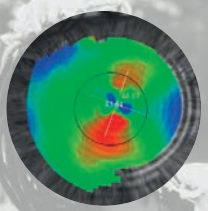

Topography

Tonic Pupil? Loosen Up

Often, practitioners are alarmed by acute isolated dilated pupils. This need not be the case. **By Michael Trottini, OD, and Michael DelGiodice, OD**

During a medical exam, an internal medicine doctor noted a difference in pupil size—left pupil larger than right—in a 62-year-old Caucasian male, which was never noted on prior visits. In addition to a referral to our clinic, his internist scheduled him to have an intracranial magnetic resonance imaging (MRI) and angiography (MRA).

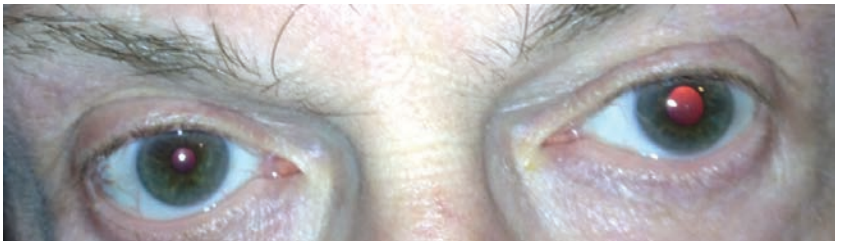

Fig. 1. The pupils of both eyes in bright light measuring 3mm and 4.5mm in the right and left eyes, respectively.

The patient himself was unaware of this change; however, upon questioning he noticed increased light sensitivity out of the left eye for at least three months. He denied any other neurologic issues or symptoms. His medical history was remarkable for hypothyroidism and high cholesterol and was taking levothyroxine and atorvastatin. He denied ever using any motion sickness patches or over-the-counter eye drops.

The patient's best-corrected visual acuities were 20/25 in each eye. Pupils measured 3mm OD and 4.5mm OS in bright light and 7.5mm OD and 7.5mm OS in dark light. Reverse flashlight testing revealed no afferent pupil defect. Extraocular muscles were full and smooth. No third, fourth or sixth nerve palsy or other neurologic deficit was noted. Anterior segment exam was remarkable for 2+ nuclear sclerosis. When examining the pupils under the slit lamp, the right pupil showed an even and normal constriction and dilation when turning the slit beam on and off. However, the left eye showed a generalized sluggish constriction as well as sectoral iris paralysis temporally *(Figure 1)*. Posterior segment exam was unremarkable. No disc edema, disc pallor, artery or vein occlusions existed and the retina was flat and attached.

The Verdict: Tonic Pupil

The history and clinical findings were most consistent with a tonic pupil. We instilled 0.125% pilocarpine in the left eye, which, after 30 minutes, constricted the pupil to 3mm, confirming the diagnosis *(Figure 2)*. We discussed the tonic pupil with our patient as well as informing his physician. Our patient was otherwise in good general health and no further workup was necessary. The MRI and MRA were subsequently cancelled. He was very minimally bothered by the light sensitivity and wears sunglasses when needed.

Discussion

Often, patients who present with an acute isolated dilated pupil are put through extensive and unnecessary testing and referrals. Clinicians fear there is a serious underlying etiology when the majority of causes for acute isolated dilation are benign; these include tonic pupil, pharmacologic dilation and transient pupillary mydriasis.

By far the biggest fear of a patient presenting with a dilated pupil is a third nerve palsy. Although in theory a compressive lesion of the third nerve may cause an isolated dilated pupil without any extraocular motility deficit or eyelid ptosis, in practice this rarely occurs.[1] In these rare cases of third nerve palsies presenting initially with only pupillary involvement, a review of the literature found that these patients also either presented with headache, seizure or other neurological deficits, or the extraocular motility deficits developed early in the course of their illness.[2,3]

Tonic pupil, as seen with our patient, is a common cause of isolated pupil dilation. Clinical features

include diminished or absent pupillary reaction to light stimulus, segmental iris paralysis, no afferent pupillary defect by reverse flashlight testing and preserved constriction to accommodation.[1,2] Most commonly seen in women ages 20 to 40, tonic pupils arise from damage to the ciliary ganglion, the cause of which is rarely identified.[2] In response to this damage, aberrant regeneration of fibers originally destined for the ciliary body instead innervate the iris sphincter.[2] This results in diminished response to light and an enhanced tonic reaction to accommodation.[1]

When this aberrant regeneration is restricted to certain segments of the iris, sectoral iris paralysis occurs.[1] This phenomenon is best seen under the slit lamp while focusing on the iris and turning the slit beam on and off. Additionally, because the ciliary ganglion is now denervated, it generates more postsynaptic acetylcholine receptors, allowing for such a low dose of pilocarpine to cause pupillary constriction.[1] Pilocarpine is not formulated in 0.125% concentration; however, it can easily be made in office by mixing one drop of 1% pilocarpine to seven drops of any artificial tear. A tonic pupil is generally a benign condition and no further workup is necessary; however, the presentation has been reported in patients with connective tissue disorders, temporal arteritis and orbital trauma.

Differential Diagnosis

When pharmacologic pupillary dilation is suspected, a careful and thorough history will often reveal the source. Anticholinergic agents such as scopolamine patches and pesticides as well as various over-the-counter eye drops containing mydriatic

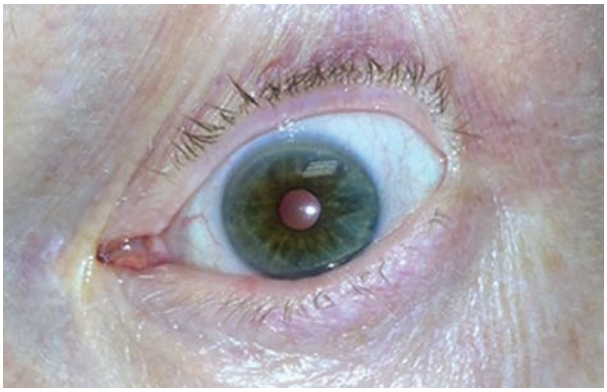

Fig. 2. In the same patient, we observed constriction of the left pupil 30 minutes after instilling 0.125% pilocarpine.

properties can all cause pupil dilation. If no agent is identified during questioning, 1% topical pilocarpine can be instilled to test for pharmacologic blockade. A pupil that is dilated due to pharmacologic blockade will not constrict to the pilocarpine, but one dilated secondary to a third nerve palsy will.[1,2] The effects of pharmacologic pupil dilation are transient and the pupil will return to its normal size in a relatively short time frame.

While isolated acute pupillary dilation can be alarming at first, it is almost always the result of a more benign process.

Transient pupillary mydriasis or episodic mydriasis has been observed in patients with a history of migraine.[1] One report looked at 24 patients who presented with episodes of transient pupillary mydriasis.[4] Nineteen patients were female, 14 had a history of migraines and the median age was 31.[4] The median duration of the episodes was 12 hours with a frequency of two to three episodes per month.[4] No underlying neurological disorders were identified in any subjects after testing and workup.[4] If the transient or intermittent nature of these episodes can be established either by the patient's medical history or observed during clinical evaluation in isolation without any other findings, they generally do not require any additional workup or neuroimaging.[1]

While isolated acute pupillary dilation can be alarming at first, it is almost always the result of a more benign process. The main concern with this finding is to ensure it is not in association with a third nerve palsy. That is why clinicians must review and understand the various clinical features of these other benign disorders to save the patient from undergoing various unnecessary testing and anxiety. ■

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HIGH PATIENT AND ECP PREFERENCE FOR AIR OPTIX® PLUS HYDRAGLYDE® CONTACT LENSES

Melissa Barnett, OD, FAAO, FSLS
Sacramento, California

Dr. Melissa Barnett was compensated by Alcon for her authorship of this advertorial.

The daily lives of contact lens wearers are full of obstacles to comfortable lens wear, including dry or smoky air, digital device use and long days of lens wear. These obstacles have their impact: in a recent survey, 2/3 of contact lens wearers said they experience dryness/discomfort with their current lenses.[1]

At my practice, my goal is to provide contact lenses with improved technologies that promote patient comfort, healthy eyes and contact lens compliance. Studies show that monthly replacement lenses promote better replacement compliance than 2-week lenses.[2,3] Patients and ECPs agree that AIR OPTIX® plus HydraGlyde® monthly replacement contact lenses provide a positive wearing experience.[1]

I recently participated in a survey of contact lens satisfaction in habitual wearers of 2-week or monthly replacement contact lenses (N = 229). We surveyed our patients about their habitual brand, and then about AIR OPTIX® plus HydraGlyde® contact lenses after a 1-month trial.[1] Patients used CLEAR CARE® PLUS Cleaning and Disinfecting Solution or OPTI-FREE® PureMoist® Multi-Purpose Disinfecting Solution for daily lens care. Both of these lens care products feature HydraGlyde® Moisture Matrix. At the end of the trial period, patients surveyed expressed strong satisfaction with AIR OPTIX® plus HydraGlyde® contact lenses: more than 9 out of 10 patients surveyed agreed these lenses felt comfortable upon insertion each day,[1] and four times as many agreed (vs disagreed) that AIR OPTIX® plus HydraGlyde® lenses felt comfortable through the end of the day.[1] Finally, four times more patients surveyed preferred AIR OPTIX® plus HydraGlyde® contact lenses (plus daily HydraGlyde® lens care), over their previous lenses, after wearing them for 1 month.[1]

Among the eye care professionals who participated in the survey (N = 20), three out of four agreed** that AIR OPTIX® plus HydraGlyde® contact lenses will be the preferred monthly replacement lens in their practices. The same ratio also agreed† they would proactively recommend their 2-week and monthly replacement wearers switch to AIR OPTIX® plus HydraGlyde® contact lenses.[1]

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Recent studies demonstrate the wettability of AIR OPTIX® plus HydraGlyde® contact lenses. In one, Placido rings were projected onto surfaces of several lenses.[11] A wet surface reflects a stable image of the rings, but as the surface dries, the reflections become distorted. Directly out of pack, AIR OPTIX® plus HydraGlyde® contact lenses showed more stable reflections at 2 minutes than several competitors ($P<0.01$), demonstrating excellent lens surface moisture retention. In another study, time to lens surface moisture breakup (the time it takes for the first "spot" of dryness to appear on the lens) was measured after soaking lenses in PBS solution for 16 hours.[12] The time to lens surface moisture breakup of AIR OPTIX® plus HydraGlyde® contact lenses was longest (19 seconds), indicating lasting lens surface moisture. (Figure)

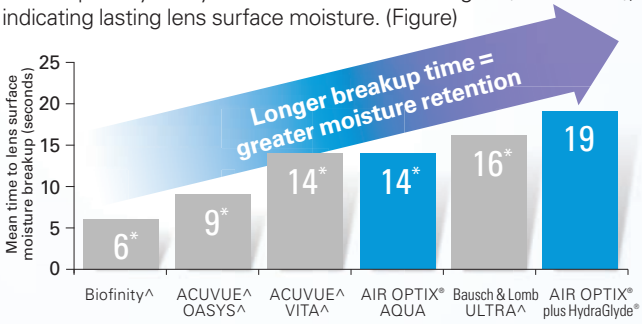

Figure: AIR OPTIX® plus HydraGlyde® Contact Lenses Provide Long-Lasting Lens Surface Moisture Retention After 16 Hours of Simulated Wear[12]
Mean time to lens surface moisture breakup after 16-hour soak in phosphate-buffered saline solution. Ten lenses per brand were analyzed.
*$P<0.05$ vs AIR OPTIX® plus HydraGlyde® contact lenses.
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Recommend lens care solutions with HydraGlyde®—CLEAR CARE® PLUS with HydraGlyde® or OPTI-FREE® PureMoist®—as the perfect combination with AIR OPTIX® plus HydraGlyde® contact lenses to keep outstanding comfort going all month long,[1] so your patients can see, look and feel their best!

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†Based on ECPs who "agreed" or "somewhat agreed" with the statement: "I plan to proactively recommend to my 2-week and monthly contact lens wearers that they switch to AIR OPTIX® plus Hydraglyde® contact lenses."
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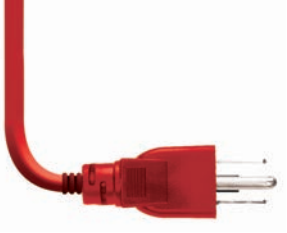

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Your Phoropter on Steroids?

High-tech refraction systems tout greater accuracy, comfort and time savings. Skeptics ask if such gains justify choosing this over other practice-enhancing investments.

By Jane Cole, Contributing Editor

The tried-and-true phoropter is one of the most venerable devices in the profession and likely one of the first pieces of equipment you added to your practice. It may very well have predated you at the office, in fact. But are the days of spinning dials and asking the patient, "Which is better? One or two?" going to become a thing of the past? Advocates of digital refraction systems believe it's possible.

Photo: Kambiz Silani, OD

High-tech digital refraction systems add a 'wow factor' to modern optometric practices, says Kambiz Silani, OD, of Beverly Hills (pictured).

"In general, the purpose of these devices is to make the process more expedient, more consistent and more accurate than manual refraction," says Alan Kabat, OD, a professor at Southern College of Optometry. "Digital refraction systems can minimize not only the time a doctor spends performing this procedure but also the physical wear and tear on his or her body. Any optometrist who has done retinoscopy on 20 or 30 patients in one day can relate."

Today's high-tech upgrades range from digitizing the phoropter to replacing it outright, while others incorporate wavefront aberrometers to improve accuracy. Still, not all are convinced of their benefits over the standard phoropter—at least not enough to justify the price.

Here's a look at the latest high-tech refracting systems and some dissenting opinions from those who believe the venerable phoropter suffices, especially with the opportunity cost of forgoing other upgrades to your practice's suite of equipment when investment budgets are tight.

Greater Accuracy?

Dr. Kabat is involved in a study using the Voice Activ Subjective Refractor, or VASR (Vmax Vision,) a wavefront autorefractor that uses a concept called subjective point spread function (PSF) refraction. "From my own personal experience, I have found the device is at least as accurate as I am in attaining 20/20 vision. But according to the manufacturer, this platform can routinely deliver 20/16 or even 20/12."

Note that most patients under age 65 see better than 20/20 and can be refracted as such with a conventional phoropter, says Mark Wilkinson, OD, a clinical professor of ophthalmology and visual sciences at the University of Iowa. "It is not hard to get them to their best acuity level with a phoropter and standard refraction techniques," he says. "We have to remember that Snellen acuity was developed during the Civil War, and 20/20 is simply a number, not the best vision a person can attain."

The idea behind PSF is that the best refraction target is a point as opposed to letters or images that are two-dimensional, which may fail to fully reveal the extent of astigmatism or higher-order aberrations in the eye, according to Vmax Vision. PSF uses point spread images, which automatically correct for both low- and high-order aberrations in the final prescription. The PSF target appears blurred when looking at the endpoint, rather than smaller and darker as in Snellen letters, which prevents over-minus, according to the company.

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Acuity Pro is well known for its flexibility. The Windows program resides on a USB thumb drive. Acuity Pro can be moved from a failed computer to a new one in minutes. Or, it can be transferred to a laptop for use in nursing homes and school screenings. Or, in Pacific University's case, the drive is installed on two all in one systems in their new mobile clinic designed to see patients in unserved areas.

Dr. Sarah Martin, community outreach assistant director, leads students on outreach vision screenings and exams in the community and rural areas of Oregon. Acuity Pro donated two all in one systems for the mobile clinic, allowing for a clean, compact, and accurate means of testing visual acuity in all populations.

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From the patient's point of view, it is easier to discern differences using a point target than a traditional Snellen chart, making the entire process faster and creating less "refraction anxiety," Dr. Kabat says. "The result is an easier scenario for patient and doctor, and the potential for enhanced acuity by a more precise assessment."

David Geffen, OD, of La Jolla, Calif., has been using the PSF system for the past three years and says he has found patients with a previous 20/20 prescription now are able to read 20/15, or in some cases, 20/10 letters.

A Menu of High-Tech Refractive Options

Optometrists looking to trade in their phoropter have several choices to consider. Here's a look at some of the refractive systems on the market, as promoted on the manufacturers' websites and in testimonials from proponents:

TRS-5100 and Epic-5100 Refraction Workstation (Marco): The Epic decreases workup time to under 10 minutes and performs the refractions in three to five, according to Marco. Practitioners control the refraction process from a small, portable keypad. The TRS-5100 is programmable, and all the lenses are moved at the touch of a button. It improves the patient's ability to compare previous and new prescriptions. The device can configure in the traditional lane or as part of the Epic workstation.

CV-5000 (Topcon): This automated phoropter offers fast lens rotation and comfort for the doctor and patient, according to Topcon. The device provides three interfaces for operation. The first option is a one-dial controller that uses a 10.4-inch color LCD touch screen. The second and third options allow you to load the controller software on a PC or the PC integrated inside the CV-5000 to be controlled by the PC mouse. The CV-5000's automatic phoropter head also provides fast lens rotation for user and patient comfort, Topcon says.

PSF Refractor, Perfectus and VASR (Vmax Vision): The PSF refractor offers a more accurate prescription derived from refraction data measured in 0.05D increments—five times more precise than a phoropter's 0.25D increments, according to the company. The system uses a subjective PSF, and 20/12 is an achievable goal, Vmax says. The Perfectus integrates wavefront autorefraction, and the VASR is patient-driven.

"This is a small company, but pretty impressive, since they are using a very different system," says Paul Karpecki, OD, of Lexington, Ky. "Patients simultaneously get upper and lower images that seem easier to decipher, including those individuals with cataract or keratoconus." Adds Dr. Karpecki, the ability of these and other systems to measure night vision as well comparing their previous refraction (with Snellen letters) with a single touch of a button is valuable for the patient.

Dr. Kabat says the VASR system uses artificial intelligence software and verbal commands to guide the patient through the process. "With an alert and responsive subject, the procedure typically takes about five minutes for both eyes," he says. "Thus far, the results we've obtained and the attitudes of our patients towards this new technology have been very positive."

Vx55 (Visionix): The Vx55 enables bluetooth communication between the refractor and the tablet, plus connection and transmission to other devices. "It shows an image on the tablet of a phoropter," Dr. Karpecki says, "and you just take your finger and press the buttons." These types of automated phoropters cut down on shoulder injuries that are common in professional optometry, he adds.

For Kambiz Silani, OD, of Beverly Hills, Calif., a Visionix advisory board member, this is his system of choice; he says it is "as easy to use as an iPad." His practice uses the entire Visionix suite that also includes an automated lensometer, Vx40, to measure spectacle lenses and the "technician friendly" Vx120, which performs numerous diagnostic testing of anterior segment health as well as wavefront autorefraction. "As a bonus, these devices intelligently communicate with one another to make the transition smoother and more efficient for the technician, the optometrist and the patient," says Dr. Silani. The company also offers the Vx60 digital autophoropter system, which comes with a control panel.

Huvitz HDR-7000 (Coburn): This digital refractor features a slim design with wide viewing angles to improve precision, according to the company. It features fast and silent lens loading with a dual cross cylinder lens, automatic occlusion and automatic convergence, Coburn says. It includes 18 visual acuity test charts, 26 vision test charts and up to 35 user-defined unit test charts. Additionally, the device offers wireless communication and is equipped with a 21-point exam package. According to Coburn, the HDR-7000 alleviates difficulty by displaying results for easy reading for examiners and patients.

Visuphor 500 (Zeiss): This subjective digital refraction system, which is modular and expandable, offers an intuitive operation through a touch-screen interface, its manufacturer states. Preconfigured workflows save physicians' time preparing refraction tests, according to Zeiss, as settings for each test are optimally adjusted, making standard refraction tests simple and fast. Settings and workflows can also be individually configured in the freestyle mode. Patient contact with the headrest is continuously monitored and displayed on the screen.

Phoroptor VRx Digital Refraction System (Reichert): "This automated phoropter is a quiet and smooth system," Dr. Karpecki says. It offers fast lens exchanges, motorized prisms, split cylinder lenses and a quiet operation with a touchscreen display and ergonomic keypad, according to the manufacturer. Dr. Karpecki adds the system is easy to learn and offers numerous pre-program tests. The device connects to electronic medical records systems as well as numerous models of pre-test and acuity devices from both Reichert and non-Reichert brands.

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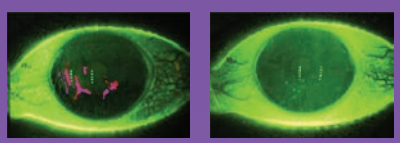

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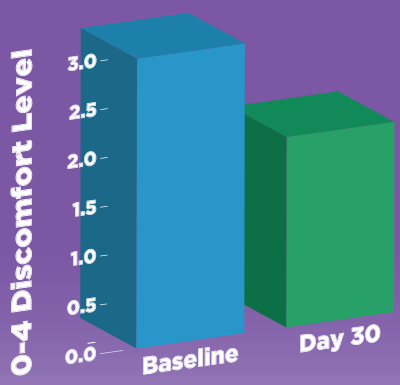

"A lot of doctors just stop at 20/20. And that's not fair. If we can get someone to 20/12, we should try," Dr. Geffen says. The system allows a patient to see in a more refined way than standard phoropters, allowing for an increase in best-corrected acuity, he says.

By using some of the higher-tech devices, you are essentially neutralizing the normal aberrations of the eye, Dr. Geffen adds. The PSF is five times more accurate than a phoropter, he says, and digitized spectacle lenses allow a doctor to custom fit lenses to virtually to any prescription.

"We aren't creating a wavefront lens, but rather using a point spread function to create a more accurate prescription," says Dr. Geffen. "When you refract that way, it optimizes those aberrations, so we are giving the patients better glasses, and that's why we are getting better vision results. It is better because we are able to refine the patient's Rx to a much higher degree than with Snellen letters. We're optimizing patients' aberrations."

However, even though a prescription can be determined in 0.05D increments, lens manufacturing is still fabricating lenses in 0.25D increments if a wavefront lens is not used, Dr. Wilkinson says. "Also, given that we expect each 0.25D to equal one line of vision for a normally sighted person, I am not sure how a person is going to appreciate a 0.05D difference in refraction power, even if fabricated in a digital lens."

Dr. Geffen also notes that patients set to undergo refractive or cataract surgery can now be corrected to a much higher level of precision and "doctors still using phoropters are basing their readings on older technologies" that may not fully characterize the quality of the surgical outcome.

Also, the high-tech refractors can benefit patients with reduced visual acuity from conditions such as keratoconus or macular degeneration, Dr. Geffen adds. With a keratoconic patient, a diopter or two of difference in 10 to 15 degrees of astigmatism on Snellen letters can be achieved. "But with point spread function [devices], we can bring them down two or three lines," Dr. Geffen says. In patients with macular degeneration, if the size of the point spread is increased, the patient can see smaller differences and sometimes a line of acuity is picked up, he adds. "It's a big deal for those patients."

Photo: Vmax Vision

Dr. Geffen (pictured) says the PSF Refractor provides more accurate readings than the standard phoropter through its point spread function.

Dr. Wilkinson agrees that a phoropter should not be used for this type of patient but doesn't believe the only way to improve their correction is via digital refraction. "A trial frame refraction is best for refining acuity in folks with less than normal vision," he argues.

Upgrading the Experience

Paul Harris, OD, of Southern College of Optometry, notes that the ability of these systems to dial in the patient's habitual prescription and save it is a major plus, in addition to being able to show the patient their old Rx and their new one with just the touch of a button.

Dr. Geffen, who had carpal tunnel surgery 10 years ago—a condition he says is common in the profession—finds his new system is faster and less physically taxing. "With the phoropter, we sat there and used our computer and then we spun dials. Now all I have to do is use a mouse to refract. So, it's easier on the doctor, and I feel the patients find it easier to tell differences than looking at the Snellen letters and being asked, 'Which is better? One or two?'"

"Automated phoropters offer a refreshing and modernized addition to the exam lane," according to Kambiz Silani, OD, of Beverly Hills, Calif. "Patients have grown accustomed to seeing the same manual phoropter for decades, so embracing the updated digital refracting systems adds a coolness factor to the millennial practice. In our office, patients have responded enthusiastically toward the latest gadgets with countless positive remarks."

While not all these technologies are considered standard of care, they are increasingly present in practitioners' offices. Adds Dr. Geffen, "we keep adding all this high-tech equipment, so the patient goes through this huge pretest regimen where they have an OCT done, topography, visual fields and a special camera [for ultra-widefield imaging], and then they go into your exam room and you have a flat screen and all this pretty stuff and what do they see? That same thing hanging on the chair that optometrists have used for 100 years. So they respond, 'Oh, are you still using that, doc?' If your practice is based on adding the latest technology, it doesn't look very cutting edge to be using the same piece of equipment you used when your 50-year-old patient was five."

Should You Make the Switch?

It's not so much a decision of whether but rather when to replace the manual phoropter, Dr. Kabat argues. "Let's face it—health care has become an exceedingly complex business over the last 20 years, with greatly increased responsibilities for data collection and medical coding." As reimbursements from third parties have declined, doctors have been forced to accommodate more patients into their schedules to maintain profitability, he says. "To remain competitive, doctors must be able to adapt and take advantage of products that can not only help patient flow but also provide a better overall patient experience. I suppose the most important question an optometrist can ask is, 'Do I want to continue to be successful?'" If so, upgrading to next-generation equipment is one way to help foster that.

Photo: Visionix

Vx55 enables bluetooth communication between the head and the tablet.

Additionally, Dr. Kabat says doctors should consider:

- Financial constraints, i.e., how much is available to spend, in terms of upfront purchase or fixed payments.
- Office space and desired patient flow.
- Number and type of ancillary personnel.

These variables can help to determine what type of unit is best for a given practice, whether it will be used in the exam room or an ancillary testing room, and who will primarily operate it.

Stumbling Blocks

Yet optometrists should consider the costs of these systems and whether they are the best use of investment dollars put toward the success of their practice, Dr. Wilkinson says.

When considering whether to trade in your phoropter for a high-tech model, the most commonly cited obstacles to consider are as follows:

Costs. For Dr. Geffen, the biggest perceived con is cost. "They are expensive units and cost more than the standard phoropter." Some doctors may view refracting equipment with a don't-mess-with-success mindset, since they feel their phoropter does the job and they prefer to stick with what already works. Adds Dr. Kabat, "whether we admit it or not, most of us are creatures of habit and immensely dislike change."

Support. Some of the companies manufacturing the newer devices are small and might not offer the customer support of a company with a national footprint, should trouble arise, Dr. Geffen says. "If you're somewhere out in a more rural location and you need help, it might be hard to get." In his experience, his system has needed minimal service, and his practice is located near where it's manufactured.

Design limitations. If performing retinoscopy is a standard part of an exam, the digital systems will not suffice as the only setup, Dr. Harris notes. "Most of the digital systems I've had contact with have a set of controls off of the instrument head itself." These types of systems are "back saving," he says, since a doctor doesn't need to reach around a patient to change a lens, and some patients prefer to be across the room from the doctor during the exam. "Retinoscopy, though, requires one hand on the scope and for us to be on the visual axis of the patient. Since our visual attention is on the reflex, it can't be on the controls of the system, so a must for such a system, if it can be used for retinoscopy, would be touch controls with haptic feedback for sphere, cylinder power and axis," he says.

Diminishing the doctor's role. Additionally, optometrists may worry that the new technology will somehow be "too good," making the role of the doctor obsolete, Dr. Kabat adds. "In essence, ODs are concerned they will be replaced by these machines. Of course, that is nonsense. These individuals confuse the process of refraction with the art of refractive prescribing. A doctor's knowledge and experience can never be replaced by technology, but technology can greatly enhance a doctor's ability and efficiency."

"Optometrists are far more than 'refractionists,' in that what we derive is a range of lens powers, under different conditions—for example, objective and subjective—to which we apply professional judgment," says Dr. Harris. "This is what leads us to write final prescription numbers that resemble, but were not exactly found in, any of the measurements taken of our patients' eyes." Optometrists, he stresses, do not "blindly transfer measurements to a script. We take into account how patients use their visual systems to find formulas that help them process visual information more efficiently and effectively, and minimize future negative changes."

The Role of Refraction

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Some of the newer refracting systems are geared toward the doctor and designed to be operated in the lane. This expedites the refraction process by incorporating automation and integration between the autorefractor, the subjective presentation and the electronic medical records, Dr. Kabat says. But techs could use other systems, such as the VASR, during pre-testing, he says.

In some offices, technicians already perform the bulk of refractions, but optometry has not embraced this practice as quickly or completely as ophthalmology, Dr. Kabat says. "As a clinical professor, I've been delegating refractions for nearly 25 years. And while that responsibility goes to an optometry student rather than a technician, the outcome is the same." Someone else collects the refractive data and refines it as best as possible, then he decides what is most appropriate to prescribe. "If the data I receive seem invalid or unusual, I check it myself. This process applies to digital technologies, but the consistency of outcomes is far better, while the need for adjustment is far less."

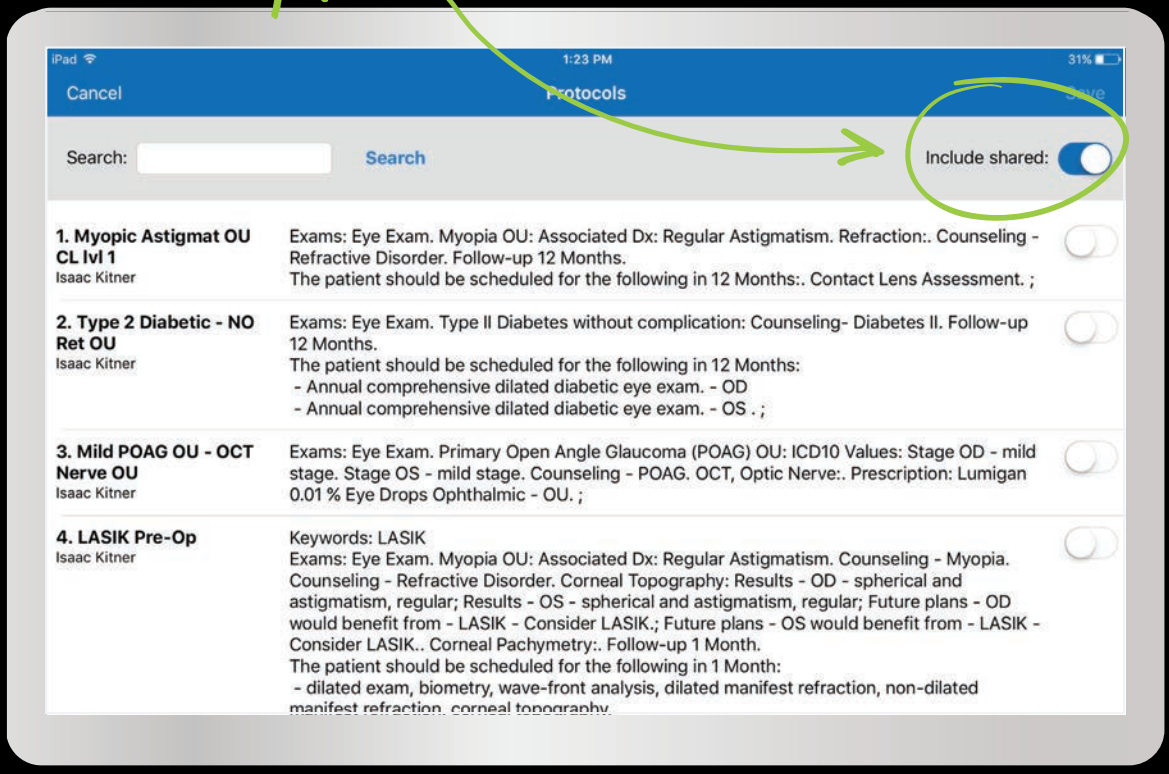
Unburdening doctors from the tedium of refraction can mean more time spent on medically focused eye care while still providing accurate spectacle and contact lens prescriptions for patients, Dr. Kabat says.

Others bristle at the notion. "Refraction should remain a critical component of optometric care," says Dr. Wilkinson. If it is not, optometry will be indicating that online refractions and refractions done by anyone with an autorefractor are acceptable. We'd then move away from our roots, with refractions done by a non-optometric person."

Whether to pass the refractive reins over to a tech remains an individual choice. "Delegating the objective pre-testing tasks to a technician allows doctors to spend more chair time with the patients to discuss and review treatment options and less time just collecting data, adds Dr. Silani. "As far as performing, it's still common practice to be performed by the eye doctor as well as expected and appreciated by the patient."

Will the new technology make phoropters a thing of the past? Not so fast, says Dr. Harris. For the short term, he believes the standard phoropter will remain the mainstay. Clinical staples of the refractive exam, specifically retinoscopy, can be difficult with newer systems, he says. However, "I can foresee a time when the things leaving me reticent to shift over have been addressed and the benefits begin to outweigh the standard phoropter's use. I look forward to the day when these technologies help me change people's lives more than I can now." ■

Awesome!



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Downsize Your Technology to Enhance Your Practice

Here's a look at the many handheld devices that can provide significant benefits for your practice. **By John Wimbish, OD**

Convenience and portability are the drivers of much of today's innovation, and now unlimited communication, information and entertainment are only the click of a button away. As consumers, our patients have come to expect the same seamless integration of technologies into health care.

In optometric practice, the push toward electronic health records (EHRs) and better technological integration has spurred innovation in diagnostic and patient management devices, including for refraction, intraocular pressure measurement and imaging of pathologies and anatomic variants, just to name a few.[1]

But with these new technologies comes increased expenses. In addition, each instrument has a footprint that has to fit into our sometimes-cramped office spaces with an ideal position for efficient use of time and space—not to mention designed with patient convenience and comfort in mind. That's where portable technologies come in.

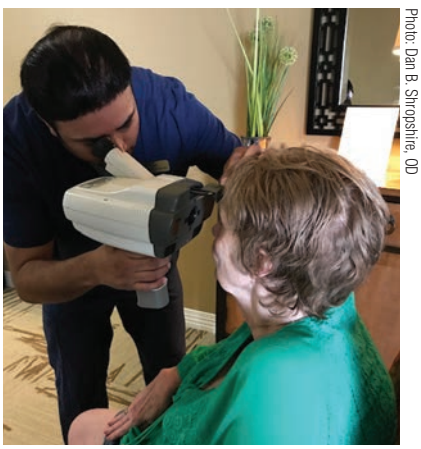

Photo: Dan B. Shropshire, OD

Portable autorefractors allow optometrists and their staff to obtain crucial data in any setting.

The Benefits of Minimization

Smaller instruments have saved the day for many working in tight spaces, and they can improve many aspects of practice, including:

Efficiency in the office. With handheld instruments, practitioners can overcome the limitations of space, allowing better time efficiencies and patient flow with no loss of volume or quality of diagnostic information. Handheld devices eliminate the need to move the patient from place to place or instrument to instrument during the work up. Using multiple portable devices allows several patients to receive testing simultaneously, and the practitioner can more efficiently review the information.

Flexibility outside the office. Handheld instruments free practitioners to provide much-needed care for patients who cannot come to the office. In states that allow mobile optometry, portable cameras, phoropters, autorefractors and slit lamps expand the patient base into nursing facilities and hospital rooms, for medical mission work and during emergencies as a valued first response team member.

Communication. More and more handheld instruments are adding the essential ability to communicate directly with EHRs while practitioners are caring for patients out of the office. The need for integrated care will drive continued improvement in this technology.

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Profitability. The cost for most handheld devices is often less than the desktop mounted versions, and third-party reimbursements are the same as with the more expensive, larger instruments. In addition to these direct savings, proper use of handheld instruments can speed up pretesting and other data gathering, potentially increasing patient volume without sacrificing clinical services.

Because these instruments do not require extra room, small-office doctors can use them to increase their billable services, despite their current space limitations.

There's Always a Downside

While desk-mounted devices tend to rely less on the operator for quality data collection, the same is not true with handheld devices. Less-than-careful alignment and timing on the moment of data collection will lead to inaccurate or poor quality information when using a portable device. Practitioners will need extra time to first learn the proper use of the instrument and then to carefully train staff members.

Handheld devices are also more fragile than their desk-mounted counterparts. It's nearly impossible to knock over and damage a desk-mounted device, but every use of a handheld instrument is an opportunity for it to be dropped, shaken or otherwise damaged.

While connectivity is improving for handheld instruments, desk-mounted devices are still more securely hard wired into today's EHRs for immediate availability of information. Even as the technology improves, handheld devices will still require wifi or cloud-based technology for data transfer, which can increase security risks for the patient's information.

Although handheld devices, including smartphones, can often improve data collection in small spaces, for special patient populations and outside the office, they may also cause disruption when used specifically for communication. One study looked at clinicians' use of smartphones to communicate in a hospital setting and found it improves efficiency over the use of pagers.[2] However, smartphone use also increases interruptions, creates a gap in perceived urgency, weakens interprofessional relationships and is linked to more unprofessional behavior.[2] The researchers suggest that, while the technology has its benefits, improvements are necessary to balance the positives with the negatives.[2]

Handhelds in the Office

Here's a look at some of the portable tools changing optometric practice:

Wavefront aberrometers. These devices are lost-cost options that often perform similarly to commercial autorefractors in objective refraction.[3] Research suggests the need for more patient education, however. One study found clinicians must communicate that the measurement takes several seconds to complete—possibly limiting its utility with some patients.[4]

Autorefractors. Research suggests handheld autorefractors can provide refractive readings consistent with those captured with more traditional refractive techniques.[5] One study assessed the refractive error, with and without cycloplegia, of 50 visually normal patients using a handheld autorefractor and compared the results with those obtained with retinoscopy, subjective refraction and two commercially available tabletop autorefractors.[5] The results show the handheld device provided a refractive error not significantly different from the refractive error recorded by the other methods.[5]

Photo: Nathan Stevens, OD

Handheld tonometers, such as the iCare, can make taking IOP readings a breeze.

Although older portable autorefractors were not useful for pediatrics, the same may not be the case with newer technology.[6] New research found handheld autorefractors agree well with cycloplegic retinoscopy and noted they could be

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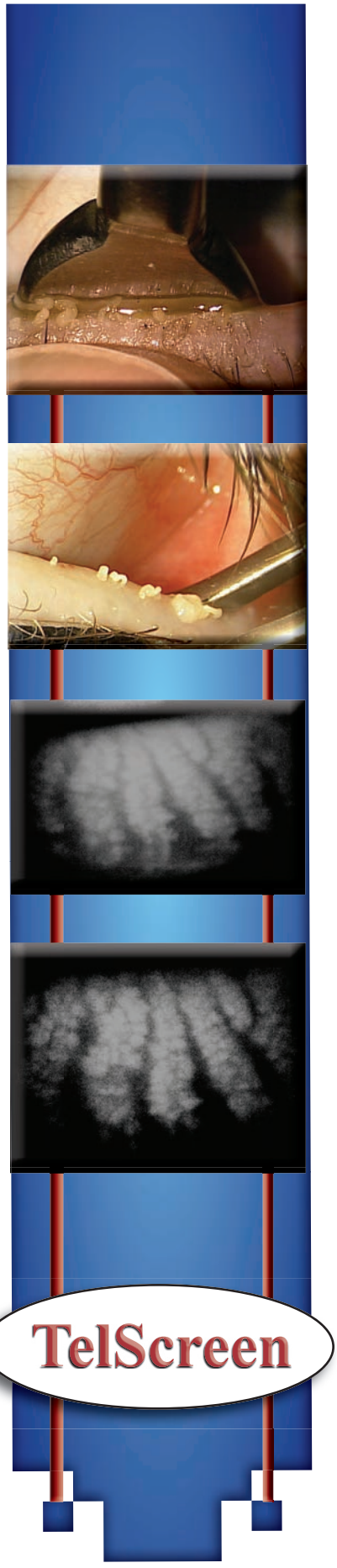

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useful in screening pediatric patients ages four to 12, as they may eliminate the need for cycloplegia.[7]

Autorefractors are even becoming a smartphone accessory, which may be particularly useful in areas with limited access to eye care.[8] These newer technologies are also providing expanded capabilities such as built-in fogging procedures to control for accommodation.[9]

Phoropters. While there is no reason clinicians would need to replace their in-office phoropter with a handheld one, a portable option is a must when visiting patients beyond the clinic. Many phoropters can be portable with a travel stand, but newer designs that put the phoropter in the patient's hands removes the need for an extra stand.[10]

Tonometers. Many practices already use a handheld tonometer in practice. In one study, for example, the handheld PT100 tonometer (Reichert) was in agreement with traditional Goldmann applanation tonometry (GAT) within 3mm Hg or less in nearly 93% of eyes in normal patients.[11] The devices can be particularly helpful for special patient populations as well, such as pediatrics. Another study found handheld rebound tonometry readings were likely to be accurate for children with normal eyes, sparing children an examination under anaesthesia.[12] However, research does suggest handheld rebound tonometry frequently and significantly overestimates IOP compared with traditional GAT.[12]

Researchers also found acceptable agreement between GAT and the iCare handheld tonometer when testing adults, including control patients, automated-lamellar-therapeutic keratoplasty patients and Descemet-stripping-automated-endothelial keratoplasty patients.[13] The agreement was poor in penetrating keratoplasty patients and those with an edematous graft.[13] These researchers recommend clinicians suspect truly elevated IOP in patients with edematous graft when both GAT and handheld tonometry produce high IOP readings.[13]

Photo: Dan B. Shropshire, OD

The Tono-Pen is another handheld tonometer that allows clinicians to check a patient's IOP without bulky equipment or a designated testing area.

Corneal topographers. These are great for measuring corneal shape and screening for corneal dystrophies, degenerations and irregularities.[14,15] Researchers found readings from a handheld device were in agreement with traditional keratometry, but clinicians should perform topography in an upright position, as rotation can affect the data.[16]

Photo: Dan B. Shropshire, OD

Portable slit lamps are a must for examining patients unable to sit behind a traditional slit lamp or at a patient's bedside outside the clinic.

Retinal imaging. Although retinal imaging is integral to the diagnosis and management of myriad ocular and systemic conditions, access to fundus photography can be limited by patient morbidity, high equipment cost and shortage of trained personnel.[17] Portable options transcend these limitations and provide imaging capabilities regardless of patient—or practice—constraints.

Many investigators have studied the use of portable retinal camera screening for the progression of diabetic retinopathy (DR), for example.[17-19] One study assessed 301 patients with Type 2 diabetes using standard seven-field digital fundus photography and a portable smartphone-based retinal imaging system.[18] The investigators found the portable camera has substantial agreement with conventional retinal photography and is a good tool for screening and diagnosing DR and sight-threatening DR.[18]

Another study suggests portable retinal cameras, in conjunction with telemedicine technologies, can provide significant automation, sensitivity, specificity, portability and miniaturization for point-of-care diabetes diagnostics.[19]

Smartphone-based fundus cameras also allow clinicians to capture high-quality, widefield images that can be stored on the device and evaluated on site or transmitted for remote assessment.[17]

Optical coherence tomography (OCT). This is quickly becoming integral to any medical practice, but its footprint can be a limiting factor for some practices. Luckily, research suggests portable OCT imaging is comparable to images obtained with conventional OCT and can be useful for pediatric patients in particular.[20] Investigators were able to obtain useful images from all 30 patients, ages seven months to almost 10.[20]

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The portability allowed the researchers to obtain imaging without sedation in children as young as three years of age, and all children tolerated the tests well.[20]

Other investigators looked at the use of a handheld spectral-domain (SD) OCT device during macular surgery and found it is an efficient method for visualizing macular pathology.[21] Although a small study of just eight patients, it suggests portable SD-OCT may help confirm or identify disease in some cases.[21]

Slit lamps. Portable slit lamps have been touted as indispensable in certain situations since the '40s, as they can provide a "flexible aid in the diagnosis of more superficial pathologic changes, as well as a means of rapid and satisfactory inspection of minute foreign bodies."[23] Today, they can be used in the clinic for patients unable to sit behind a traditional slit lamp or at a patient's bedside outside the clinic.

One study found smartphone ophthalmoscopy agreed substantially with slit-lamp examination when two masked glaucoma specialists estimated vertical cup-to-disc ratios in 110 patients with ocular hypertension or primary open-angle glaucoma.[24] The researchers concluded smartphone ophthalmoscopy can be beneficial in glaucoma screening, especially in low-resource settings.[24]

Pachymeters. Researchers investigated the accuracy and repeatability of the SP-100 Handy (Tomey) portable pachymeter by assessing the corneal thickness of 57 right corneas of patients ages 18 to 44. They compared the results with readings obtained from two conventional pachymeters and found the handheld device provided reliable corneal thickness measurements, even when considering a single reading.[25]

While operator dependability is a concern for many devices, one study found a handheld pachymeter provided excellent intra-observer repeatability and inter-observer reproducibility by an ophthalmic nurse and an ophthalmologist.[26]

Electroretinogram (ERG). Even though electrodiagnostic testing isn't common in an average optometric practice, a portable full-field ERG may eventually make it more feasible. Clinicians could potentially assess signs associated with many ocular conditions, such as DR, glaucoma and acquired and inherited retinal diseases, without shuffling patients around.[27]

Smartphone-based devices and apps. Many instruments are now integrated with smartphones, making them even more accessible. Practitioners can use their own device to capture anterior segment and retinal images, and even obtain autorefraction readings.[5,8,17,19] In addition, the selection of ophthalmic apps is exploding to provide many testing and patient education options with just a swipe of a touchscreen. Amsler grid and color testing, vision therapy, blink exercises and basic eye exam readings are all available in an app to help practitioners test, follow and educate patients in the office and beyond.[26,28]

With ophthalmic technology, bigger isn't always better. Downsizing equipment can reap significant rewards, especially for optometrists working with tight spaces and beyond their four walls. Smaller instruments can help to improve

Beyond Brick-and-mortar

In his strictly mobile nursing home practice that travels throughout the area surrounding Dallas, Texas, Dan B. Shropshire, OD, knows first-hand the wonderful benefits of handheld instruments in patient care. He and his highly trained team of optometrists and assistants provide exceptional and comprehensive eye care for this terribly underserved and growing demographic.

"When I started one day a week back in 1996, there were not many handheld equipment choices," says Dr. Shropshire. "But I loved my nursing home practice so much that I sold my private practice within six months and went full time into long-term care."

That kind of dedication took some imagination more than 20 years ago, when equipment ran big and bulky. But today, his practice has benefited from recent technological advances.

"I began using a spot retinoscope with handheld lenses for my refractions, Schiotz tonometer for pressures and a corded BIO," Dr. Shropshire says. "After acquiring my Tono-Pen, Nikon Retinomax and my battery-operated slit lamp and BIO, I was able to perform an exam very similar to any office."

When deciding on what equipment to incorporate into his practice, Dr. Shropshire focuses on what will ensure his patients receive the care that is as close to an in-office exam as possible. He also has to take into account his specific patient population: geriatrics.

"There are many choices for each piece of equipment that I need," he says. "Not all are appropriate for a geriatric population; my patients have poor fixation and cooperation. Add in poor positioning from kyphosis or being bed-bound, and these complexities will eliminate many of the choices out there. Trial and error will winnow out the practical from the impractical."

Regardless of equipment choice, the most important part of the equation is patient satisfaction. "I have been told time and again that my exam was the most thorough exam they ever had." The secret to his success? The tools at his disposal in today's market.

"Without handheld instruments I would be going back to the '50s—not so bad for movies and car designs, not good for eye care," Dr. Shropshire jokes. "My practice runs as well as most practices, maybe even better, because of the convenience of handhelds. Without them it would be impractical."

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Dr. Wimbish is co-founder and clinical director at Allen Eye Associates. He is interested in fitting medically indicated contact lenses for conditions such as keratoconus, corneal transplants and post refractive corneas. He primarily focuses on managing ocular surface disease within the multi-doctor private practice. He was voted Best Optometrist and Optometric Practice in his community the past four years.

He would like to thank Dan Shropshire, OD, for his contributions to this article.

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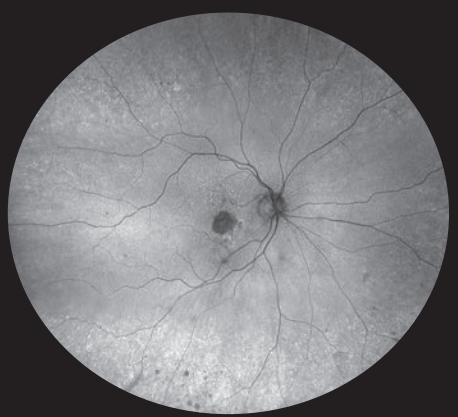

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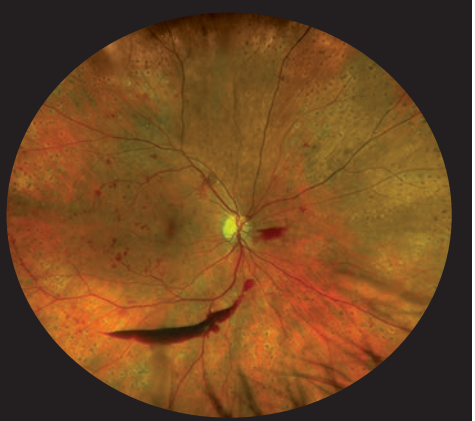

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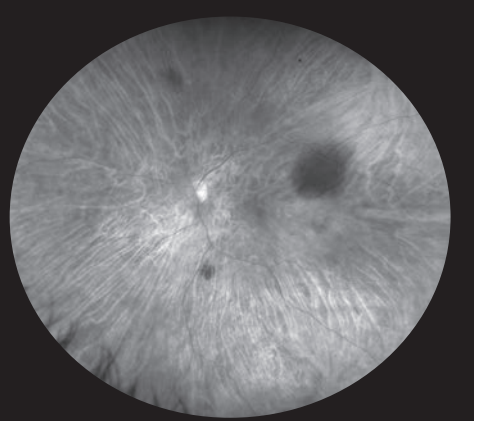

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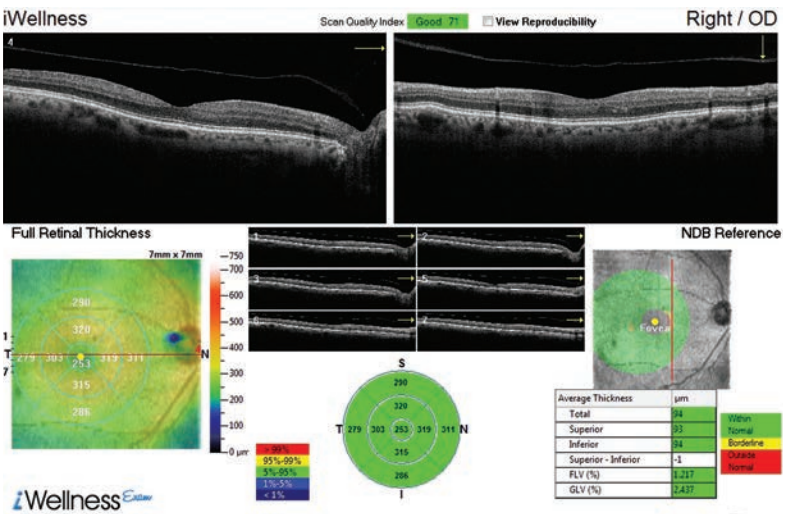
PEER-REVIEWED RRA ARTICLE

How Early Diagnosis Can Improve AMD Outcomes

Functional tests are giving optometrists the ability to diagnose earlier and more precisely than ever. **By Jeffry Gerson, OD**

Managing age-related macular degeneration (AMD) is a significant focus in primary care optometry, and no responsibility of ours is more urgent than identification. Though we may comanage active disease with the retina specialists who administer anti-VEGF injections, our most vital activity is to maintain vigilance for AMD in vulnerable patients. However, many cases go overlooked due to the absence of structural findings. In fact, a recent study reveals that both optometrists and ophthalmologists miss AMD approximately 25% of the time.[1] In addition, 30% of the undiagnosed eyes in that study had large drusen, a known risk factor for wet AMD.[1]



This OCT screening shows a patient who displayed no macular pathology but for a detached vitreous face. The macular exam showed no visible drusen. However, this patient did have early functional signs of AMD when we tested dark adaptation.

There's plenty that optometrists can do to help patients with early-stage disease. Clinically, many have undetected functional deterioration that could yield to diagnostic testing. With an early diagnosis, ODs can take potentially life-altering steps long before patients hit the intermediate stage and are forced to struggle with vision loss. The longer clinicians can keep these patients from advancing to wet AMD and needing injections, the better off they will be.

This article describes the value of diagnosing AMD in its earliest stages using functional tests and how you can bring these methods into your clinic today.

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[1] Koob TJ, Lim JJ, Zabek N, Massee M. 2014. Cytokines in single layer amnion allografts compared to multilayer amnion/chorion allografts for wound healing. J Biomed Mater Res Part B 2014:00B:000-000

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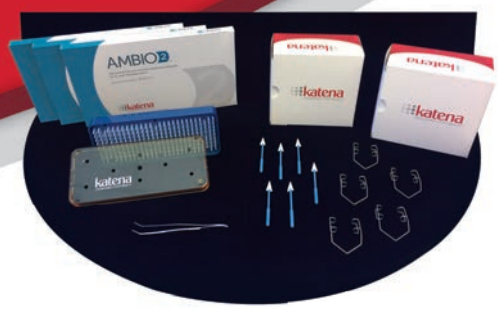

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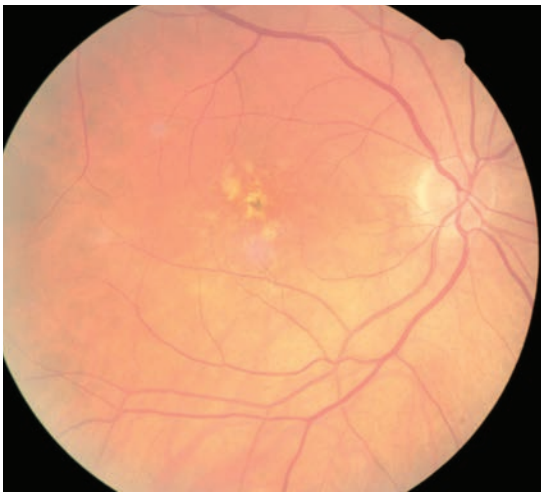

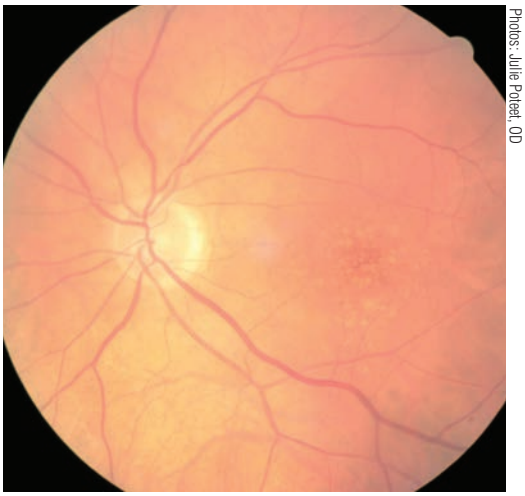

Photos: Julie Poteet, OD

At left, this eye qualifies as intermediate AMD per the AREDS classification system. At right, the patient qualifies as intermediate AMD and is progressing to the advanced stage due to the pigment clumping centrally.

Research Shows AMD is Going Unnoticed

A recent study reveals how frequently dilated eye exams miss incidents of AMD.[1] The cross-sectional study, which included 1,288 eyes in 644 adult patients who were enrolled in the Alabama Study on Early Age-Related Macular Degeneration (ALSTAR), revealed that doctors miss AMD about 25% of the time.[1]

The authors set out to determine the extent to which AMD is underdiagnosed by ODs when the disease is actually present. In the study, they reviewed the medical records of adults 60 years or older.[1] To be eligible, the patient's medical record from the most recent comprehensive dilated examination could not indicate a diagnosis of AMD in either eye, nor could the medical record notes contain terms that signified the signs of AMD.

Each patient in the ALSTAR study had digital color fundus photos taken, which were reviewed by masked, trained graders who determined the presence or absence of AMD findings according to the Clinical Age-Related Maculopathy Staging system.[2] The types of AMD-associated lesions also were noted.

The results revealed that one in four eyes studied was not diagnosed with AMD during the dilated fundus examination, despite having macular characteristics indicative of AMD in the fundus photos. Approximately three quarters of the 320 undiagnosed eyes had 10 or more small drusen (77.8%) or intermediate drusen (78.1%), or both, and 96 (30.0%) of the undiagnosed eyes had large drusen.

As this study reveals, even the most well-trained primary eye care doctors can easily miss AMD. Missing this diagnosis can have serious consequences for patients and result in severe vision loss.

Also, the prevalence of undiagnosed AMD in the study was not any different between ophthalmologists and optometrists.

Although the investigators of this study admit the reasons for the missed diagnoses remain unclear, they point out that improved AMD detection strategies may be necessary in primary eye care offices, since many of these patients would have been candidates for therapeutic intervention.

In short, current technology may lead to a sense of complacency and does not always enable us to detect AMD early enough. This is potentially overcome when dark adaptation testing is available.

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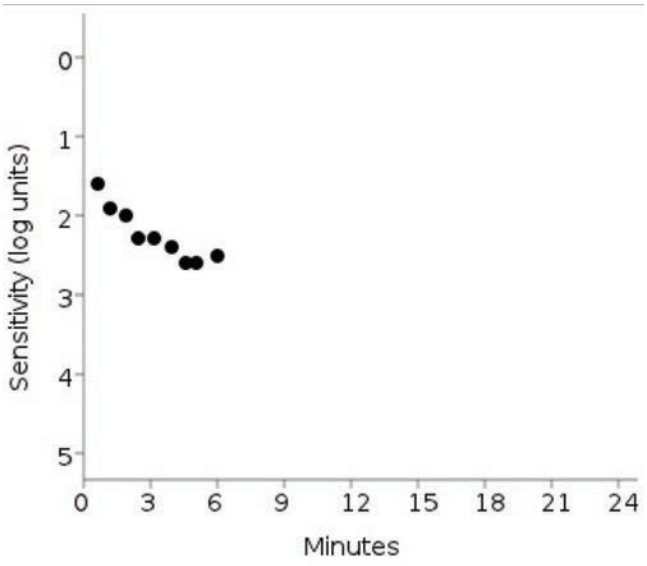
tomography (OCT), which is tremendously helpful in imaging an array of retinal diseases, including AMD. However, OCT looks only at structure. In fact, OCT's great precision in visualizing structural change has likely tipped the priorities of eye care providers more toward this vein of diagnostic data at the expense of monitoring the patient's functional visual status. Depending on the interpretation, an OCT image of a patient with a few small drusen may be misleading and can potentially underestimate the extent of disease. By the time photoreceptor ellipsoid layer thinning is visible on OCT, macular function is likely greatly impaired.[2-4]



This print out shows the failed dark adaptation screening test of the same patient imaged using OCT on page 50. Anything greater than 6.5 minutes is indicative of impaired dark adaptation time. Also, the patient's fixation rate was tracked at 0% in this patient.

Histopathological and clinical research suggests that subclinical AMD has both structural and functional consequences before drusen are even evident in clinical imaging.[2-4] Most current technology has not enabled us to detect AMD soon enough in the vast majority of cases.[5,6] Up to 78% of AMD patients have substantial, irreversible vision loss at first treatment, including 37% who are already legally blind in at least one eye.[5,6]

But some technologies are showing promise for more advanced testing. For example, functional testing can detect dark adaptation impairment before AMD is clinically evident, which is helpful considering the disease appears to manifest through impaired dark adaptation before drusen are visible.[7,8] In a recent study, subjects with impaired dark adaptation were twice as likely to develop clinically evident AMD and eight times as likely to advance beyond the earliest stage of AMD.[7] Some clinicians find reduced contrast sensitivity to be another early signal of incipient disease.

Usually expressed as "night vision difficulties," impaired dark adaptation is often among the first detectable consequences of AMD and a method of identifying patients with potential subclinical disease.[8] Dark adaptation can be used to evaluate whether small drusen are focal deposits or the visible tips of the lesions caused by AMD.[8]

A nuanced understanding of the histopathology of this vision-depleating disease, combined with a keen knowledge of its functional consequences, will provide optometrists the clinical inght necessary to fight AMD earlier than ever before.

Histopathology

Research shows that retinal pigment epithelium (RPE) cells deposit locally generated cholesterol beneath the RPE cell layer and in

Opinion: Get Off the "Stage"

If we could easily identify patients and improve outcomes, why isn't every doctor doing it? AMD labels and definitions are partially responsible for the current atmosphere of confusion and have prevented us from looking critically at ineffective practice protocols. For example, consider the limited value of the clinical staging of AMD in optometric practice. If, like me, you were taught that "Stage 1 AMD" is "No AMD," be aware that this is dangerously misleading. Stage 1 AMD is, in fact, AMD. Even though noticeable central vision loss has not yet occurred at this early stage, it one day may, and by the time that day comes heroic measures will be necessary.

The clinical staging of AMD is misinterpreted and has led to a defeatist view of what we can achieve in terms of more effective management of dry AMD. By the time we can visualize change, damage has already been done.

Bruch's membrane before drusen are formed.[2] With disease progression, this cholesterol continues to accumulate, resulting in focal areas that are sufficiently thickened enough to be identified as drusen.[2-4] Essentially, drusen caused by AMD are merely the "tip of the iceberg" of the earliest lesions caused by AMD.[2-4] And more dysfunction is present than is apparent based on the clinical or even OCT appearance of drusen.[2-4]

This cholesterol accumulation causes three primary insults to the retina: (1) inflammation, (2) oxidative stress and (3) disruption of oxygen and nutrition supplied to the outer retina.[3] For instance, transport of vitamin A—critical for rod-mediated dark adaptation—is a functional role disrupted by AMD.[3] Research shows that this vitamin A availability disruption dramatically slows dark adaptation.[4]

Additional research shows dark adaptation function is impaired from the earliest stages of AMD, retinitis pigmentosa and other retinal degenerations/dystrophies, with increasing impairment as the diseases progress.[1,4,5] By prevalence, the most common disease which causes dark adaptation impairment is AMD and second is retinitis pigmentosa, which is typically diagnosed before the age of 50.[1,4,5]

How Adaptometry Works

Dark adaptation testing may allow clinicians to detect subclinical AMD at least three years earlier than it is clinically evident, according to investigators.[4,5,9] Unlike macular pigment optical density (MPOD) or genetic testing, dark adaptometry does not measure the risk of AMD; it is diagnostic of AMD, meaning that the disease is already present.

Dark adaptation is a rod-mediated measure, using the rod intercept (RI), and is not dependent on cones. RI is the number of minutes it takes for the eye to adapt from bright light to darkness and can be measured with the commercially available AdaptDx dark adaptometer (Maculogix). Obtaining a patient's RI is easy both from a technical perspective and for the patient. A bleaching flash is used and then responses to stimuli are recorded until the RI is achieved. Like an automated visual field, all the technician needs to do is encourage fixation and attention. The patient's experience is similar to a threshold visual field and may take anywhere from a few moments to approximately 20 minutes.

The RI number provides a clear and objective measurement of retinal function with 90% sensitivity and specificity.[10] An RI of less than 6.5 minutes indicates normal dark adaptation consistent with healthy photoreceptor function.[10] An RI higher than 6.5 minutes indicates impaired dark adaptation, most often due to AMD, unless there is evident retinal degeneration or a systemic vitamin A deficiency.[10]

Helping Early AMD Patients

Many changes to an AMD patient's lifestyle can help avoid further central vision loss and retinal damage. Once diagnosed with early AMD, optometrists can encourage patients to take the following steps:

More frequent examinations. Assuming that nothing can be done to effectively intervene at the earliest of stages of AMD ignores the value of informed follow-up. Although more research is needed to most effectively address subclinical AMD, patients with early stage disease should still be monitored more closely because the transition from dry to wet AMD can happen rapidly and, if left untreated, can lead to vision loss fairly quickly.

Moving from a 12- to a six-month follow-up interval is useful for monitoring disease progression. More frequent visits provide increased opportunity to detect choroidal neovascularization (CNV) before visual acuity loss. Often, home monitoring by Amsler grid is ineffective, or the patient defers reporting symptoms between office visits. ODs can also consider shortening the follow-up visit interval to every three or four months for patients who are fast progressors or are at high risk of CNV. As a result, AMD patients are likely to benefit from a much better outcome than they would have without being closely monitored.

Stay healthy. Following a healthy diet, exercising regularly and maintaining overall health are sound goals for all patients.[11]

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Counseling patients on lifestyle changes is easy and impactful. For example, smokers must be informed of the dramatic escalation of consequences tied to their habit. Much like someone who's been diagnosed with prediabetes might choose to make dietary changes, most patients diagnosed with AMD will want to take action. With some guidance, clinicians can empower patients to take meaningful steps in the right direction.

One study found that women who followed a healthy diet, engaged in physical exercise and avoided smoking had substantially lower risk of early AMD compared with women who did not follow these healthy lifestyles.[12] Epidemiological studies have found substantial benefit from higher dietary intake of foods rich in omega-3s, especially DHA.[13] A Mediterranean diet is another consideration, as studies suggest those who regularly consume a Mediterranean-style diet carry an overall lower risk of developing advanced AMD compared with those who regularly consume a traditionally Western diet.[14]

Advocate for an active lifestyle. Research shows this can reduce the risk of progression to CNV.[15-17] One study found those who participated in cardiovascular exercise of any intensity three or more times per week had one third the incidence of CNV compared with those who exercised less than three times per week.[17] For those who walked one or more blocks per day, the incidence of CNV was half compared with those who walked less than one block a day.[17]

Recommend supplements and blue light-blocking lenses. Although AREDS showed no benefit to the use of AREDS supplements for patients who have less than intermediate AMD, further research on this specific population is needed.[20-22] Some doctors may also recommend carotenoids, omega-3 and blue light-blocking lenses.[20-22] These recommendations may not only prove beneficial in slowing disease, they may also improve visual function.[5,7] More importantly, an early diagnosis of AMD provides our patients the freedom to choose whether they want to invest in supplements. Evidence strongly suggests that patients should be prescribed nutritional supplements because, on average, treated patients have better outcomes than untreated patients.[9,16,17]

Photos: Paul Karpecki, OD, and Diana Shechtman, OD

At top, this patient shows signs of early AMD associated with drusen and areas of RPE disruption. This patient is already at risk for vision loss. With dark adaptation, ODs may be able to begin management of AMD before this clinically evident stage is even reached, in the hopes of preventing progression to the advanced stages of AMD, as seen in the lower two images.

Three primary options for the selection of an appropriate nutritional supplement exist. The first is to prescribe a macular pigment supplement (i.e., the xanthophylls: lutein, zeaxanthin, meso-zeaxanthin). The second is to prescribe a supplement containing both xanthophylls and antioxidants, including zinc and vitamins E and C (e.g., an AREDS2 supplement).

The third option is to prescribe a xanthophyll supplement to patients with subclinical and early AMD and a xanthophyll-antioxidant combination supplement to patients who have or progress to intermediate AMD.

The relative merits of each option are debatable, and knowledge continues to expand about the many factors that contribute to AMD progression.

Timely referral to a retina specialist. Early knowledge that AMD is present can trigger a change in management. For example, follow-up visits are likely to include careful funduscopic examination, fundus photography and OCT scans, as necessary, and repeat dark adaptation testing to gauge for progression.[23]

This careful approach helps ensure patients are referred to a

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Opinion: Use New Techniques, but Keep the Old

By Jay M. Haynie, OD

Dark adaptation does allow optometrists to measure the functional component of macular disease, including AMD; however, in my opinion, a patient who performs poorly on this test should not necessarily be offered AREDS supplements. Doctors can use dark adaptation testing as a factor in determining risk and to educate patients about AMD monitoring and prevention.

Nutritional supplements have been a part of eye care for decades now and we have some great scientific data on the type of patient that may benefit from supplements, such as AREDS, and which patients showed little to no benefit in the larger studies—embracing new testing techniques doesn't change that. Evidence shows AREDS supplements have been beneficial for patients with phenotypic findings, such as medium-to-large drusen, although more research is needed on whether they can truly prevent the onset of clinical AMD. In addition, investigations raise controversy regarding the use of high levels of zinc contained in supplements. Furthermore, if a patient has a genotype which includes two complement factor H alleles and no ARMS2 allele, zinc may actually contribute to the progression of the disease process.[1]

This prompts the question as to whether nutritional supplements which contain zinc should be advised for patients who have a low dark adaptation performance. There has been enough controversy regarding the antioxidant zinc that prior to putting a patient on a supplement for what may be decades we must think about potentially doing harm to those individuals.

Dark adaptation does have a role to play in optometry and it could be used as a screening tool for patients who have a strong family history of AMD, patients who have subjective symptoms specific to night vision loss, or patients with phenotypic changes suggestive of early AMD (small or intermediate drusen). Upon conclusion of the test, if the patient is found to have poor dark adaptation, my clinical theory and thoughts in private practice would be to begin counseling them about lifestyle changes. These discussions must include the modifiable risk factors of AMD which include good daily nutrition, use of ultraviolet protection, cholesterol reduction, blood pressure monitoring and tobacco cessation.

If either the doctor or the patient is considering a supplement, I recommending starting with a carotenoid.

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retinal specialist for more aggressive treatment, such as injections, as soon as it becomes necessary.

With an earlier diagnosis, optometrists can do more than let AMD run its course and eventually rob patients of their sight. Though no one diagnostic tool is sufficient on its own, given the complexities of AMD's pathophysiology and the wide variety of presentations possible in the preclinical phase of development, adding these tools and techniques to our practice allows ODs to keep a closer eye on progression and respond promptly to changes. Improved outcomes begin in the optometrist's chair—it is well within optometry's scope of practice to preserve AMD patients' vision and, ultimately, improve outcomes. ■

Dr. Gerson practices at Grin Eyecare in Olathe, Kan.

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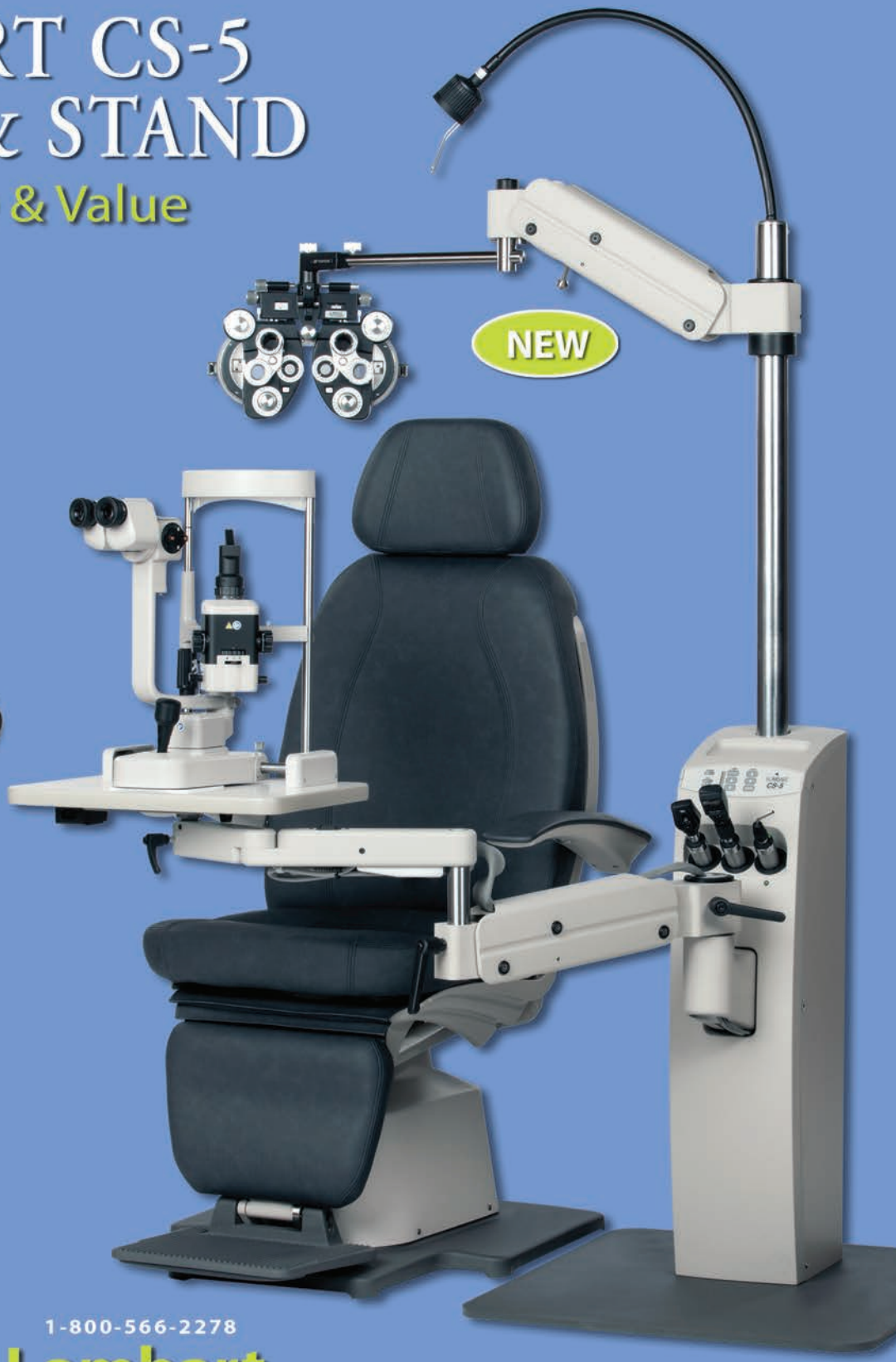

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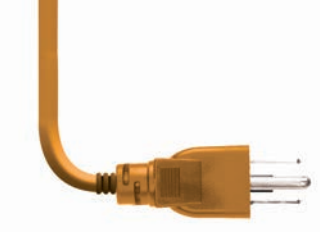

PEER • REVIEWED • PRA • ARTICLE

Modernize Your Exam of Glaucoma Patients and Suspects

Additional testing can help you customize your glaucoma workup.

By Jarett Mazzarella, OD, and Justin Cole, OD

With the US prevalence of glaucoma expected to rise to roughly 6.3 million by 2050, optometrists must be vigilant in screening patients to catch even the earliest signs of disease.[1] Today's technologies are integral to assessing patient risk and providing the data necessary to diagnose and treat long before permanent visual loss. Clinicians can use any number of tools—including corneal hysteresis (CH) testing, OCT angiography (OCT-A), 10-2 visual field testing and even electrodiagnostics—along with their routine workup to aid in glaucoma diagnosis and management. Here, we discuss how clinicians can implement these ancillary tests on a patient-by-patient basis to better diagnose and monitor glaucoma.

Corneal Hysteresis

The viscoelastic properties of the cornea and its ability to absorb energy are represented by CH, and low CH is a risk factor for glaucoma and progression.[2,3] Currently, the only commercially available instrument to measure CH, based on the definition and applying bidirectional applanation tonometry, is the ocular response analyzer (ORA) from Reichert Technologies.[2]

An alternative device, the Corvis ST (Oculus), uses corneal deformation by Scheimpflug imaging or OCT, via a combination of corneal topography, bidirectional applanation and high-speed photography.[4] Although this device has FDA clearance for sale in the United States, it is not as well studied as the ORA. However, the Corvis ST does provide video of the cornea deformation during testing, and it captures more than 4,000 images per second, which may reduce variables of poor fixation and reduced tear film while increasing the intraocular pressure (IOP) measurement accuracy.[5] Other technologies evaluating corneal biochemical properties include electronic speckle pattern interferometry, ultrasonic elastography, high-frequency ultrasonographic analysis and Brillouin microscopy.[6]

For the ORA more specifically, its value denotes the pressure difference between the applanation of the cornea by an air jet and the recoil outward force by the corneal tissue, measured by an infrared laser to measure CH. In addition to CH, ORA also provides a calculated measurement of the corneal resistance factor (CRF), representing the elasticity of the cornea. Research shows CH and CRF are reduced in ocular conditions such as Fuchs' dystrophy, as well as systemic conditions such as diabetes and lupus. Both measurements are also lowered in cases of corneal ectasia and after refractive surgery.[7]

The ORA is also designed to estimate IOP.[8] It acquires both the corneal-compensated IOP (IOPcc) and the Goldmann IOP (IOP_G). The IOPcc may be less dependent on corneal thickness compared with applanation tonometry, and research shows the IOP_G is comparable with Goldmann tonometry.[6,8]

Studies demonstrate that CH is a repeatable value that typically shows correlation between the right and left eye and no variation between sex.[9,10] In normal eyes, CH has no significant diurnal variation, but it does decrease with age similar to central corneal thickness (CCT).[11,12] Studies estimate the average CH normative value ranges from 10.07mm Hg to ± 1.61mm Hg.[13] Obtaining a baseline CH value prior to treatment is particularly important, as CH tends to increase as IOP decreases with ocular hypotensive therapy.[1,2,14] Differences in CH based on ethnicity remain poorly defined.[11,12]

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%

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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

Concerning glaucoma and disease progression, research shows reduced CH is associated with a greater optic nerve head cup depth and size.[15] Studies also demonstrate that patients with increased CH values have more optic nerve deformation when eye pressure was high, thereby allowing the ocular tissue to dissipate the pressure, providing protection against mechanical damage to the optic nerve.[16]

Patients with glaucoma tend to have decreased CH and CCT compared with normal subjects, and reduced CH has been correlated with progression of visual field loss.[17] Studies also confirm that patients with reduced CH have a faster rate of glaucoma progression.[18] When CH is not similar in both eyes of glaucoma patients, research shows a faster rate of progression in the eye with lower CH.[19,20] CH may be predictive of how a patient will respond to ocular hypotensive therapy, as patients with low CH may have a larger IOP reduction compared with patients with high CH values.[14] Therefore, if a patient with high CH takes an IOP-lowering medication, the IOP reduction may be less than is seen in an eye with low CH. This does not mean the drop is not efficacious, but the target IOP should be reassessed based on the cornea's biomechanical properties.

CH may be more strongly associated with glaucoma risk and progression than CCT, which has been standard of care in glaucoma evaluations since the Ocular Hypertensive Treatment Study.[18,21] The exact mechanism of action between CH and glaucoma pathogenesis is not completely understood, but research indicates it may play a viable role in glaucoma management.[12]

Table 1. SD-OCT-A Advantages and Limitations[1,2]

Advantages	Limitations
• Fast acquisition • Noninvasive (no dye needed) • Captures volumetric scans • Able to segment layers of the retina/choroid (localize depth) • Easily repeated • Can visualize both retinal and choroidal vasculature • Accurate in size and location of defined scanning area	• No quantifiable measurement of flow (units) • Susceptible to artifacts (specifically shadow projections from inner retinal layers and vessels to deeper retinal and choroidal layers) • Susceptible to motion artifacts • Limited field of view • No reference data • Cannot detect leakage as with FA/ICG • Cannot detect slow blood flow areas

1. Akil H, Falavarjani KG, Sadda SR, Sadun AA. Optical coherence tomography angiography of the optic disc; an overview. J Ophthalmic Vis Res. 2017;12(1):98-105.
2. de Carlo TE, Romano A, Waheed NK, Duker JS. A review of optical coherence tomography angiography (OCTA). International J Retina and Vitreous. 2015;1:5.

OCT-Angiography

Ocular blood flow (OBF), oxidative stress, reduced axoplasmic flow and genetics have all been implicated in the pathogenesis of glaucoma.[22] Research shows that OBF tends to be lower in glaucoma patients, specifically normal-tension glaucoma, compared with healthy controls.[23] The result of low OBF is reduced perfusion pressure, which leads to reduced oxygen supply and release of hypoxic mediators. This mechanism may be secondary to autodysregulation within the vascular system.[23] Here, OCT-A technology can help to uncover an OBF impairment to the optic nerve and peripapillary retina. Research demonstrates that reduced OBF is a potential risk factor for glaucoma development and progression, as blood flow to the optic nerve, derived from the central retinal artery and posterior ciliary arteries, can be reduced in glaucoma patients (*Table 1*).[24-27]

However, OBF was difficult to quantify, as the peripapillary capillary network is poorly characterized with fluorescein angiography.[28] OCT-A now provides a view into the microcirculation of the optic nerve and peripapillary retina, confirming an association between glaucoma and the severity of OBF impairment.[22] This may provide a means of detecting the disease in its earliest, "pre-perimetric," stage.

While a dense microvascular capillary network exists around the optic nerve head in normal eyes, glaucomatous eyes have delicate capillaries that can be easily attenuated and disrupted, leading to capillary dropout.[29] Disc flow index and vessel density values represent quantifiable measures of perfusion around the optic nerve and are derived from algorithms using OCT-A. These values—although not currently a component of commercially available OCT-A software in the United States—could potentially help establish a normative standard data set to differentiate between normal and glaucomatous eyes, as well as stratify damage in glaucoma.[25]

Research shows a direct correlation between optic nerve perfusion and structural and functional damage in glaucoma.[30] Specifically, reduced flow index and vessel density measurements correlating to reduced peripapillary and optic nerve perfusion are associated with increased visual field mean deviation and OCT retinal nerve fiber layer (RNFL) and ganglion cell-inner plexiform loss.[25,31]

Using OCT-A, investigators may better understand whether OBF causes glaucoma or if it is a structural manifestation of the process. Researchers know microvasculature dropout occurs in the peripapillary retina in the location of RNFL defects in glaucomatous eyes.[32,33] The volume of capillary loss and optic nerve damage in

glaucomatous eyes is proportional, suggesting the loss of peripapillary capillary structure is the result of the proceeding RNFL atrophy and glaucomatous neuropathy.[34]

Visual Field Testing

Research shows the central 16 degrees of the retina contains approximately 50% of the total ganglion cell distribution but only represents approximately 7% of the total retina area.[35] The advances in OCT technology have made it easier to see this more concentrated area of ganglion cells on a pseudocellular level with ganglion cell analysis (GCA). This is especially useful when evaluating glaucoma suspects with normal RNFL and no defects on a 30-2 or 24-2 (white on white) visual field. Recent studies indicate that early glaucomatous damage can occur within the central 10 degrees of the visual field with little to no change noted at the neuroretinal rim.[36] When thinning at the rim is present, it tends to be located at the inferior temporal neuroretinal rim, which projects to the inferior temporal region of the retinal ganglion cells

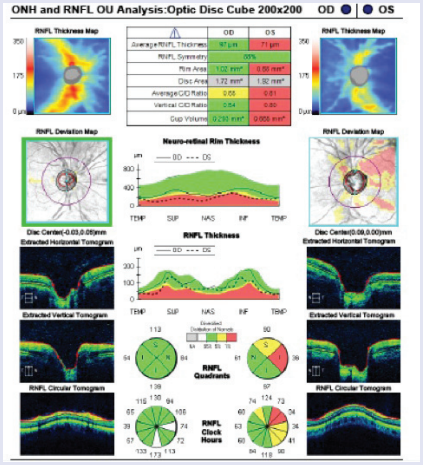


Fig. 1. The SD-OCT optic disc cube scan shows significant asymmetry in average thickness OD to OS. Superior temporal RNFL thinning is seen on the deviation map compared with normative data OS. The quadrant and clock hour plots show associated thinning OS.

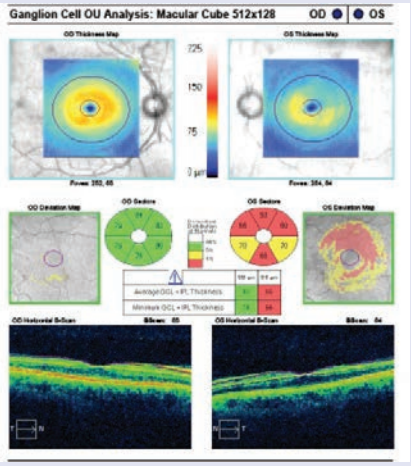


Fig. 2. The GCA shows significant asymmetry in both the average and minimum thickness. Depressions in the RGC-inner plexiform layer thickness are illustrated in all sectors OS, greatest superiorly, correlating with the RNFL findings OS.

Case 1

A 47-year-old male presented with best-corrected visual acuity of 20/20 OD and OS. Slit lamp examination revealed Krukenberg spindles on the corneal endothelium and midperipheral iris transillumination defects OU. His IOPs were 16mm Hg OD and 21mm Hg OS by GAT. Using 4-mirror gonioscopy, ciliary body band was seen 360 degrees, with 2+ pigment and a 35-degree angle approach with no anterior synechiae, recession or neovascularization of the angle OU. Upon dilation, we found his cup-to-disc ratios were 0.55 x 0.55 OD and 0.75 x 0.75 OS with pink and distinct margins OU.

We ordered ancillary testing, including SD-OCT (*Figures 1 and 2*), SD-OCT-A (*Figures 3 and 4*) and visual field 24-2

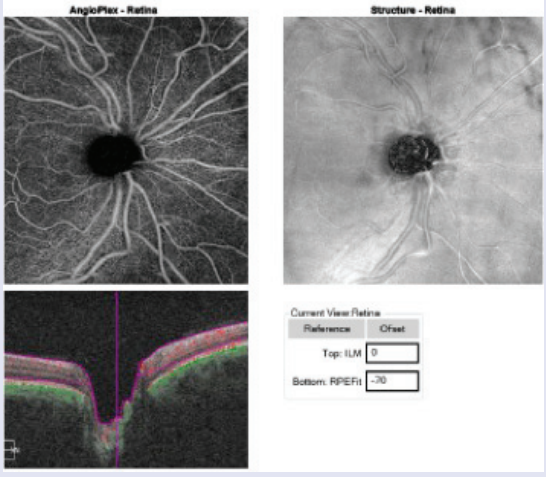


Fig. 3a. This OCT-A angioplex *en face* scan of the patient's OD (left) with segmentation of the retina shows a dense microvascular network surrounding the right optic nerve.

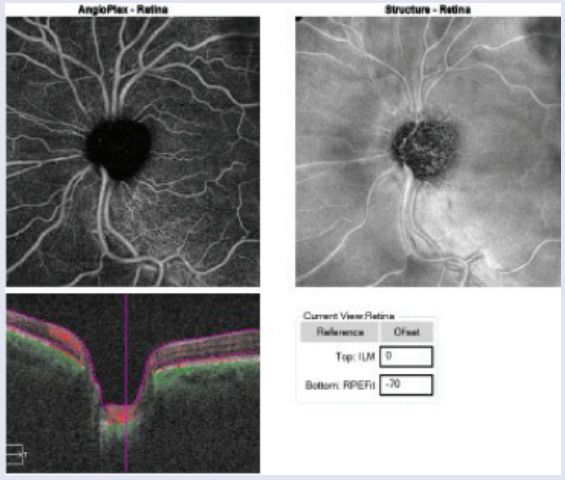


Fig. 3b. The OCT-A angioplex *en face* scan OS (right) with segmentation of the retina shows the significant dropout in the peripapillary capillary network greatest superior temporal to the optic nerve.

(RGCs) of the macula—known as the macular vulnerability zone (MVZ).[37] These RGCs are at a higher risk of damage due to their projection to the inferior region of the optic disc, which is more susceptible to damage. Typical visual field protocols fail to thoroughly and accurately detect subtle abnormalities in this region. Due to the projection of the axons from the RGCs within the MVZ, damage will consequently give the patient a superior paracentral scotoma—visible on a 10-2 visual field—classified as arcuate, comma-shaped or nasal step (*Figure 5*), which depicted structural but not functional findings of pigmentary glaucoma OS.

In this case of pre-perimetric, unilateral pigmentary glaucoma, peripapillary capillary density is affected in conjunction with the RNFL and ganglion cell structural findings prior to functional field loss.

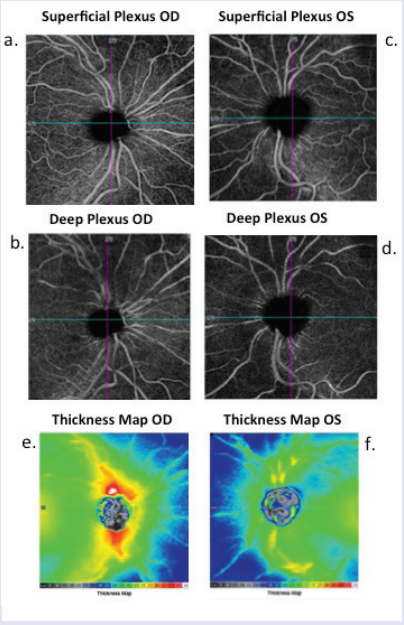


Fig. 4. The superficial and deep capillary plexus segmentation of the OD and OS show significant asymmetry in the peripapillary capillary densities OD>OS (a and b, c and d, respectively). This asymmetry is most evident in the superficial capillary plexus OS. Images (e and f) depict significant thickness asymmetry OD>OS.

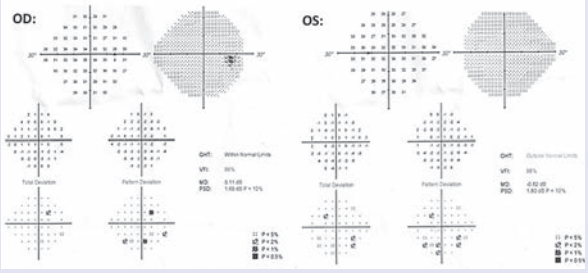


Fig. 5. These 24-2 visual field results show scattered point depressions OD and OS with good reliability. No glaucomatous cluster defects were noted OU.

Case 2

We saw a 64-year-old male with suspicion of glaucoma secondary to asymmetrical optic nerve cupping and mild ocular hypertension. We followed him with OCT of the RNFL and 24-2 visual field exams over a period of five years without treatment secondary to symmetrical RNFL exams and clean visual field exams OU. Visual acuity was 20/20 OU with normal anterior and posterior segment findings. IOP ranged from 22mm Hg to 24mm Hg in both eyes.

Over the course of one year, an RNFL slit defect formed inferiorly at the inferotemporal neuroretinal rim OD, noted on fundus photos (*Figure 1*) and OCT RNFL deviation map imaging (*Figure 2*). A 24-2 visual field was performed to ascertain if there was a correlation with this new defect (*Figure 3*). While repeat 24-2 testing showed no glaucomatous cluster defects (*Figure 4*), 10-2 visual fields (*Figure 5*) and OCT imaging (*Figures 6 and 7*) showed structural changes that warranted attention. Secondary to the appearance of the OD optic nerve, as well as IOPs in the 20s OU, we started the patient on a prostaglandin analog in each eye at night, which lowered his pressures to 15mm Hg OU.

The patient has since had selective laser trabeculoplasty with pressures in the low teens at an acceptable target range value OU.

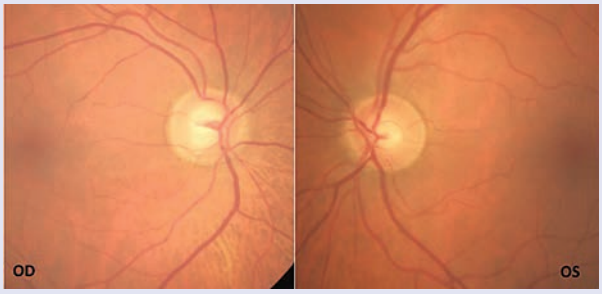


Fig. 1. The patient's fundus photos show inferior temporal rim thinning with vessel bayoneting within the optic nerve OD.

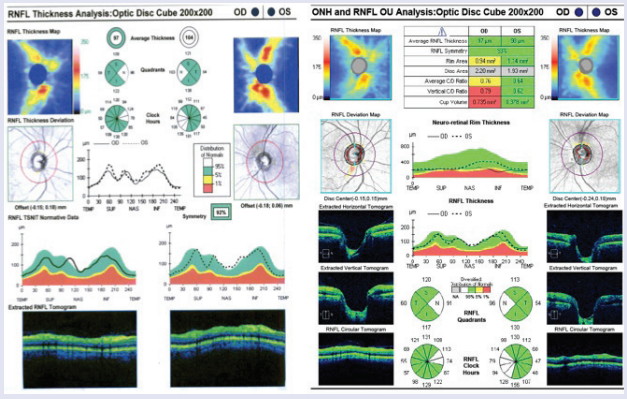
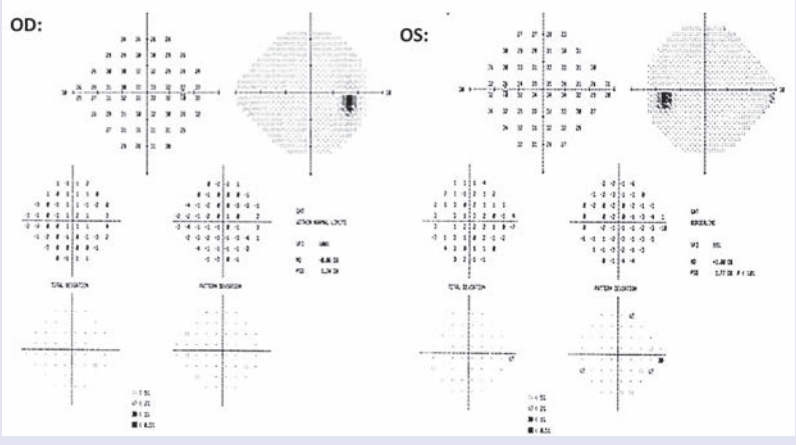


Fig. 2. Although there is a slit defect inferiorly, the RNFL analysis still interpreted this area as normal compared with the normative data. The OCT RNFL also depicts inferior temporal RNFL thinning on the deviation map and clock hour map OD compared with the normative data.



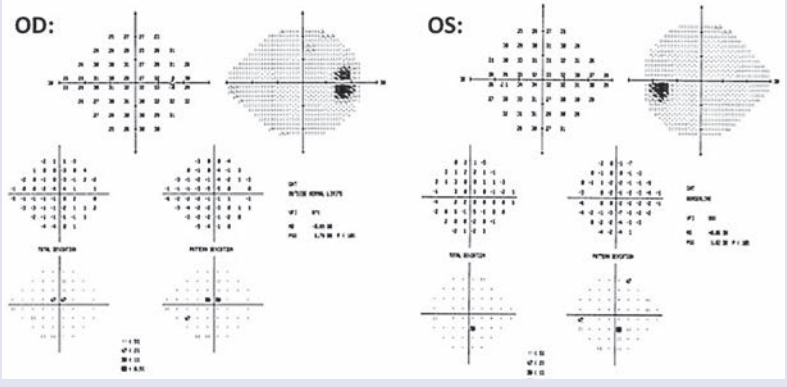


Fig. 3. Above, the 24-2 testing revealed no glaucomatous clusters OD and no correlation to the new RNFL defect OD. An inferior nasal cluster defect was noted OS and did not correlate to structural OCT findings. This cluster was found to be non-repeatable over successive fields.

Fig. 4. Repeated 24-2 testing shows point depressions and no glaucomatous cluster defects OU.

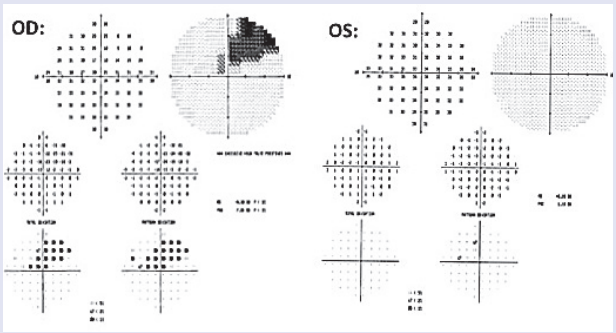


Fig. 5. A 10-2 central visual field was ordered to further investigate the 24-2 structural findings OD. It depicts a dense cluster superior/superior nasal to fixation correlating to the inferior temporal ganglion cell/inner plexiform thinning noted on GCA OD.

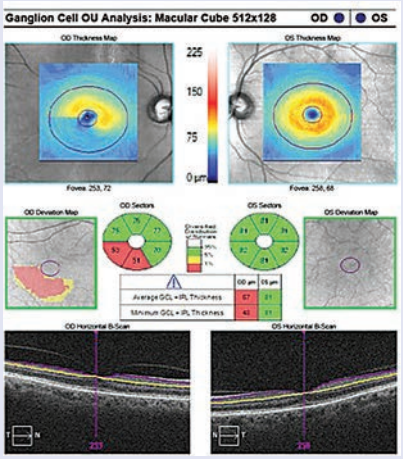


Fig. 6. The GCA illustrates inferior temporal sector RGC-IPL thinning OD correlating to the clock hour thinning of the OCT RNFL OD.

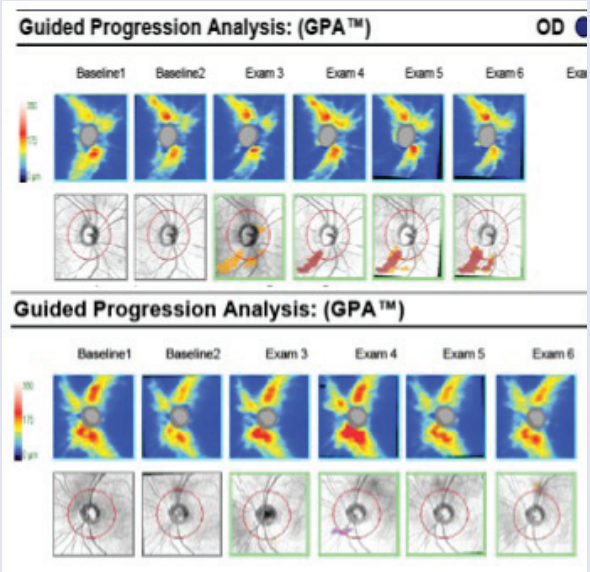


Fig. 7. Serial OCT RNFL progression analysis shows progressive RNFL thinning inferior temporally OD over successive exams compared with baseline one and two. No RNFL thinning is noted OS over six exams compared with normative data.

in appearance. The superior ganglion cell axons project to the temporal region of the optic disc, which is less susceptible to early glaucomatous damage, making an inferior paracentral visual field defect less likely.[37]

OCT GCA and 10-2 visual field testing does come with limitations. Because retinal anatomy varies from person to person and within ethnicities, limited normative databases can be an obstacle to proper diagnosis.[38]

In addition, segmentation errors due to concurrent macular pathology, specifically in the inner retinal layers, can confound the ganglion cell data. However, using 10-2 visual field testing in patients with a suspicious optic nerve appearance with unremarkable 24-2 or 30-2 visual field exams, in conjunction with OCT GCA, may help to rule out early macular damage due to early glaucomatous disease in a subset of glaucoma suspects.

Electrodiagnostics

Even in early glaucoma, research suggests one third to half of RGCs are dead or dysfunctional before defects are present on visual fields.[39,40] Electrodiagnostics may one day allow clinicians to detect this early dysfunction before the presence of defects, providing a predictive measure of conversion in glaucoma suspects. Several innovations are useful in research and may eventually be in clinical practice:

Pattern electroretinogram (pERG). Evidence suggests pERG can assist in recognizing early glaucomatous damage by assessing RGC layer dysfunction. A study comparing RNFL OCT with pERG revealed decreased pERG amplitudes correlated with reduced RNFL, indicating dysfunctional RGCs.[40] Researchers also speculate a lag time of 10 years exists before 10% of the RNFL is lost in early glaucoma.[41] In another study, glaucoma suspects had biannual pERGs over six years, approximately half of whom went on to receive IOP-lowering medication.[42] The pERG amplitudes of the untreated patients progressively declined, while the treated patients demonstrated stability or declined at a slower rate, suggesting lowering IOP may decrease the rate of RGC death or possibly reverse the RGC dysfunction in early disease.[42]

In patients with ocular hypertension, pERG testing can help predict stability or progression to glaucomatous optic neuropathy at least one year prior to visual field findings.[43] While OCT GCA shows thinning due to RGC death, pERG may be able to detect RGCs with reduced function at an early, reversible stage of the disease.

Multifocal electroretinogram (mfERG). Although mfERG shows promise for diagnosing early glaucoma, positive studies have not translated into clinical diagnostic use in human populations.[44,45] Investigators still struggle to quantify and extrapolate data to pinpoint RGC dysfunction in human populations.[12]

Full-field electroretinogram (ffERG). The photopic negative response (PhNR) of the ffERG may also correlate with RGC function. A study comparing RGC thickness with PhNR response shows a correlation within the central macula, the location of highly concentrated RGCs.[46] Another study also shows correlation with decreased PhNR amplitudes in ocular hypertensive patients.[47] These findings are promising for the early diagnostic stages of glaucoma, but more evidence is needed to apply this data in a clinical setting.

Although electrodiagnostic testing is not routinely performed in the primary eye care setting for glaucoma, it may one day serve as an adjunct tool in glaucoma management.

New diagnostic and imaging tools already aid in the early detection and treatment of glaucoma, as well as provide an avenue to better understand the pathogenesis of the disease. With more technology at our disposal, we must tailor our testing to each patient presentation by evaluating both modifiable and unmodifiable risk factors, patient demographics and long-term prognosis. Advancements help us better detect glaucomatous neuropathy at its earliest cellular manifestations, and we must consider the risk of functional visual loss over the patient's lifespan and weigh this against any negative effects of therapy on daily living. ■

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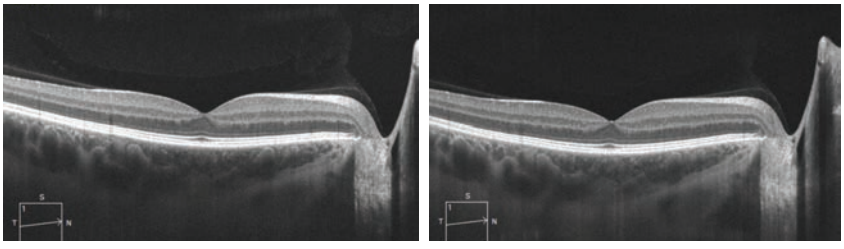
PEER • REVIEWED • PRA • ARTICLE •

UNDERSTANDING TODAY'S STATE-OF-THE-ART OCT TECHNOLOGY —AND ANTICIPATING TOMORROW'S

This family of devices has changed the way ODs diagnose and monitor disease. This guide explains when to use which application. **By Lee Vien, OD, and David Yang, OD**

Optical coherence tomography (OCT) technology has evolved drastically in the last two decades; advancing from time-domain (TD) to spectral-domain (SD) and now swept-source (SS). Each of these innovations allows for higher scan speeds, better resolution, improved sensitivity and fewer artifacts compared with earlier TD technology. These advances aren't simply helping eye care professionals see patients' anatomical structures more clearly; they're also helping us monitor the development of disease and guiding treatment accordingly.



SD-OCT line scan with (at left) and without enhanced depth imaging, which allows for visualization of the choroidal layers and the choroid-scleral interface.

Here, we provide an overview of the many OCT modalities and how you can apply them to the patients walking into your clinic.

Using OCT, Cover to Cover

Anterior segment OCT (AS-OCT) is now available on many instruments. Retinal OCTs can capture and analyze anterior segment scans

Release Date: September 2017

Expiration Date: September 15, 2020

Goal Statement: With optical coherence tomography techniques expanding, optometrists need to know when to apply which modalities to which patients. This article explains how OCT—with an emphasis on enhanced depth imaging, swept source technology and OCT angiography—can be applied clinically to diagnose and monitor macular disease, diabetic retinopathy and glaucoma, among others.

Faculty/Editorial Board: Lee Vien, OD, David Yang, OD

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with a license and software upgrade. Some OCTs require anterior lenses that attach to the instrument to provide wide-view scans to visualize the anterior chamber or angle-to-angle anatomy.

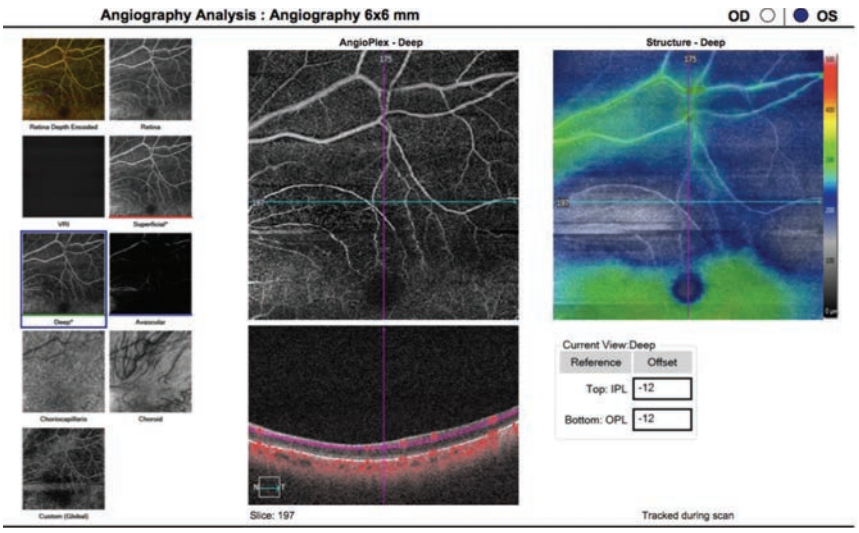
AS-OCT can analyze anterior chamber depth, angle measurements and provide pachymetry maps. Its clinical applications range from rigid contact lens fitting to corneal pathologies and complications from corneal surgeries as well as conjunctival lesions. It can also provide an assessment of the tear meniscus in dry eye patients.

AS-OCT can be a useful technique for evaluating anterior chamber inflammation in patients with uveitis. This imaging is especially useful in patients with decreased corneal clarity from corneal edema.[1]

For glaucoma evaluation, AS-OCT can provide valuable images of filtering blebs and tube positioning, as well as the anterior chamber angle, trabecular meshwork and Schlemm's canal. These images aid ODs in the assessment of structural abnormalities.[2-4]

Although AS-OCT does not replace gonioscopy in the detection of angle closure, it has increased our understanding of the disease and helped us identify new risk factors so that treatment can be targeted to those patients with higher risk.[5,6]

Posterior segment OCT provides two main types of macular scans: line scans and macular volume scans. Line scans have adjustable orientation and length and provide a higher definition image of a small area. Macular volume scans, also called cube scans, can provide a three-dimensional image of a larger area, relative to a line scan. Macular volume scans provide macular thickness measurements in the nine standard Early Treatment Diabetic Retinopathy Study macular grid subfields.[7] The boundaries of the macular thickness scans are typically the internal limiting membrane (ILM), the inner boundary to the retinal pigment epithelium (RPE) and the outer boundary.[8] Variations in the outer boundary, either above or below the RPE, are instrument dependent. Analysis software can help determine whether progression or change has occurred, allowing for the tracking of retinal thickening or thinning over time.



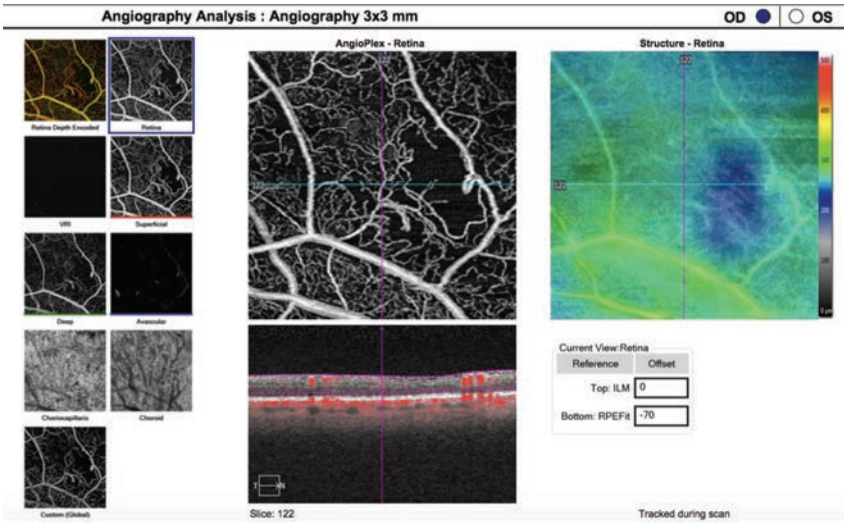
This OCT-A image (6mm by 6mm) shows a patient with a previous branch retinal artery occlusion, as indicated by the well-demarcated area of flow interruption in both superficial and deep vascular capillary plexuses visible on the scan.

For optic disc scans, OCT captures the circumpapillary retinal nerve fiber layer (cpRNFL) thickness is evaluated. The scan circle diameter can vary with each instrument. The data from the cpRNFL is sampled and thickness values are generated. These can be divided into clock hours, sectors and quadrants that are then compared with a normative database. Optic nerve head (ONH) parameters, such as disc area, rim area, cup-to-disc ratio and cup volume, are also captured and research demonstrates they are sensitive in the detection of glaucomatous change.[9,10] Thickness maps based on a thickness scale and deviation maps based on a normative database provide spatial and morphologic information of RNFL damage and can detect RNFL defects.[9,10]

Macular ganglion cell thickness is now available on SD-OCT instruments. The macular parameters are the ganglion cell complex (GCC)—which includes the ganglion cell layer (GCL), inner plexiform layer (IPL) and RNFL, and ganglion cell-inner plexiform layer (GCIPL)—which includes the GCL and IPL.[6]

For the diagnosis of early glaucoma, these macular parameters have a high discriminating power and reproducibility comparable to peripapillary retinal nerve fiber layer (pRNFL).[11] Macular ganglion cell thickness analysis is also a more reliable diagnostic parameter in the detection of glaucoma for myopic optic disc tilt than ONH parameters.[12,13] In advanced stages, research shows GCIPL glaucoma progression analysis (GPA) is more useful than cpRNFL GPA in detecting disease progression.[14]

OCT can track non-exudative age-related macular degeneration (AMD) progression and identify many features related to the



This OCT-A image (3mm by 3mm) of both superficial and deep vascular capillary plexuses shows capillary nonperfusion from severe nonproliferative diabetic retinopathy. An abnormal vascular network is visible adjacent to the area of nonperfusion.

degenerative process.[15-17] These findings include reticular drusen, subretinal drusen deposits, pseudocysts, outer retinal tubulations and drusen-associated acquired vitelliform lesions.[15-17]

OCT is now routinely used to evaluate diabetic macular edema (DME) and macular edema associated with other conditions, such as retinal vein occlusions. The instrument allows an objective quantification and localization of retinal thickening, providing the ability to track treatment response. Many large clinical trials incorporate OCT measurements into their definitions of macular edema and use those measures as a clinical endpoint.[18-22] DME, for instance, was defined by reduced vision and an increase in central subfield thickness on OCT in the RISE and RIDE studies.[18] The mean change of central foveal thickness based on OCT was also evaluated as a secondary outcome in the trials.[18]

New techniques, such as enhanced depth imaging (EDI), SS-OCT and OCT angiography (OCT-A), are providing further insight into ocular structures, such as the choroid, lamina cribrosa and ocular vasculature.

Enhanced Depth Imaging

This recently developed modality allows for improved choroidal evaluation by producing images of structures such as Bruch's membrane, the choriocapillaris complex, Sattler's layer, Haller's layer and the suprachoroidal layer.[13] Studies on choroidal thickness with EDI-OCT have identified differences with age, myopia, female gender and increased intraocular pressure (IOP), as well as in certain ocular pathologies such as central serous chorioretinopathy, polypoidal choroidal vasculopathy, vitelliform macular dystrophy and glaucoma.[24-28]

Researchers have extensively studied choroidal thickness in glaucoma with EDI-OCT.[29-33] The pathogenesis of glaucoma has been linked to ischemia that occurs in the prelaminar area of the optic nerve.[34] The blood supply to the optic nerve originates from the branches within the peripapillary choroid. OCT research supports an association between glaucoma and impaired choroidal circulation.[35] Research using EDI-OCT does not appear to highly correlate choroidal thickness with either open-angle or normal-tension glaucoma, or the severity of glaucomatous damage.[31-33]

Investigators also studied the lamina cribrosa (LC) and its association with glaucoma.[36-38] LC thickness has been associated with severity of glaucoma and found to be thinner in normotensive glaucoma eyes and pseudoexfoliative glaucoma eyes than in primary open-angle glaucoma eyes with the same severity of damage.[36-38] In addition, a thinner LC and a larger LC displacement had a significant influence on the rate of progressive RNFL thinning.[39]

Swept Source

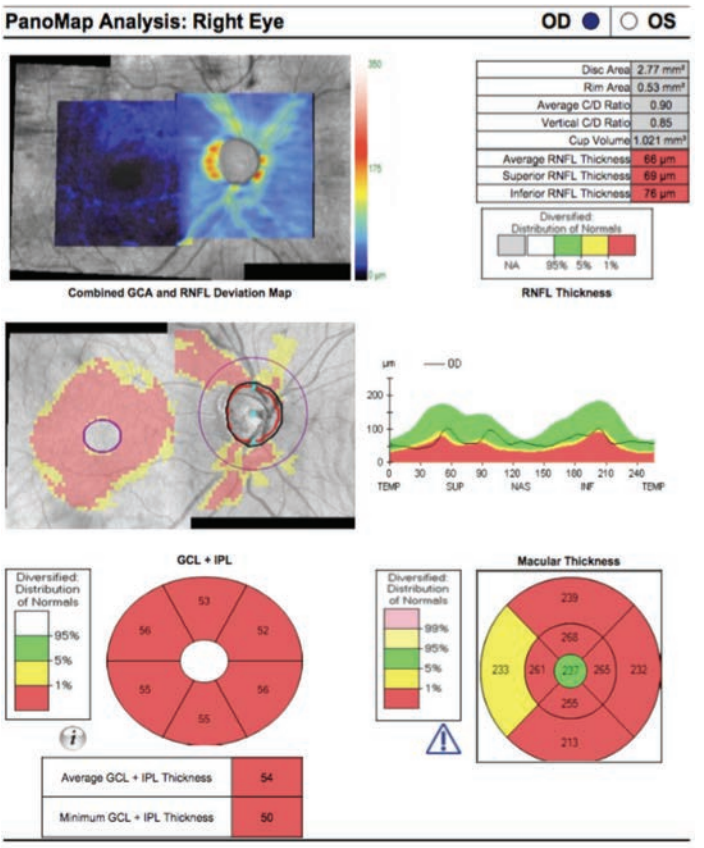
SS-OCT provides higher image acquisition speed (~100,000 A-scans/seconds), use of a longer wavelength (1050nm to 1060nm), and less variation in sensitivity with depth compared with SD-OCT.[40,41] These differences offer advantages over SD-OCT when imaging the vitreous and choroid. In addition, modalities such as *en face* and OCT-A imaging are improved with SS-OCT as they aid in imaging further into the anterior, as well as deeper into the ocular tissue, than we could with SD-OCT.[40,41] Structures in the vitreous that can be visualized with SS-OCT include the posterior precortical vitreous pockets—which play a role in various vitreomacular disorders—Cloquet's canal, posterior cortical vitreous, posterior hyaloid and vitreous opacities.[42,43]

The longer wavelength used in SS-OCT provides better penetration of ocular tissues and results in less scattering at the photoreceptor and RPE layers, allowing simultaneous visualization of the choroid, sclera and

vitreous.[42,44] SS-OCT also offers automated segmentation of choroidal thickness and choroidal thickness maps to help identify abnormalities in the choroid.

Studies comparing EDI-OCT and SS-OCT on penetration depth in choroidal imaging have found both imaging modalities to be comparable in normal eyes.[45] However, research shows in conditions such as PCV where the choroid is abnormally thicker, SS-OCT may better detect the choroid-sclera interface.[46] SS-OCT also has an advantage over EDI-OCT in imaging the posterior sclera in myopic glaucoma eyes.[47] Compared with SD-OCT, SS-OCT has similar intradevice repeatability on the assessment of cpRNFL and macular GC-IPL thickness.[48] Imaging of the LC has also been studied with SS-OCT, which can provide a three-dimensional image to better evaluate LC morphology, compared with SD-OCT.[49]

Although the literature has reported on SS-OCT technology for the last few years, the instruments were mainly prototypes for research. Recently, the FDA approved the first commercially available SS-OCT for assessment of the retina, the Plex Elite 9000 SS-OCT (Carl Zeiss) which has an axial resolution of 5.5µm and a scan speed of 100,000 A-scan points per second. This instrument—as well as the not-yet-FDA-approved Triton SS-OCT (Topcon)—offers widefield scans and OCT-A, but differ in their software and analysis. The Triton's normative database is not FDA approved in the United States, and the current Plex Elite 9000 does not have a reported normative database.



This SD-OCT PanoMap shows a glaucoma patient with thinning of the retinal nerve fiber layer as well as ganglion cell analysis.

OCT Angiography

This technique employs sequential high-speed OCT scans taken at the exact same location to detect motion. Movement is determined when the camera detects a difference in signal intensity between the two scans. Assuming steady patient fixation, the successive OCT scans should be identical, except for moving elements such as erythrocytes. The movement of red blood cells can be seen due to variation from one image to another, thus allowing for examination of blood flow in the retinal vasculature at various depths. This makes OCT-A a beneficial adjunctive test to fluorescein angiography (FA) and indocyanine green angiography (ICG).

OCT-A can delineate the foveal avascular zone and areas of capillary dropout better than FA and ICG.[50-54] However, there are limitations to OCT-A, such as a small field of view, making it a poor technique for imaging the peripheral retina. Additionally, it is not able to show leakage, staining or pooling, which FA and ICG can.[50-52,55] Moreover, current OCT-A instruments are prone to artifacts from eye movement, eyelid blinks, signal attenuation, shadows and segmentation errors. These movements can result in vessel doubling, stretch artifacts, displacement artifacts, checkerboard defects, gap defects and false-positive flow defects. Blockage and attenuation of the OCT-A signal can occur due to blinking and ocular media opacities such as cataracts, vitreal floaters and corneal scars. If the signal is sufficiently blocked, it can result in vascular areas misidentified as vasculature loss or areas of no flow. Other artifacts can result from errors in the segmentation of the various retinal layers. If layers are misidentified, data can be combined from multiple layers, resulting in inappropriate mapping.[51,53,56,57]

Vascular layers deep to the RPE can be imaged better with swept-source technology vs. SD-OCT, owing to the deeper penetration of

tissue with its longer wavelength. In a study comparing the two techniques for imaging the choriocapillaris beneath drusen, SS-OCT-A was found to be less prone to false-positive low flow areas.[58]

OCT-A is currently being used to study various retinal and choroidal pathologies, as well as ONH disorders.[50-53] Increasingly, the evidence in the literature describes its use in AMD, diabetic retinopathy and vascular occlusive disease, as well as other retinal conditions.[50-53] In some cases, it is even detecting pathology that was not seen on FA.[59,60] Doctors can use OCT-A's ability to detect movement to identify nonexudative type 1 neovascular (NV) lesions, even in asymptomatic patients with intermediate AMD.[59,60] These lesions were not appreciated on FA and there was no fluid seen on structural OCT imaging, although these areas of neovascularization were associated with plaques seen on ICG.[59,60] A recent study showed the combination of OCT-A and structural OCT can detect type 1 NV lesions in 85.7% of eyes, which was superior to structural OCT or FA alone (66.7%).[61]

In patients with diabetic retinopathy, OCT-A can better delineate the foveal avascular zone and areas of capillary dropout compared with FA, while also providing a higher percentage of gradable images.[54]

OCT-A is also being used to examine various optic neuropathies, including glaucoma, papilledema, anterior ischemic optic neuropathy and other acute and chronic optic nerve disorders.[62-64] Studies show OCT-A can provide noninvasive imaging of the microvasculature of the ONH and adjacent peripapillary tissue.[62-64] Research demonstrates decreased peripapillary perfusion—as imaged by OCT-A—has excellent correlation with areas of RNFL atrophy—as measured by structural OCT—in glaucoma patients.[63]

Additionally, other studies have revealed a decrease in vessel density of the peripapillary capillary network in eyes with various optic neuropathies. This supports the relationship of decreased retinal vasculature associated with glaucomatous and nonglaucomatous RNFL atrophy.[62-64]

Limitations of Today's OCT

Artifacts can be a source of error when obtaining OCT images, and they may affect the measurement of retinal thickness. These artifacts include motion artifacts and media opacities that can block the signal. Some pathologies can also contribute directly to segmentation errors, such as epiretinal membranes, vitreopapillary traction or vitreomacular traction. Interpretation of the OCT color-coding can be falsely positive or negative given the limited normative database for each instrument. These databases have a narrow age range, refractive error range and limited ethnic groups.[65,66] Patients with displaced RNFL bundles, such as high myopia and tilted discs, are examples of patients who may not fit the normative database. For these patients that fall outside the normative database, longitudinal evaluation of the cpRNFL is important to detect disease progression.

Advancements

Recent innovations in OCT imaging have improved the diagnostic ability of the instrument, especially in the detection of glaucomatous damage. These new OCT parameters include Bruch's membrane opening-minimum rim width (BMO-MRW) and BMO-minimum rim area (MRA). BMO-MRW quantifies the rim from its true anatomic outer border and accounts for variable orientation. Its width has a higher diagnostic accuracy for glaucoma and a stronger correlation with visual field compared with conventional rim parameters including cpRNFL, according to researchers.[67,68] The new parameter is also more specific than GCIPL in tilted disc with moderate myopia.[69] BMO-MRA is not influenced by disc size, unlike BMO-MRW, and offers superior diagnostic power to detect glaucoma compared with other parameters in a full range of very large to small disc sizes.[70] Additional studies using these new parameters are still needed to establish a normative database. However, current research shows a greater specificity of these new parameters when considering the highly variable anatomy of the ONH both within and between individuals.[67-70]

The advent of OCT technology has improved optometrists' ability to detect, understand, monitor and track progression of various ocular pathologies, as well as response to treatment modalities. Advances in imaging techniques with EDI-OCT and SS-OCT have provided the ability to visualize the choroid and gain new insights on the role of the choroid in chorioretinal conditions. SS-OCT technology has also improved imaging of the vitreous and enhanced techniques such as *en face* and OCT-A. With the introduction of OCT-A, we now have the ability to visualize abnormal blood flow in the choroid and retina.

New software and hardware upgrades have improved the repeatability, reliability and diagnostic capability of OCT scans. Recently introduced ONH parameters, such as BMO-MRW and BMO-MRA, have shown potential on its specificity, especially in glaucoma detection. OCT has many applications in clinic today, but future studies will determine the full extent to which these new technologies can improve patient care. ■

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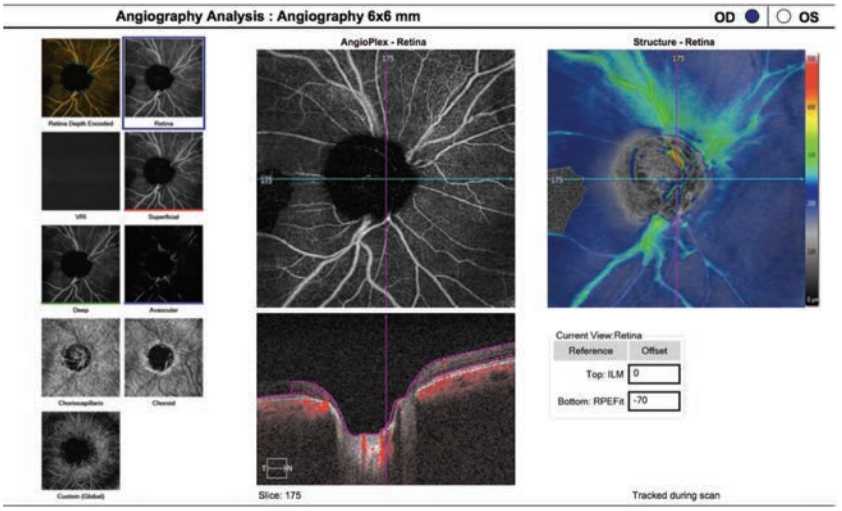

This OCT-A (6mm by 6mm) image shows the same patient as the image on page 73. Here, you can see a decrease in peripapillary capillary perfusion corresponding to the areas of RNFL loss.

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1. AS-OCT can analyze all but _____
a. Anterior chamber depth.
b. Angle measurements.
c. Intraocular pressure.
d. Pachymetry maps.

2. AS-OCT's clinical applications include:
a. Cornea.
b. Conjunctiva.
c. Tear film.
d. All of the above.

3. Assessment of the ganglion cell complex involves which layers?
a. ILM, RNFL and GCL.
b. RNFL, GCL and IPL.
c. GCL, IPL and INL.
d. RNFL and GCL.

4. The RISE and RIDE studies defined _____ by reduced vision and an increase in central subfield thickness on OCT.
a. Diabetic macular edema.
b. Retinal detachment.
c. Age-related macular degeneration.
d. Glaucoma.

5. Enhanced depth imaging allows for improved imaging of which structure, relative to standard SD-OCT?
a. Cornea.
b. Vitreous.
c. Retina.
d. Choroid.

6. EDI-OCT is being used in the assessment of glaucoma for measuring:
a. RNFL thickness.
b. BMO-MRW.
c. Lamina cribrosa thickness.
d. Neuroretinal rim thickness.

7. Posterior precortical vitreous pockets play a role in_____.
a. Vitreomacular disorders.
b. Glaucoma.
c. Retinoschisis.
d. Lattice degeneration.

8. The longer wavelength light used in SS-OCT allows for what advantages over SD-OCT instruments?
a. Better penetration of ocular tissue.
b. Less scattering of light at the RPE.
c. Simultaneous visualization of the choroid, sclera and vitreous.
d. All of the above.

9. Which of these technologies can best detect the choroid-scleral interface in conditions where the choroid is abnormally thick?
a. EDI-OCT.
b. SS-OCT.
c. TD-OCT.
d. FA.

10. Specifically in myopic glaucoma patients, SS-OCT is superior to EDI-OCT in imaging which ocular structure?
a. The posterior sclera.
b. Bruch's membrane.
c. The choriocapillaris complex.
d. The lamina cribrosa.

11. Which SS-OCT instrument is currently FDA approved?
a. Topcon Triton OCT.
b. Carl Zeiss Plex Elite 9000 OCT.
c. Carl Zeiss Stratus OCT.
d. Nidek RS-3000 OCT.

12. How does OCT-A detect motion?
a. It measures differences in signal intensity between sequential OCT B-scans.
b. It uses slow-motion imaging technology to view movement of cells.
c. It uses flicker chronoscopy of side-by-side images.
d. It requires swept-source technology to achieve appropriate scan speeds.

13. What are some potential causes of artifacts in OCT-A scans?
a. Large pupils.
b. Eye movement.
c. Dim room illumination.

OSC QUIZ

d. Insufficient contrast dye.

14. Eye movements can result in which type of OCT-A artifact?
a. Vessel doubling.
b. Media opacity artifact.
c. False-negative flow artifact.
d. Segmentation defect.

15. Which imaging technique is best in the delineation of foveal vascular zones and areas of capillary dropout?
a. FA.
b. OCT-A.
c. TD-OCT.
d. ICG.

16. OCT-A can be used to study:
a. Retinal disorders.
b. Choroidal disorders.
c. Optic nerve disorders.
d. All of the above.

17. What is true about BMO-MRW?
a. It's used in the assessment of diabetic retinopathy.
b. It's affected by tilted discs.
c. It has a high diagnostic accuracy for glaucoma detection.
d. It requires EDI-OCT.

18. Nonexudative type 1 neovascular lesions are best detected via which of the following?
a. OCT alone.
b. FA alone.
c. OCT and FA combined.
d. OCT and OCT-A combined.

19. Which pathology can contribute to OCT segmentation errors?
a. Epiretinal membranes.
b. Vitreopapillary traction.
c. Vitreomacular traction.
d. All of the above.

20. New advances in OCT technology are leading to:
a. Enhanced resolution.
b. Larger field of views.
c. Decreased motion artifacts.
d. All of the above.

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18. Ⓐ Ⓑ Ⓒ Ⓓ
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 clinical ability to employ OCT technologies in my practice. ① ② ③ ④ ⑤
22. Become familiar the imaging modalities on the market and the structures they can image. ① ② ③ ④ ⑤
23. Differentiate between which technologies are most appropriate for which disease types. ① ② ③ ④ ⑤
24. Better connect ocular structures that can be imaged with OCT technologies to disease progression. ① ② ③ ④ ⑤
25. Inform me of the current research being performed using OCT imaging technologies. ① ② ③ ④ ⑤
26. Recognize the limitations associated with the various modalities of OCT imaging. 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.

Signature _____ Date _____

Lesson 115393 RO-OSC-0917

Rhegmatogenous Retinal Detachment: How to Detect, How to Manage

Timely management is critical to successfully treating the most common form of detachments. **By Mohammad Rafieetary, OD, Stephen Huddleston, MD, and Roya Attar, OD, MBA**

Retinal detachment (RD)—a serious condition that requires urgent attention—is commonly encountered in vitreoretinal subspecialty practices. Rhegmatogenous retinal detachment (RRD) is the separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE) by fluid traversing from the vitreous cavity into the subretinal space via a retinal defect *(Figure 1)*. The intervening time between the development of a retinal defect and retinal detachment is highly variable and often unpredictable. Even giant tears may sit idly without progressing to a detachment for an extended period of time.

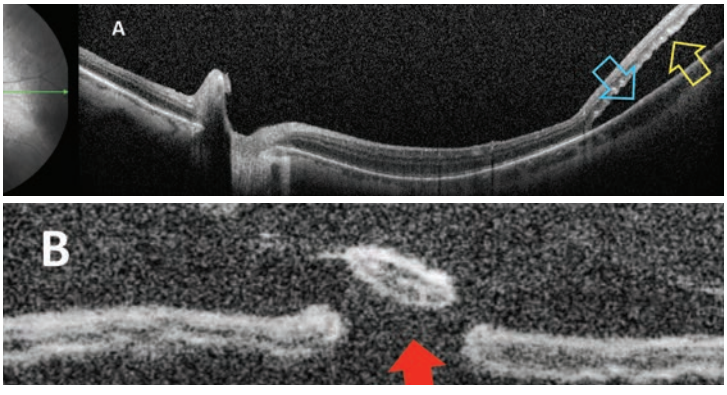

Fig. 1. Optical coherence tomography showing (A) separation of neurosensory retina (yellow arrow) from the RPE (blue arrow); (B) percolated retinal break (red arrow).

The preliminary or "triage" diagnosis is made based on the presenting history—such as sudden onset flashes, floaters, vision loss or a combination—as well as clinical and ancillary examination. At times, these presenting examination findings can be misleading, resulting in misdiagnosis and mismanagement of the patient.

Here are the principles of RRD, including pathophysiology, symptomatology, clinical findings and surgical management.

Epidemiology and Etiology

All non-exudative and non-tractional RDs originate from a retinal defect. Retinal defects are commonly described as tears or holes, and the most common way to acquire one is through the development of a posterior vitreous detachment (PVD).[1-4] Vitreous degeneration as a process of aging is well understood and is best described as the progressive detachment of the vitreous cortex from the retinal surface, which rarely creates retinal tears. Progression time from tear to detachment is unpredictable, and varies from hours to days or even months. Some tears may exist chronically for years, while others create detachments in a matter of hours. Most tears, if caught early, can be treated with a laser in a clinical setting even with the presence of a small amount of subretinal fluid. Once a retinal detachment develops, surgical intervention

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becomes necessary.

Risk factors other than age include: family history, particularly in certain genetic conditions, high axial myopia, history of RRD in the fellow eye, trauma, certain conditions such as uveitis and retinopathy of prematurity (ROP). Previous intraocular surgery, especially for cataract, is a risk factor.[5,6]

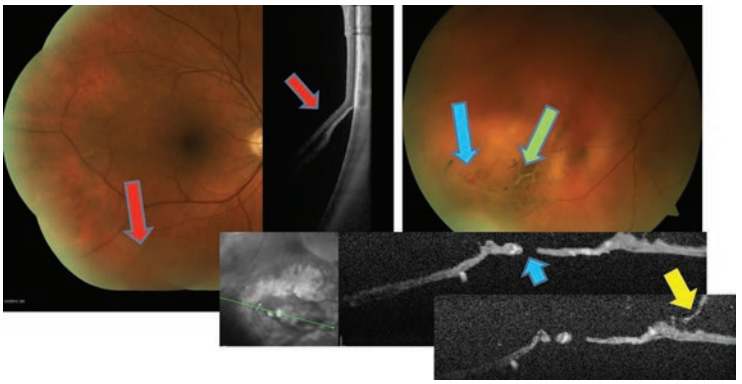

Fig. 2. This patient with preexisting lattice (green arrow), developed a retinal break (blue arrows) during PVD (yellow arrow), resulting in an inferior retinal detachment (red arrows).

The incidence of RD in the general population is between 0.01% and 0.018%.[5-15] This increases to 1% following cataract surgery and up to a four-fold increase with Nd:YAG capsulotomy.[16-21] Peripheral retinal disease and degeneration can be a predisposing risk factor for RRD, especially lattice degeneration *(Figure 2)*.[3,22] Approximately 30% of phakic patients with RRD have pre-existing lattice degeneration, whereas all patients with lattice degeneration have less than 1% chance of developing RRD.[23,24]

Symptomatology

Asymptomatic RDs are usually detected during primary eye care visits. These usually present as inferior or temporal chronic detachments originating from atrophic holes *(Figure 3)*. A non-central RD can be asymptomatic, as patients may not be as sensitive to slowly progressive peripheral vision loss. This is particularly common when RRDs occur in inferior and temporal locations because the superior and nasal fields of view are less sensitive.

Most patients presenting with an RD report recent positive visual phenomenon despite the detachment's onset possibly being weeks to months prior to the presentation of symptoms. There are many causes for this: patients do not notice flashes if they are asleep, recognition of unilateral vision loss is often delayed and RDs are usually first noticed only when they begin to affect central vision.

Symptoms may also be nonspecific. Floaters, a common problem across all age groups, are usually benign. Flashes may be associated with successful vitreous separation without retinal tears or detachment or may have non-ocular etiologies such as transient ischemic attack and acephalgic migraine. Negative dyschromatopsia may simulate flashes and visual field loss, and various other intraocular processes may decrease central and peripheral vision. Still, complaints of flashes, floaters or negative dyschromatopsia warrant a thorough evaluation, as any of these symptoms may be associated with RRD. With uncertain or unusual findings, clinicians should refer to a specialist.

Findings

The clinical evaluation should be as thorough as possible, especially the dilated fundus exam. A macula-involving RD should be easy to spot, but a far peripheral detachment, especially in the pediatric population, can be quite challenging to identify. Although the goal is to identify retinal abnormalities before they progress to detachment, if you observe any hint of subretinal fluid (SRF) or a retinal break with or without SRF, you've seen all that is needed to warrant an immediate referral. While the retinal specialist will document every break, the referring doctor can help document as many as possible before the patient sees the specialist.

If the clinical exam does not reveal an RD, scleral depression and a three-mirror contact lens examination can help to rule out small peripheral tears. Again, if you have any doubt, referral is always the best option. The presence of acute PVD, pigment cells in the anterior vitreous or vitreous hemorrhage should increase the level of suspicion and calls for further examination, either by ultrasonography or vitrectomy.[1,2,4,25-27] In particular, retinal breaks are highly probable with vitreous hemorrhage secondary to PVD, and need to be discovered or ruled out by careful examination.

Certain conditions may present similarly to RRD, including: serous retinal detachment secondary to inflammatory or neoplastic disease, tractional retinal detachment, vitreous opacities such as vitreous hemorrhage as well as retinoschisis *(Figure 4)*.

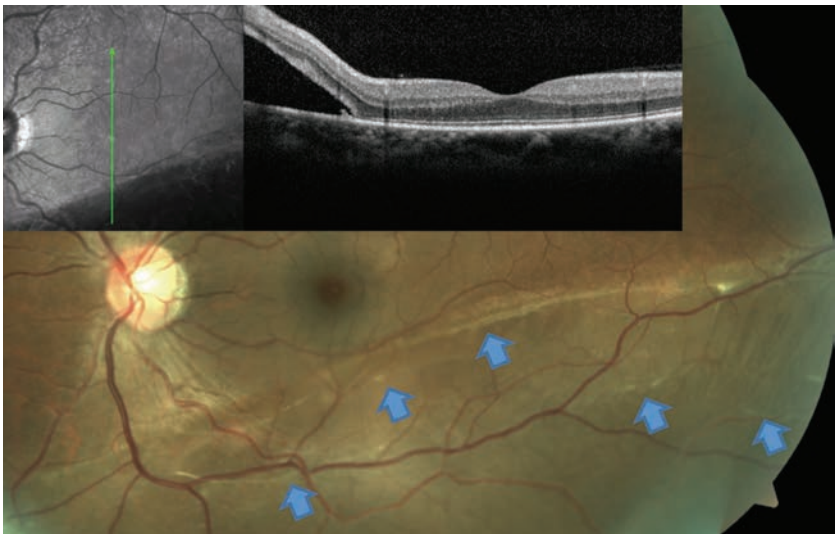

Fig. 3. This RRD patient's OCT image, layered over a fundus photograph, shows partial involvement of the macula, even though the patient is totally asymptomatic from this slowly progressive inferior RD. Blue arrows point to a number of subretinal bands and partial demarcation lines, a testament to the chronicity of the condition.

Auxiliary Testing

Though the most effective technique to diagnose RRD is a thorough dilated fundus exam, certain ancillary tests can also help clinicians better document the severity of the retinal abnormality. Widefield fundus photography, often with conventional white flash, can help, as can scanning laser ophthalmoscopy devices. Optical coherence tomography (OCT), particularly in widefield mode, is helpful in the differential diagnosis of RRD and can help clinicians assess the macular status at the time of diagnosis.[28] Surgical intervention is more time-sensitive in macula-on as opposed to macula-off detachments. This role is reversed in tractional RD, where macular involvement requires quicker surgical intervention. Using B-scan ultrasound is usually not necessary to diagnose RRD when the clinical view is clear; however, it is essential in the presence of media opacification, as well as in the differential diagnosis of masquerading conditions.[29]

Fluorescein angiography (FA) and visual field (VF) testing are also not essential, though they may aid in the differential diagnosis. VFs may be necessary in cases of injury, workers' compensation, litigation or assessment of functional vision loss.

Management Options

Timely management of RRD and its associated findings is critical to decrease the chance of long-term vision loss.

Round holes, horseshoe tears and giant breaks can all be treated with laser or cryotherapy in an office equipped and staffed for it *(Figure 5)*.[30,31] Small amounts of associated fluid may also be surrounded with laser or cryotherapy to prevent progression. The presence of substantial subretinal fluid warrants either a pneumatic retinopexy or operative RD repair. Localized, slow-growing chronic RDs, which are usually described as subclinical based on their usually asymptomatic nature may be

walled off with laser as well *(Figure 6)*.[32] Laser treatment is performed with either a slit lamp laser or laser indirect ophthalmoscopy (LIO). Both are highly effective; however, LIO is usually required for far peripheral treatment.

Laser treatments are only effective in regions where neurosensory retina and the RPE are either in normal apposition or when they can be brought together through scleral depression; therefore, the laser is applied at the border of the attached and detached retina. Scleral depression can help to drive the fluid away by increasing the surface area on which the laser can be applied.

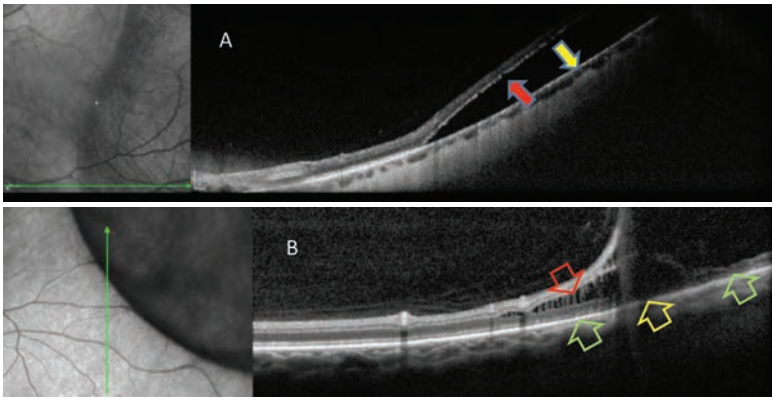

Fig. 4. OCT is helpful in differentiating RRD from retinoschisis. (A) Neurosensory retina is separated from RPE in RRD. This patient requires surgical intervention. (B) In retinoschisis, separation and spilling of neurosensory is seen. Normal apposition of the outer retina to RPE is seen (green arrows) on either side of the outer retinal break (yellow arrow). This patient may be closely monitored.

Although the consensus is to monitor asymptomatic retinal tears, there is still a 5% chance of RD with need for more complex management and potential of vision loss.[33] This statistic, we believe, makes a strong case for treating all retinal defects with laser treatments to guard against RD. Horseshoe tears should be treated immediately, while round holes are less urgent. Prophylactic retinopexy is normally an extremely low risk procedure when performed by a skilled operator, and in absence of retrobulbar block.[33]

Pneumatic retinopexy is best used for RDs where the retinal break is localized to the superior 180 degrees of the retina and, if more than one break exists, they are localized in such a way that an expansible bubble can adequately cover them with proper patient positioning.[30] However, this technique is not effective in the presence of proliferative vitreoretinopathy (PVR). Pneumatics may also be used in some cases as a temporary tam-

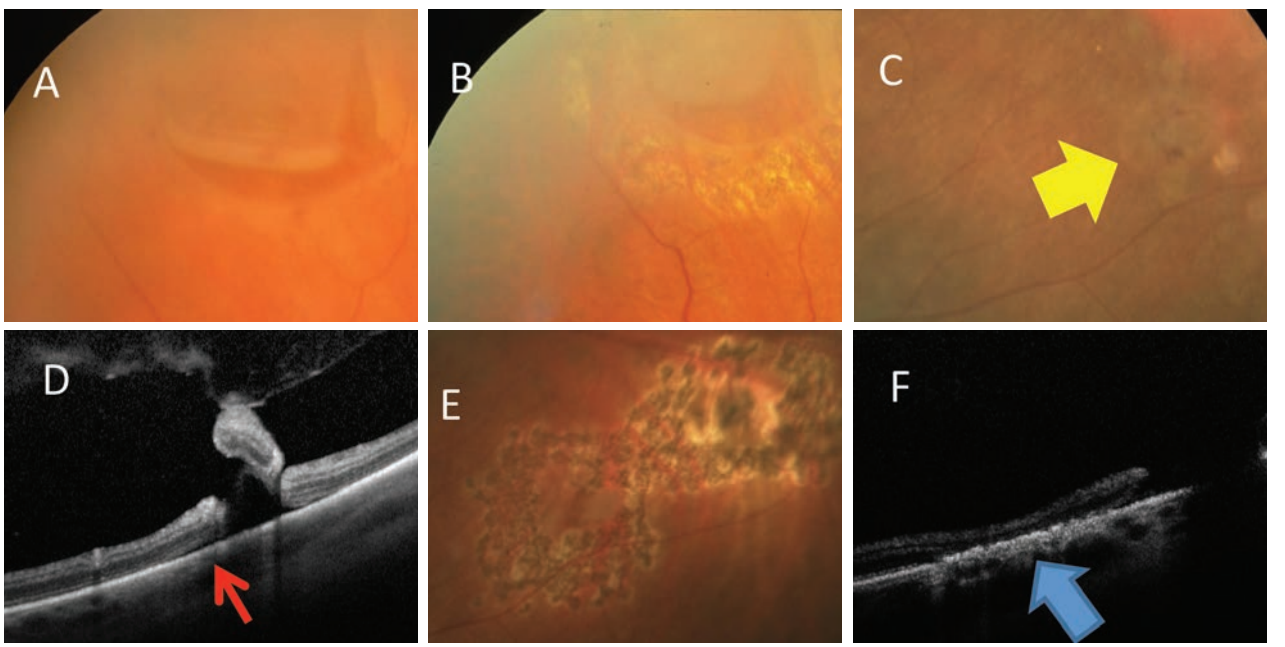

Fig. 5. (A) Horseshoe retinal tear is well bordered by laser (B). (C) Retinal breaks (yellow arrow) with (D) associated subretinal fluid (red arrow) well bordered by (E) laser. (F) Alteration of the RPE resulting in a watertight tissue (blue arrow).

ponade when an operating room procedure is delayed for logistical reasons. The procedure involves carefully positioning the patient, then injecting a bubble of pure perfluoropropane (C3F8) or sulfur hexafluoride (SF6) though the pars plana. The anterior chamber is either tapped prior to or immediately after the gas is injected to normalize the intraocular pressure (IOP). Retinopexy can be performed as either cryotherapy, which has to be applied to the breaks prior to gas injection, or with a laser, which is performed after several hours or days once the retina is re-attached.

Procedural Pearls

The selection between C3F8 or SF6 depends on the individual surgeon's preference and comfort with each gas. C3F8, which is a long-acting fluorinated gas that expands to a greater volume than SF6, is used for all pneumatic retinopexies we perform. However, in our practice we use an iso-expansile SF6/air mixture for all rhegmatogenous RDs needing vitrectomy. Complications related to gas use in vitreoretinal surgery include: increased intraocular pressure in the short term (due to incorrect gas mix or pupil block in aphakia, for example) and cataract formation in the medium term (due to incorrect patient positioning). The self-elimination could also be a weakness, mainly due to the insufficient time for chorioretinal adhesion formation, resulting in failure of the primary surgery.

Small-incision pars plana vitrectomy (PPV) using 25- or 27-gauge instruments is a safe and effective surgical procedure.[34,35] In RRD repair, several agents in addition to C3F8 and SF6, such as silicone oil or the perfluorocarbon liquid perfluoron (PFO) are used for retinal tamponade.[36] The attraction between the individual molecules in these substances creates surface tension, which prevents aqueous egress back through retinal defects and prevents retinal re-detachment immediately after surgery to give the laser the time needed to form a strong chorioretinal scar. Factors that influence the choice of agent include: location and extent of the RD and the associated retinal breaks, presence of other clinical findings such as PVR and the patient's ability to position.

Silicone oil is a good option when the likelihood of retinal re-detachment is high in the immediate postoperative period due to an inability to adequately treat retinal breaks (albinism, blood-obscuring known breaks, loss of visualization in surgery), an inability to correctly position and PVR, for example.

Unlike C3F8 and SF6, which are slowly absorbed out of the vitreous cavity and replaced with aqueous, silicone oil can remain in the vitreous cavity for a long time. However, the long-term duration of silicone can result in some complications, including shearing off multiple small bubbles through the detergent action of intraocular proteins and patient motion, which is termed silicone emulsification *(Figure 7)*. These bubbles may then migrate to the anterior chamber and cause blockage of the trabecular meshwork, resulting in increased IOP and subsequent glaucoma or corneal decompensation. Silicone oil removal or replacement requires another trip to the retinal surgeon.

The final vitreous substitute is PFO. This is a liquid with high specific gravity that will sink to the inferior vitreous cavity. PFO can be used as an intraoperative

tool to unfold the retina in giant RDs, as well as to drain subretinal fluid out the original tears without creation of a retinotomy. PFO can also be used in the medium term by leaving the PFO in for one to two weeks after performing laser retinopexy around all breaks for the laser to heal. Another trip to the surgeon is required for removal. While an uncommon technique, it works in the right situation. PFO causes a modest foreign body inflammatory reaction in some patients if used on a medium-term basis or if a small amount is retained after surgery. PFO will also migrate to the subretinal space if used inappropriately for tractional RDs or PVR.[30] If droplets are retained in the anterior chamber, they may be removed at the slit lamp in the office relatively easily.

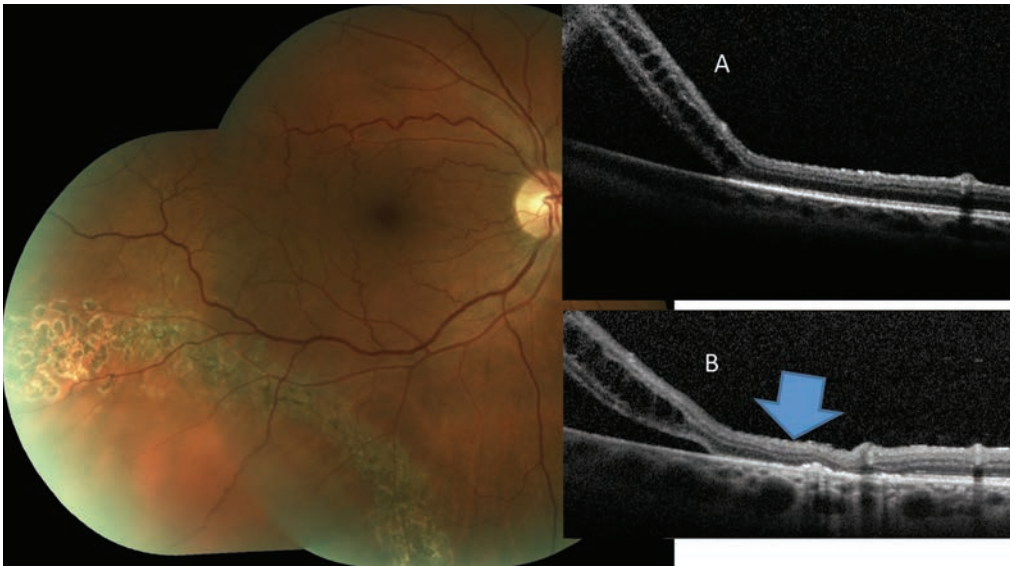

Fig. 6. This asymptomatic patient with subclinical RRD not involving the macula is effectively treated by laser bordering. The alteration of the outer retina-RPE junction can be noted when the pretreatment OCT (A) is compared to the posttreatment scan (B) (blue arrow).

Significant RDs are treated either with vitrectomy alone or scleral buckle with or without vitrectomy.[30] The use of scleral buckle has decreased in favor of vitrectomy.[37,38] The discomfort, poor refractive outcomes and tissue erosion caused by scleral buckles can be avoided with vitrectomy alone without compromising outcome rates.[39,40] No studies show scleral buckle is superior to vitrectomy-based surgery alone. Scleral buckling induces unpredictable myopic shift, pain, increased phorias and tropias, buckle extrusion, ptosis and ocular surface disorder. Nevertheless, good outcomes are still achieved with scleral buckle, and most retina surgeons still advocate their use in subclinical RDs without proliferative vitreoretinopathy in children and young adults.

Regardless of tamponade agent or use of a buckle or not, the most common cause of RRD repair failure is PVR.[30,41] Other causes include inadequate retinopexy, poor patient positioning and unrelieved vitreous traction resulting in additional tears forming after leaving the operating room.

Complications following vitrectomy include increased rate of progression of cataract, endophthalmitis, suprachoroidal hemorrhage and re-detachment.[42]

RRDs, without proliferative vitreoretinopathy, can have single surgery success rates as high as 75% with pneumatic retinopexy, and up to 90% with vitrectomy, scleral buckling or a combination of the two.[35] Proliferative vitreoretinopathy at presentation implies chronicity and a higher likelihood of requiring multiple surgeries. The crucial factors in obtaining good outcomes include timely diagnosis, selection of the right intervention and careful follow up. Optometrists play a significant role as the primary eye care providers and the entry point of most patients with signs and symptoms suggestive of retinal detachment. ■

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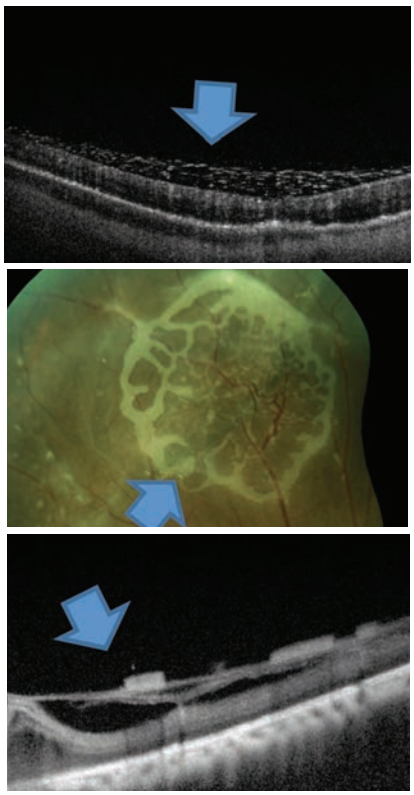

Fig. 7. This is one presentation of emulsified silicone seen on the retinal surface.

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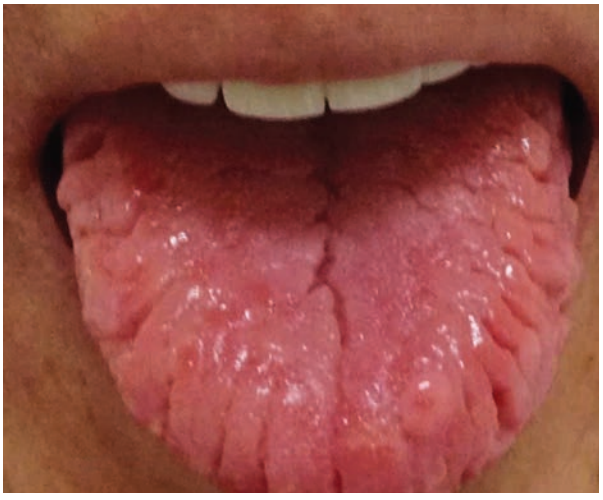
Dry Eye and Systemic Disease: What's the Association?

As principal gatekeepers, we need to be able to spot these cases right away.

By Chandra Mickles, OD

Our understanding of dry eye disease (DED) has grown tremendously in recent years. As the prevalence of associated systemic conditions grows, optometrists must be aware of the connections between systemic conditions and DED.[1,2] Autoimmune diseases such as Sjögren's syndrome (SS), rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) are often linked to DED, for example.[3,4] Diabetes mellitus (DM) also is an important systemic cause of dry eye.[5] Consequently, both DM and SS are key systemic conditions clinicians should explore when examining DED patients. These clinical clues can help optometrists intervene sooner to provide the best management options.



A sore, cracked tongue is suggestive of Sjögren's syndrome.

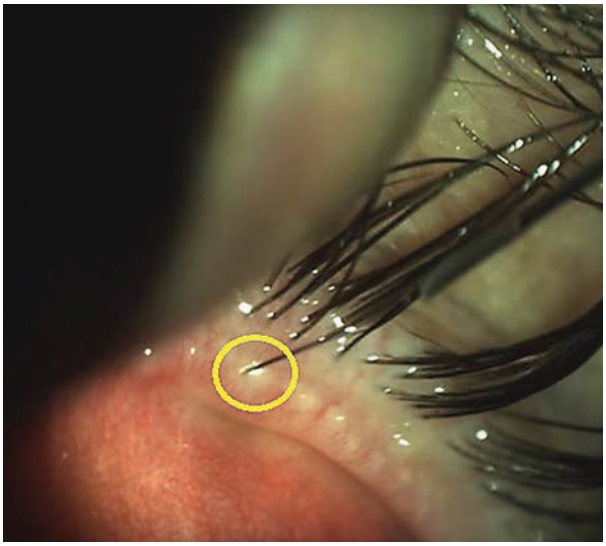
Diabetes

Research suggests more than 50% of patients with DM have dry eye, and significant associations exist between DED and the duration of diabetes and diabetic retinopathy.[6-9] Meibomian gland dysfunction (MGD) is also associated with DM, and one study shows a significant increase in the frequency of MGD in diabetes patients compared with those unaffected.[10,11]

Clinical clues. Polyuria and polydipsia are known symptoms of DM, and patients often report typical dry eye symptoms such as burning, foreign body and gritty sensations and sore eyes.[6-8] However, practitioners should be mindful that patients with longstanding diabetes may report fewer symptoms as a result of diabetic corneal neuropathy.[5] A closer examination is warranted in patients presumed to suffer from DED who present with insignificant symptoms or none at all.

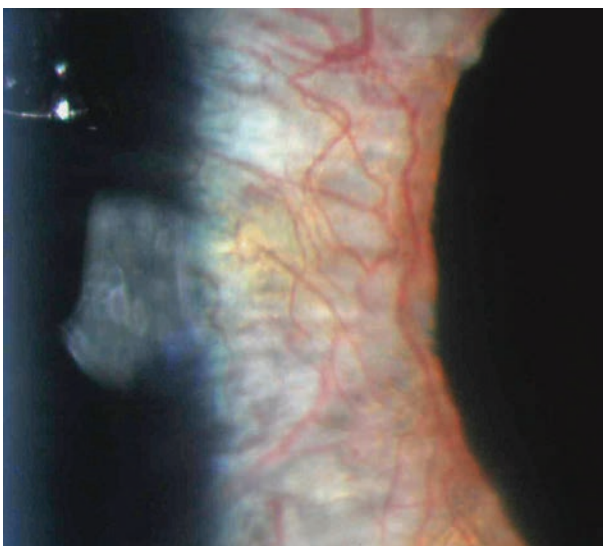
While many DED diagnostic tests may be useful, common tests such as the Schirmer's type 1 test without anesthesia, tear film break-up time (TBUT) and ocular surface vital dye staining may underestimate the presence of DED in patients with Type 2 diabetes.[12] Tear osmolarity (Tearlab) may be particularly useful to detect DED in patients with Type 2 diabetes.[12] A study comparing tear osmolarity with common DED diagnostic tests in patients with Type 2 diabetes found osmolarity had a greater diagnostic accuracy than Schirmer's type 1 test, TBUT, rose bengal and fluorescein staining.[12]

Corneal staining also may be useful in predicting the presence of DED associated with certain systemic diseases. Patients with diabetes are prone to corneal



Intraductal meibomian gland probing, a manual procedure to open the meibomian orifices, can be considered in ocular rosacea patients with MGD.

Photo: Aaron Bronner, OD



Marked neovascularization of the iris in this patient was caused by uncontrolled proliferative diabetic retinopathy.

erosions, and one study found corneal sodium fluorescein staining of the inferior corneal zone was predictive of DED in diabetic patients.[13,14]

Customized care. While the current treatment recommendations for diabetic and non-diabetic DED are essentially the same, recent research can guide us in customized treatment plans.

Dietary supplementation with omega-3 fatty acids (FAs), for example, may prevent or even reverse insulin resistance while also improving tear film function and DED symptoms in Type 2 diabetes patients.[15-17]

The Dry Eye Assessment and Management (DREAM) study—the first large-scale, multicenter, double-masked randomized controlled trial investigating omega-3 FA use for DED—may soon provide a clearer understanding of the efficacy and safety of omega-3 FAs in treating DED. The estimated completion date of the DREAM study is October 2017.[18]

As chronic hyperglycemia is a risk factor for diabetes-associated DED, patient education on glycemic control is still fundamental, as is education on the chronic nature of their dry eye.[5,19] These patients can be asymptomatic, and encouraging both annual ocular surface and fundus evaluations is imperative. Assessment of the retina and the ocular surface should become an integral part of our routine exams when diabetes is suspected.

Referral to an internist, including requests for blood glucose and HbA1c testing, is vital to our management of these patients as well.

Sjögren's Syndrome

When you suspect a DED patient may have an autoimmune disease, RA, SLE or even sclerodermas are among the conditions on the list. But when a DED patient cites a sore, cracked tongue, one of the most prevalent systemic diseases associated with DED, Sjögren's syndrome, should move to the top of the list.[20]

Don't Miss These Often Overlooked Associations

While Type 2 diabetes and Sjögren's syndrome have a high prevalence in our DED patient population, a variety of other systemic diseases warrant consideration.

Graves' disease. Dry eye is a frequent cause of ocular discomfort in Graves' disease, a common autoimmune condition.[1,2] While the association between Graves' disease and dry eye is well established, the pathophysiology is not completely understood. The latest evidence points to mechanical impairment of the eyelids, as well as immune-mediated lacrimal gland dysfunction and meibomian gland dysfunction. In a study examining morphologic changes in the meibomian glands of patients with Graves' orbitopathy compared with controls, a higher prevalence of MGD and more severe dry eye symptoms existed in the Graves' orbitopathy group than in the control group.[3]

Multiple sclerosis (MS). This demyelinating disease can lead to severe dry eye. In MS, poor corneal sensory impulse conduction can result in insufficient tear production, and lagophthalmos-associated DED can occur due to poor motor control.[4] These patients may have limited ability to instill drops and perform lid hygiene properly; thus, punctal plugs, humidifiers and in-office blepharitis therapies may be crucial.

Rosacea. Many patients with cutaneous rosacea cope with ocular symptoms.[5] Patients with cutaneous rosacea can develop blepharitis, MGD and associated DED; sometimes, these precede the cutaneous signs. Conventional blepharitis and MGD therapies such as eyelid hygiene and warm compresses, respectively, are mainstay therapies for mild cases. More advanced cases may necessitate topical or oral azithromycin and newer therapies such as intense pulse light and intraductal meibomian gland probing. Rosacea patients should also avoid any triggers such as stress, spicy food, alcohol and excessive sun exposure.[6]

Pediatric dry eye. Today, we are seeing more DED in children, likely due to the growing use of electronic devices.[7] Nonetheless, dry eye in otherwise healthy children is not common and should prompt investigation into a systemic cause such as juvenile RA, juvenile-onset SLE, diabetes, vitamin A deficiency and Riley-Day syndrome.[8,9]

Systemic medications. While necessary to treat many conditions, pharmaceuticals such as diuretics, antihypertensives, beta-blockers, antidepressants, antipsychotics, antihistamines, decongestants, gastrointestinal medications, oral contraceptives and analgesics may all cause or exacerbate dry eye.

Alcohol. The link between DED and alcohol is controversial. One study found that alcohol consumption may be a significant risk factor for dry eye.[10] Others suggest alcohol serves a protective role in the development of DED and that drinking and DED may be unrelated.[11,12] Based on a meta-analysis of 10 studies, researchers surmise that alcohol-induced peripheral neuropathy may falsely reduce the prevalence of DED.[10]

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While SS can occur alone in its primary form, about half of SS is found in the presence of another autoimmune connective tissue disease such as RA, and is known as secondary SS.[21,22] Immune-mediated damage to the exocrine glands, in particular the lacrimal and the salivary glands, results in the hallmark symptoms of dry eye and dry mouth.[22] These and a host of other systemic symptoms can be subtle, episodic and nonspecific, resulting in diagnostic delays.[21] Dry eye is often an early symptom, and a timely diagnosis can mitigate serious systemic complications such as lymphoma.

Clinical clues. To identify SS-associated DED (SS-DED), ask about classic dry eye and dry mouth, as well as prolonged persistent fatigue and joint pain—common SS symptoms.[23] Because some classic SS symptoms may not be obvious (a patient may not realize they have dry mouth), clinicians should also explore other nonobvious symptoms such as difficulty talking and swallowing and dental or gum problems.[23]

As in diabetes-related DED, SS patients may have objective signs of DED without subjective symptoms. A recent study indicates as many as 40% of SS patients with clear objective evidence of DED reported no symptoms.[24,25]

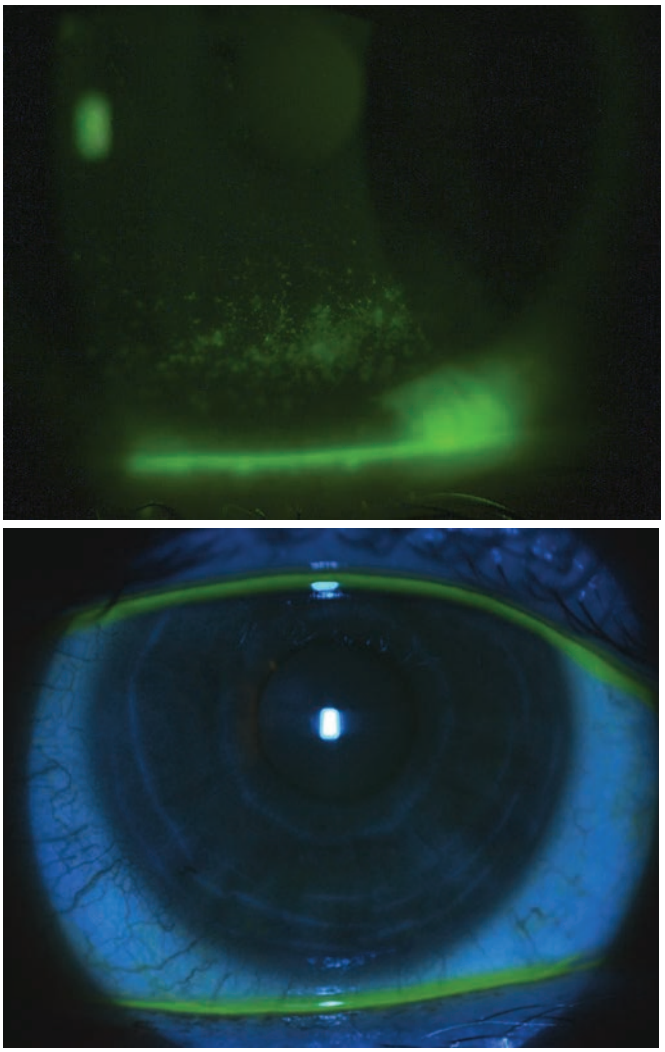
Important clinical tests in this patient population include ocular vital dye staining and Schirmer's type 1 testing. The most recently proposed 2016 classification criteria for SS by the American College of Rheumatology/European League Against Rheumatism includes an Ocular Staining Score equal to or greater than five and Schirmer's type 1 test less than 5mm in five minutes.[26]

Although SS falls under the umbrella of aqueous-deficient DED, SS patients may have a combination of aqueous-deficient and evaporative DED.[4,27] Additionally, ocular surface changes in SS patients may be attributed in part to MGD.[28] In a recent study comparing MGD in patients with SS, non-SS dry eye and non-dry eye controls, SS patients had more severe MGD with poorer mean meiboscore and meibomain gland expressibility than non-SS dry eye patients.[29]

Evaluating DED in patients with SS should include assessment of tear film stability and meibomian gland function. SS-DED may be differentiated from dry eye that is associated with other systemic conditions through various in-office means, such as ocular staining patterns. In one study, researchers showed temporal conjunctival rose bengal staining is a sensitive test for SS and perhaps can be used to differentiate SS-DED from other forms of dry eye.[30] Additionally, patchy, central corneal fluorescein staining may also be a good indicator of SS.[31]

In some states, optometrists can use the Sjö test (Bausch + Lomb), a simple in-office blood test, to diagnose this condition early. Otherwise, patients can be sent to a local laboratory for testing. Referral to a rheumatologist is still advised even when blood tests are negative but symptoms and signs of SS are present. If you or the rheumatologist suspect a false negative test, salivary biopsy may be necessary to confirm the diagnosis.[32]

Customized care. Once the diagnosis is confirmed with a rheumatologist, ODs play a key role in providing customized DED management with new clinical practice guidelines for SS in the United States.[33] The recommended DED treatment strategies include both conventional and unconventional therapies such as artificial tears, topical corticosteroids, punctal plugs, autologous serum eye drops (ASEDs) and therapeutic contact lenses.[33]



At top, sodium fluorescein corneal staining of a Sjögren's syndrome patient prior to scleral contact lens wear. At bottom, reduced sodium fluorescein corneal staining of the same patient three weeks post-scleral lens wear.

Since investigators believe SS-DED is driven by an inflammatory mechanism, anti-inflammatory or immune-modulatory agents are well-suited to treat DED in these patients.[34] Research shows Restasis (cyclosporine 0.05%, Allergan) is an effective treatment and may also help SS patients with evaporative DED by treating MGD.[35-37]

Compounded topical cyclosporine at higher concentrations—including cyclosporine 0.5% to 2% suspension in gum cellulose and cyclosporine 0.2% ointment—may help patients unsuccessful with Restasis.[38] Sun Pharma recently announced successful Phase 3 clinical trial results for Seciera (cyclosporine A 0.09% ophthalmic solution).[39]

Tacrolimus is an another option for SS-DED. In one study, topical 0.03% tacrolimus eye drops improved tear film stability and ocular surface status in SS-related DED.[40]

Xiidra (lifitegrast ophthalmic solution, Shire) has been successful in randomized, double-masked, multicenter, placebo-controlled studies in improving DED symptoms and signs—promising results for SS patients.[41]

Omega-3 FA supplementation is also a good anti-inflammatory management option. However, high intake of omega-3 FAs can cause excessive bleeding in some patients, and patients should take then under a physician's care.[25]

Although SS patients are treated with Plaquenil (hydroxychloroquine, Sanofi-Aventis) systemically, it is known to cause retinopathy and its benefit for SS-DED is controversial.[42,43,44] While research shows Plaquenil improves dry eye in primary Sjögren's subjects, other studies show no clinical benefit.[42,43] Clinicians should follow the 2016 updated visual screening guidelines for long-term Plaquenil therapy.[44]

The secretagogues oral pilocarpine and cevimeline are indicated for dry mouth in SS patients, but their benefit for SS-DED is controversial.[31] While some research shows they improve DED symptoms and signs, others show they were ineffective in improving DED signs.[25]

Recently, scleral gas permeable lenses have gained attention for their success in treating SS-DED. Although no studies specifically investigate the outcomes of scleral lenses in patients with SS, these patients have been

included in several study groups and have benefited from scleral lenses.[45-47] Anecdotally, practitioners find SS patients have improved symptoms and signs with scleral lenses and indicate a significant improved quality of life.

SS-DED can be severe, and other therapies typically reserved for refractory DED such as sutureless amniotic membranes and autologous serum drops may be helpful for these patients. Sutureless amniotic membranes act as bandage and restore the epithelium and control inflammation.[48] ASEDs also promote epithelialization and can be an effective DED treatment for SS patients.[49-51] Punctal plugs combined with ASEDs may have an additive effect and perhaps should be considered in recalcitrant cases after using ASEDs alone.[51] ■

Dry eye disease is associated with several systemic diseases with serious complications. Fortunately, optometrists are in a vital position to address these patients' dry eye needs and manage their holistic health approach.

Dr. Mickles is associate professor at Nova Southeastern University College of Optometry and its Dry Eye Service coordinator. She is a fellow of the American Academy of Optometry.

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Do APDs Matter? It's All Relative

Here are the staple points to consider about this important clinical marker.

By Bisant A. Labib, OD

The testing of the pupillary response to light is routinely performed by the eye care practitioner and imparts valuable information regarding the presence, laterality or magnitude of diseases that mainly affect the optic nerve, such as glaucoma.[1] A relative afferent pupillary defect (APD), when present, is an important marker in the evaluation of unilateral or asymmetric function of the anterior visual pathway.[1-4] It serves as an indication of reduced afferent input from the retina or optic nerve when compared with the fellow eye.[3]

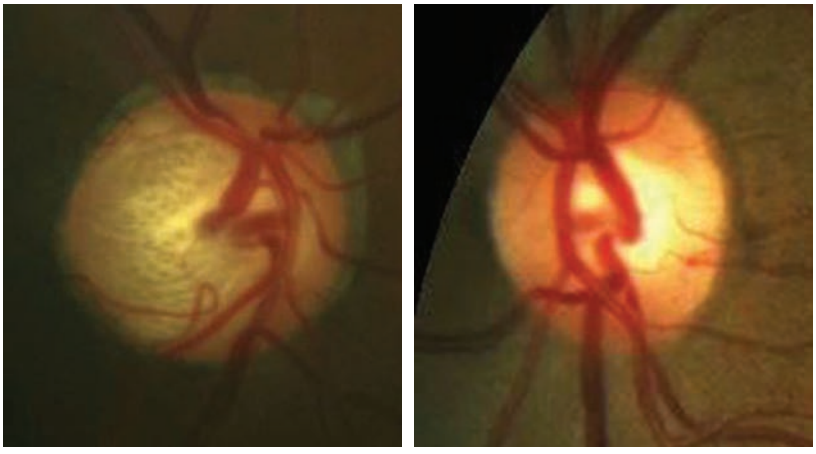
Because it's so commonplace, clinicians may tend to focus more on its diagnostic role than the physiology being tested. Understanding both, however, will provide a fuller picture of your patient's pathology.

The A&E Channel

The pupil response to light is best explained through the understanding of the light reflex pathway, which is comprised of two arms: the *afferent* and the *efferent*.

As the first arm, the afferent portion signifies the nerve impulse sent from stimulation of the retinal ganglion cells (RGCs) to the optic nerve by light, continuing down the optic tract, decussating at the chiasm and ultimately reaching the pretectal nucleus of the midbrain.

The efferent arm consists of the nerve impulse that exits the midbrain, traveling along the oculomotor nerve to the ciliary ganglion, resulting in constriction of both pupils in a healthy patient.[5]



Unilateral glaucoma, as seen in these fundus photos, more severe in the right eye than the left, could elicit an APD in the right eye.

APD and Glaucoma

Determining the presence or absence of an APD is of great clinical value, particularly in guiding the practitioner in the course of treatment for their glaucoma patients. Though glaucoma is typically a bilateral condition, it often initially presents unilaterally or bilaterally with variation in severity between the two eyes. It is well documented that patients with glaucoma exhibit lower amplitude, slower velocity and acceleration of pupil subjects when compared with healthy controls.[6] Therefore, practitioners' ability to determine the presence of an APD is a critical step in caring for patients suffering from glaucoma as well as those suffering from other types of optic neuropathies.

Checking for an APD

Three major methods exist to determine the presence of an APD *(Table 1)*. Although these tests are generally tried and true, one is best left alone with a preference for the first two.

Swinging flashlight test. Most commonly, the presence of an APD is evaluated in office using the swinging flashlight test, in which each pupil is illuminated and the velocity and amplitude of the pupillary response is compared.[2] In a healthy patient, light stimulation into one pupil results in equal constriction of both. In a patient with an APD, the affected eye will have less pupillary constriction or will dilate in significant defects.[6]

The response can be quantified using a neutral density filter that is placed to dim the eye without the suspected APD. The filter is placed starting with 0.3 logs, and then

Table 1. Comparison of Pupil Testing Methods and Their Application

Pupil Test	Strengths	Weaknesses	Common Uses
Pupil Cycle Time	• Can be tested unilaterally	• Long test duration and lack of test standardization	• Not used
Infrared Video Pupillography	• Quantitative and objective method • Very sensitive	• Requires more high tech equipment • APD recorded in healthy subjects at times	• Clinical trials
Swinging Flashlight Test	• Easily performed chair side	• Subjective and requires technical skill for subtle APDs	• Routine eye exams

gradually increasing the degree of light absorption. Once the APD is neutralized, the endpoint is reached as measured by the log unit necessary to achieve neutrality.[5] Even in the case of a unilateral, fixed pupil, an APD is detectable through observation of the direct or consensual response of the reactive pupil.[5] Animal studies show that a 0.6 log unit APD is observed in response to an approximate unilateral loss of 25% to 50% of RGCs.[2,6]

The test has its limitations, in that it requires a considerable amount of expertise by the examiner, especially in the detection of subtle defects.[1] Due to its subjective nature, discrepancies between examiners are not uncommon, and small APDs are sometimes missed.[7] Other difficulties in assessment of this test lie in patients with anisocoria, dark irises or very small pupils.[2] Young patients may also exhibit hippus, which causes slight fluctuations in pupil size despite constant light stimulation.[5]

Infrared video pupillography. This technique, first introduced in 1958, illuminates the pupil by an infrared source and images it by a video camera, allowing observation of pupil characteristics in dark settings.[6,7] By adjusting the stimulus between the two eyes until they reach the same amplitude, an APD can then be measured quantitatively by comparing the difference or latency between eyes.[6] The precise calculation uses the ratio of the amplitude to the latency of the light reflex in each eye.[7]

The RAPDx (Konan Medical) device is used most often in clinical studies to objectively calculate an APD using log units.[8] This high resolution device has demonstrated greater sensitivity in the detection of smaller APDs when compared with the swinging flashlight method.[6] The machine's duration, interval and intensity of the stimulus are fixed, maintaining a stable light reflex and eliminating examiner discrepancy.[7]

Because of the significance of APD testing and measurement in determining the diagnosis and progression of glaucoma, several studies investigated infrared video pupillography in its accuracy with glaucoma patients. One study reported the presence of an APD in 56% of glaucoma patients using pupillography, compared with only 29% detection using the swinging flashlight method.[2,3]

One disadvantage is that a small but detectable APD is sometimes observed in healthy subjects, limiting the specificity of this test modality.[6]

Besides the detection of an APD, pupillography may be used in other cases when pupil size is of diagnostic importance, such as differentiating physiological anisocoria from Horner's syndrome or measuring pupil size for refractive surgery.[9]

Pupil cycle time (PCT). This technique, first presented in 1944, requires a much longer duration and observation of the patient through the slit lamp.[6] Unlike the swinging flashlight test, this method does not require the fellow eye for comparison. Instead, a 0.5mm thick horizontal beam of light is oriented right at the inferior pupil margin, resulting in constriction. The beam remains in place until the constricted iris blocks the light and then re-dilates until the edge is placed in its initial position at the edge of the inferior pupil margin. The cycle continues and the oscillations are recorded to calculate the PCT using the average of five total measurements and multiplied by 30.[6]

No significant information is available regarding the exact correlation between prolonged PCT and optic nerve disease such as glaucoma. Additionally, the intensity of light stimulus has not been standardized, which renders this technique one with little clinical value.[6]

Clinical Significance

Aside from glaucoma, optic neuropathies such as ischemic optic neuropathy, optic neuritis, optic nerve compression from a mass lesion or traumatic optic neuropathies may also manifest with an APD in the affected eye.[5]

The correlation between an APD and macular disease is not yet well established. As 30% to 50% of all RGCs are located in the macula, it is likely to contribute to the pupillary light response.[3] Recent emphasis

has been placed on the detection of early glaucoma through analysis of macular integrity, but the degree to which the macula must be affected to elicit an APD is typically substantial.[3] This is also true for macular diseases such as age-related macular degeneration.[4]

Anterior segment conditions, such as dense corneal opacities and amblyopia, do not affect the pupil response to light. Though it is rare for a cataract to cause an APD, one study reported that in severe, unilateral cases when vision is counting fingers or worse, an APD is often observed but, in the better-seeing eye, that later disappeared following extraction. The mechanism for this was undetermined, but the researchers concluded that in these particular cases, a defect in the visual pathway should only be suspected when the APD is noted in the eye with the dense cataract.[10]

Pupil testing is routinely performed on all patients, and can provide critical clinical insight, especially in cases of glaucoma, which is often difficult to diagnose in the early stages. Understanding the physiology governing our ability to evaluate pupil function and make sense of the data is imperative for not only narrowing down the disease differentials, but also in monitoring the course and progression of ocular disease.

Testing is a means to end. Prompt diagnosis allows us to heal our patients and stave off damage. Our abilities to diagnose, ultimately, are grounded in an understanding of the processes by which the various components that comprise the ocular system function and operate. ■

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Managing Migraine Headache

Migraine headache frequently has ocular symptoms and associations, and ODs need to be ready to care for these patients. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

Migraine, which affects more than 20% of people at some point in their lives, is considered the most common disabling brain disorder.[1,2] It is a type of primary headache, meaning that the pain is due to the headache condition itself, not secondary to another cause, such as sinusitis.[1] When chronic, it may severely impair a person's ability to accomplish everyday activities.

Migraine has a strong genetic component, and the majority of people living with migraine are women.[1,2] In addition, women older than 45 years of age with a history of migraine have a higher incidence of major cardiovascular disease.[1,3]

In our last column, we reviewed the basics of primary and secondary headaches, as well as the epidemiology, pathophysiology and diagnostic guidelines for migraine headache disorders.[3] Here, we focus on how optometrists can work with other members of the health care team to bring relief to these patients.

Migraine Aura

Migraine attacks may present with or without preceding aura (*Table 1*). When present, most auras are visual, commonly consisting of floaters, flashes, zigzag patterns and blind spots. Patients often report a severe, unilateral, throbbing or pulsing pain, which may be accompanied by visual alterations and photophobia.[1,2]

For some individuals, the typical aura is always followed by the migraine headache. For others, however, the attacks with aura are followed by a less distinct headache, or even no headache. Finally, a number of patients have, exclusively, the typical aura without headache.[1,4] There are several subtypes of migraine with aura, as well as episodic syndromes that may be associated with migraine.[4]

In cases where an aura occurs for the first time after age 40, or when the aura symptoms consist exclusively of negative phenomena (e.g., hemianopia), or if the aura is either prolonged or very short, clinicians should rule out other causes, such as transient ischemic attacks.[1,2]

Eye care clinicians should be on the lookout for two forms of headache that require a more specific diagnosis and directly involve the eye and visual system:

Retinal migraine, a form of migraine with aura, is a rare cause of transient monocular visual loss. It is characterized by repeated attacks of unilateral visual disturbances, including scintillations, scotomata or blindness, all associated with the headache. Researchers have noted cases of permanent monocular visual loss associated with retinal migraine.[4] Appropriate investigations are required to exclude other causes of transient monocular blindness (*Table 2*).

The term ***ophthalmoplegic migraine*** is a misnomer in that it is likely not a variant of migraine but rather a recurrent cranial neuralgia. A more appropriate name might be "ophthalmoplegic cranial neuropathy" or "ophthalmoplegia with migraine-like headache."[5,6]

Table 1. Types of Migraine[1,4]

Type/Name	Description
Migraine without aura (previously known as a common migraine)	Recurrent headache with attacks lasting four to 72 hours. Unilateral location, pulsating quality, moderate to severe intensity, aggravation by routine physical activity and association with nausea, photophobia, phonophobia or a combination of all three.
Migraine with aura (previously known as a classic, complicated, ophthalmic, hemiplegic or aphasic migraine)	Recurrent attacks lasting minutes. Unilateral, fully reversible visual, sensory or other central nervous system symptoms that develop gradually and are usually followed by headache and associated migraine symptoms.
Chronic migraine	Headache occurring on 15 or more days per month for more than three months, which has the features of migraine headache on at least eight days per month.

Symptoms, Triggers and Treatments

The United Kingdom's National Institute for Health and Care Excellence provides

published guidelines on the diagnosis and treatment of migraine, and both the American Headache Society and American Academy of Neurology have expert consensus guidelines.[1,2,5] In general, three approaches exist to managing migraine: lifestyle and trigger management, acute treatments (administered during an attack or exacerbation of chronic pain) and preventive treatments (medication or other interventions designed to reduce the tendency to experience attacks).

This is one patient's approximation of the zig-zag visual disturbance experienced as a migraine aura. According to the patient, it moves and vibrates, expanding and slowly fading away over the course of about 20 minutes.

Lifestyle. Many patients find that lifestyle adjustments such as regularizing meals and sleep can reduce the frequency of their attacks. Patients should avoid triggering factors that precipitate a migraine attack, such as fatigue, lack of sleep, stress, certain foods and use of vasodilating agents.[1,2] To help patients better avoid triggers, clinicians can suggest patients use a daily diary to document the headaches and daily activities that might have triggered them. Patients may need to reduce or discontinue any medications that exacerbate their headaches such as oral contraceptives and hormone replacement therapy.[1,2,5]

Acute management. Combination analgesics containing aspirin, caffeine and acetaminophen are an effective first-line abortive treatment for migraine. Ibuprofen at standard doses is also effective for acute migraine treatment.[1,2] As opposed to analgesics, triptans and ergotamines are more migraine-specific classes of medications (*Table 3*).

Acute treatment is most effective when given within 15 minutes of pain onset and when pain is mild.[2] Antiemetics (e.g., chlorperazine or promethazine) can be used to treat the emesis associated with acute migraine attacks.

Preventative medication. Some form of medication or other treatment is almost always necessary for those with chronic migraine. Roughly 3% to 13% of migraine sufferers are using preventive therapy, although research suggests the need is much greater, with estimates that 38% of patients with migraine would benefit from a preventive agent (*Table 4*).[1,2,5]

The goals of preventive therapy are to reduce attack frequency, severity or duration, improve responsiveness to acute attacks and reduce general disability. Many medications are used to treat migraine prophylactically, including antiepileptic drugs, beta and calcium channel blockers, several subtypes of antidepressants and some antihypertensives, as well as supplements, herbs and vitamins.[8]

Many prophylactic meds for migraine work by inhibiting cortical spreading depression (CSD), which is considered the basis of migraine aura.[8] Others have antiadrenergic or serotonin modulatory effects, or enhance the effect of the neurotransmitter gamma-aminobutyric acid, leading to a decrease in neuronal firing.[8]

In addition, a 2002 survey shows that more than 85% of patients suffering from migraine headache use complementary and alternative medicine (CAM)—such as herbal agents, deep breathing and meditation, yoga and progressive relaxation—and 60% felt the CAM provided some relief.[9] Biofeedback and behavioral changes are frequently part of the care for a migraine patient, for example.

Table 2. Differential Diagnosis of Transient Monocular Vision Loss[7]

- Orbital/ocular ischemia (ophthalmic artery)
- Retinal ischemia (central retinal artery and its branches, central retinal vein)
- Optic nerve ischemia (short posterior ciliary arteries/ophthalmic artery)
- Choroidal ischemia (posterior ciliary arteries)
- Hyphema
- Angle closure glaucoma
- Retinal detachment
- Papillitis
- Optic nerve compression
- Uhthoff's phenomenon (demyelination)

Studies have demonstrated the effectiveness of the herb butterbur (*Petasites hybridus*) in preventing migraines.[10] Patients on butterbur require monitoring of liver enzymes.[10]

Table 3. Acute Migraine Treatment Options[1,2,5]

- Paracetamol (1g)
- Aspirin (900mg to 1200mg)
- Ibuprofen (400mg to 800mg)
- Naproxen (250mg to 500mg)
- Triptans
  - Sumatriptan (50mg to 100mg orally, 10mg to 20mg nasally, 6mg subcutaneously)
  - Almotriptan (12.5mg)
  - Eletriptan (40mg to 80mg)
  - Frovatriptan (2.5mg)
  - Naratriptan (2.5mg to 5mg)
  - Rizatriptan (5mg to 10mg orally or sublingual melt)
  - Zolmitriptan (5mg to 10mg orally or sublingual melt, 5mg nasally)
- Combinations
  - Sumatriptan (50mg) and naproxen (250mg to 500mg)

(All of the above are taken alone or with domperidone (10mg orally) or an alternative antiemetic)

- Single-pulse transcranial magnetic stimulation
- Vagal nerve stimulation

Table 4. Preventive Treatments for Chronic Migraine[1,2,5]

	Starting Dose	Target Dose
First Line		
β-blockers		
• Propranolol	10mg TID	40mg to 80mg TID
• Metroprolol	25mg BID	100mg BID
• Atenolol	25mg once daily	100mg BID
Angiotensin blockers		
• Candesartan	4mg once daily	12mg to 16mg once daily
Tricyclics		
• Amitriptyline	10mg at night	75mg to 100mg at night
• Nortriptyline	10mg at night	75mg to 100mg at night
• Dosulepin	25mg at night	75mg to 100mg at night
Second Line		
Anticonvulsants		
• Topiramate	12.5mg at night	50mg to 100mg BID
• Sodium valproate	200mg at night	400mg to 800mg BID
Flunarizine	5mg once daily	5mg to 10mg once daily
Onabotulinum toxin A (Botox)	31 small injections of 5 units each placed at prescribed locations over the forehead	
Supplements		
Riboflavin (vitamin B2)	400mg daily	
Magnesium citrate (or taurate)	600mg daily	

Other natural supplements such as riboflavin (vitamin B2), coenzyme Q10, melatonin and magnesium appear to be effective and well tolerated for migraine prophylaxis as well.[8,11-13]

A basic working knowledge of the common primary headaches is key to properly diagnosing patients with migraine. A comprehensive ophthalmic workup with ancillary testing such as threshold perimetry and cranial nerve evaluation can help the optometrist rule out more serious secondary headache syndromes. Having a game plan for managing these patients will ensure they get the relief they need. Comanaging migraine sufferers with neurologists and other headache specialists can confirm the diagnosis and provide a substantial number of acute and preventive treatment options that can improve the lives of our patients. ■

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Through the Eyes of a Child

Can these images of a child with suspected poor distance vision reveal a diagnosis?

By Jenna Blechman, OD, and Mark T. Dunbar, OD

A couple presented to our clinic with concerns about their 23-month-old who, they say, was "cross-eyed since birth." They also had some concern about his ability to see at distance. The child was delivered full-term with normal growth and development. He had no pertinent medical history. Family history was reported as unremarkable.

Upon exam, the child was able to fixate and follow with each eye. His pupils were equal, round and reactive to light, without afferent defect. Confrontation visual fields appeared full but with poor cooperation. Extraocular motility testing was full; we observed an esotropia. Anterior segments of both eyes were unremarkable. The patient was dilated and a limited fundus exam was performed, but based on the findings, we felt an exam under anesthesia (EUA) was warranted.

During the EUA, intraocular pressures (IOP) by Tono-pen (Reichert) measured 10mm Hg OD and 9mm Hg OS. Fundus exam, indirect ophthalmoscopy with scleral depression and fluorescein angiography (FA) were performed. Fundus images (*Figure 1*) and FA images (*Figures 2 and 3*) were obtained.

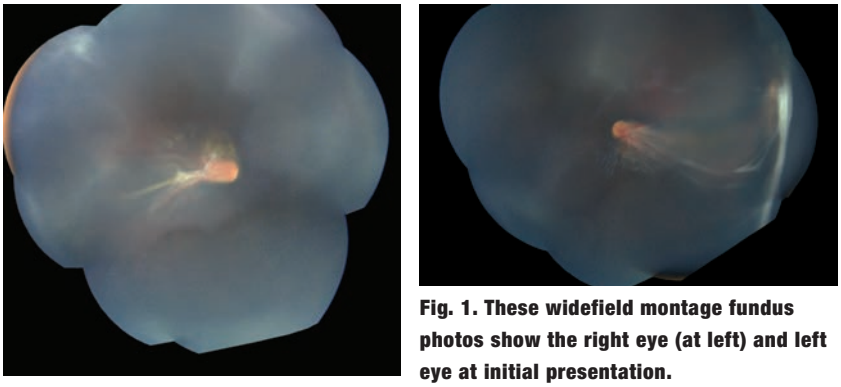

Fig. 1. These widefield montage fundus photos show the right eye (at left) and left eye at initial presentation.

Take the Retina Quiz

1. What is the likely diagnosis?
a. Retinopathy of prematurity.
b. Familial exudative vitreoretinopathy.
c. Coats' disease.
d. *Toxocara canis* infection.

2. What does the FA reveal?
a. Avascular tissue.
b. Temporal dragging of vessels.
c. Both a and b.
d. This is a normal FA.

3. What is the inheritance pattern of this condition?
a. Autosomal dominant.
b. Autosomal recessive.
c. X-linked recessive.
d. All of the above are possible.

4. What other testing is indicated in the management of this patient?
a. Genetic testing of patient and family members.
b. Visual field testing.
c. MRI of brain and orbits.
d. No additional testing.

For answers, see page 114.

Discussion

The EUA revealed a large retinal fold involving the macula of both eyes with temporal dragging of the vessels. In the right eye, we observed an abnormal vascularity and significant areas of avascular tissue, which was highlighted by the

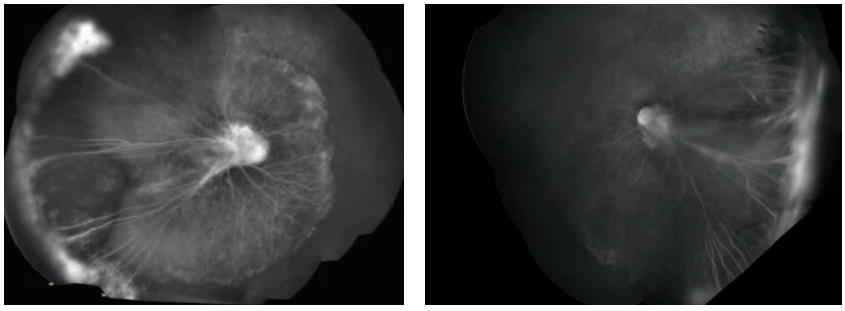

Fig. 2. Do these montage FA images of the patient's right eye (at left) and left eye, taken under anesthesia, point to a diagnosis?

FA. We saw no retinal detachment. Exam of the left eye revealed some vitreoretinal traction, especially towards the inferotemporal periphery, but we observed no tractional retinal detachment. Peripheral neovascularization was evident on both clinical exam and FA. Based on the clinical presentation, a diagnosis of familial exudative vitreoretinopathy (FEVR) was made. This was later confirmed by genetic testing. The patient was treated with peripheral laser 360 degrees in both eyes.

FEVR is, as its name suggests, familial and can be inherited in an autosomal dominant, autosomal recessive or X-linked recessive pattern.[1-3] It is caused by mutations in FZD4, LRP5, TSPAN12 and NDP genes, which impact the wingless/integrated (Wnt) receptor signaling pathway.[3] Disruption of this pathway leads to abnormalities of vascular growth in the peripheral retina.[2,3]

It is typically bilateral, but asymmetric, with varying degrees of progression over the individual's lifetime. Age of onset varies, and visual outcome can be strongly influenced by this factor. Patients with onset before age three have a more guarded long-term prognosis whereas those with later onset are more likely to have asymmetric presentation with deterioration of vision in one eye only.[2-3] However, because FEVR is a lifelong disease, these patients are at risk even as adults.[2] Ocular findings and useful vision typically remain stable if the patient does not have deterioration before age 20.[2,4] Due to the variability and unpredictability of the disease course, patients with FEVR should be followed throughout their lifetime.

Clinical presentation can vary greatly. In mild variations, patients may experience peripheral vascular changes, such as peripheral avascular zone, vitreoretinal adhesions, arteriovenous anastomoses and a V-shaped area of retinochoroidal degeneration.[4] Severe forms may present with neovascularization, subretinal and intraretinal hemorrhages and exudation.[4] Neovascularization is a poor prognostic indicator and can lead to retinal folds, macular ectopia and tractional retinal detachment.[2,4]

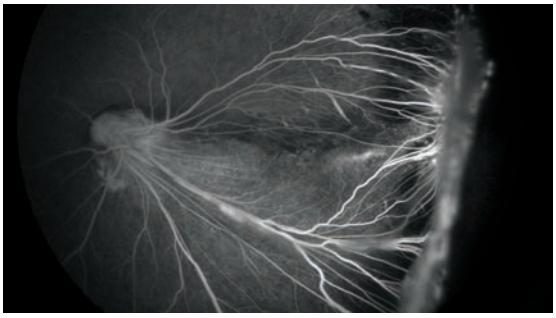

Fig. 3. Close-up of the left eye FA at initial presentation.

Widefield FA has been crucial in helping to understand this disease, as well as helping to confirm the diagnosis. An abrupt cessation of the retinal capillary network in a scalloped edge posterior to fibrovascular proliferations can be made using FA.[2,3,5] Patients can also show delayed transit filling on FA as well as delayed/patchy choroidal filling, bulbous vascular terminals, capillary dropout, venous/venous shunting and abnormal branching patterns.[2,3,5]

The staging of FEVR is similar to that of retinopathy of prematurity. The first two stages involve an avascular retinal periphery with or without extraretinal vascularization (stage 1 and 2, respectively).[4] Stages three through five delineate levels of retinal detachment; stage 3 is subtotal without foveal involvement, stage 4 is subtotal with foveal involvement and stage 5 is a total detachment, open or closed funnel.[4] Because there was neovascularization in the absence of retinal detachment, our patient was considered to have stage 2.

Treatment is based on the stage of the disease. Stage 1 does not require treatment and should be observed.[4] Neovascularization (stage 2) responds well to laser ablation or cryotherapy.[2,4] Eyes with retinal detachments (stages 3 through 5) require surgery, with earlier stages requiring scleral buckles and later stages ultimately needing vitrectomy.[2,4]

More recently, the efficacy of anti-VEGF intravitreal injections has been studied. In one study, these injections, as an in adjunct with laser, helped early stages achieve stabilization, but further investigation is needed.[6]

Our patient was followed every three months with EUA and received laser five more times in each eye. Subsequently, at the age of 2.5 years, a tractional retinal detachment developed in the left eye, and the patient underwent a pars plana vitrectomy/scleral buckle/membrane peel procedure. At the two month postoperative visit, his uncorrected vision, at near, was 20/60 in the right eye and 20/160 in the left eye. He continues to be monitored closely with EUA. ■

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Make Your Brown Eyes Blue

A review of cosmetic procedures suggests that vanity often has a high cost.

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

Everybody loves blue eyes. According to a recent study, people rate blue eyes as one of the five most attractive characteristics when assessing beauty.[1] Perhaps this is because blue eye color is comparatively rare. In the United States, about 27% of individuals have blue eyes, according to one survey.[2] So, what is an unhappy, brown-eyed individual to do?

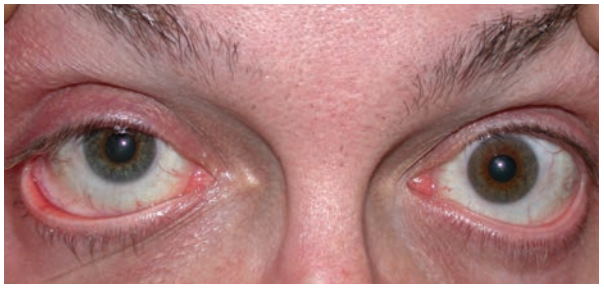

Photo: Aaron Bromer, OD

Patients with heterochromia, as seen here, have some procedural options, but with varying degrees of safety.

Since the mid-1980s, many have opted for cosmetic soft contact lenses to help satisfy their desire for differently colored eyes. Veteran optometrists will likely remember (with varying degrees of fondness) the Durasoft 3 Colors line (Wesley Jessen), or the Mystique line (Coopervision). These lenses became a fashion trend in the 1990s, growing to even greater popularity after the introduction of disposables.

But with this new fad came difficulties. The demand for colored contact lenses led to instances of unlicensed sale and dispensing by unqualified individuals, in such unlikely places as beauty shops and flea markets. This in turn led to increased cases of sight-threatening complications, such as microbial keratitis.[3] While opaque cosmetic contact lenses are still available today, they are viewed with greater skepticism and dispensed far less frequently than in their heyday.

Another, more radical effort to alter eye color arose several years ago when physicians in Central America began using the NewColorIris implant (Kahn Medical Devices). The device, and others like it, consists of an annular silicone iris diaphragm with a central opening and several small peripheral flaps designed to anchor it in the anterior chamber. Prosthetic iris devices like these are not new; they were originally developed to treat pathologic conditions such as iris colobomas, traumatic iris defects and aniridia.[4]

But the makers of NewColorIris instead began marketing its implant to healthy patients as "a safe and permanent way to modify eye color," with a "favorable risk profile over cosmetic contact lenses."[5,6]

In reality, a substantial number of patients who had these devices implanted experienced complications and irreversible vision loss.[5-15]

Complications

Glaucoma was the most common complication associated with these implants.[6] Out of 128 cases reported between 2008 and 2016, 59 developed secondary glaucoma as a result of the procedure.[6] Elevations of intraocular pressure (IOP) were recorded as high as 68mm Hg.[13] Forty-four of the 128 eyes suffered severe endothelial cell loss, with some having subsequent corneal decompensation.[6] The etiology of these complications is likely threefold:

(1) Compression of the trabecular meshwork, possibly causing chronic inflammation.

(2) Contact between the implant's flaps and peripheral endothelial cells, causing cell death.

(3) Direct contact with the iris, leading to atrophy and pigment dispersion.[6]

Although warranted in these cases, removal of these devices introduced additional complications, including hyphema, suprachoroidal hemorrhage and iatrogenic iris defects.[14,15]

Laser Options

Despite the significant risks demonstrated through prior modalities, there are still potential avenues for those wishing to modify their natural eye color.

The latest reported procedure to emerge involves laser treatment to the iris surface. NewEyes Laser (Clínica Eyecos) has been advertising this technology on its website for several years. Another organization called Mantis, located in Istanbul,

Turkey, describes itself as "a high scale and quality organization that delivers a safe and advanced eye color changing procedure to its patients."[16] In the United States, a company called Stroma Medical is developing a platform to deliver this technique into the hands of willing ophthalmologists, projecting an estimated base of 2.34 million patients a year, with a potential revenue for physicians of $10.5 Billion.[17] In 2015, the American Academy of Ophthalmology warned consumers about laser surgery to change eye color, stating that "the procedure has yet to undergo clinical trial testing in the United States to determine any potential safety risks."[18] The organization expressed concern that liberation of pigment during the procedure could cause glaucoma, and warned of other potential risks such as uveitis.[18]

The proposed mechanism of action for this treatment involves destruction of intracytoplasmic melanosomes in the anterior iris; the melanin granules absorb laser energy via a process known as selective photothermolysis.[19,20] It is this same principle that is used in selective laser trabeculoplasy (SLT), which causes structural changes in the trabecular meshwork that help to reduce IOP.[18]

Since all irises contain only brown pigment, and the perceived hue is merely the result of pigment density, it is theoretically possible to lighten the color of the iris by reducing the amount of melanin in the tissue.

Lasers in the Literature

Only two reports in the published literature detail this procedure. The first was an experimental animal model in which energy from a frequency-doubled 532nm wavelength Nd:YAG laser was applied to the iris surface.[19] The animals, all of whom had brown irises, were treated in a series of three sessions spaced two weeks apart. Clinical examinations were conducted after each session to assess iris color (as compared with baseline), postoperative inflammation and IOP. The authors reported a noticeable iris color change, which was most evident at 60 days following the initial procedure. The researchers noted no statistically significant elevation of IOP in any of the subjects and only a mild inflammatory reaction that was energy dependent (i.e., eyes treated with a higher energy pulse had greater inflammation) and resolved without treatment.[18] Additionally, none of the eyes showed complications such as corneal edema, hypopyon, posterior synechia, transillumination

defects, lens opacity, vitreous haze or alteration of pupillary function.[19]

The second report detailed a single patient who was treated with an identical 532nm Nd:YAG laser for unilateral sectoral heterochromia iridis.[20] The treatment involved three sessions over three consecutive days, and the cycle was repeated one month later. Pilocarpine 2% was instilled prior to each laser application for miosis, and nepafenac 0.1% was given QID for one week after treatment to suppress inflammation. The procedure was successful in producing a homogenously blue iris, more closely resembling the fellow eye. There were no postoperative complications reported.[20]

It is too early to tell if laser treatment will emerge as a viable method for altering eye color. More clinical trials are certainly necessary to determine if this procedure is indeed safe, effective and predictable. But if the past is any indication, there will certainly be no shortage of individuals willing to give it a try. As optometrists, we must remain informed about new techniques like these to provide accurate information to our patients and effectively safeguard their ocular health. ■

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Contact Lenses

Patient-Friendly Packaging

Alcon launched new US-specific packaging for its Dailies AquaComfort Plus spherical contact lenses to assist patients with proper lens wear and care. The new packaging features new patient-friendly educational elements, including detailed insertion and removal illustrations plus a toll-free patient helpline, email address and website for more support. Additionally, the US flag symbol will appear on the new packaging to identify the boxes as specific only to US patients.

Visit www.dailies.com.

Toric Scleral Lens

Blanchard has added the Sym-Toric scleral lens design, a front toric that the company says enhances patient comfort, visual performance consistency and simplifies the fitting experience when using the Onefit Scleral Lens Platform diagnostic lens set. The lens does not rely on the prism ballast or toricity in the haptic as is typically used to stabilize axis orientation. While keeping the same edge elevation 360 degrees, the posterior surface aligns to the different radii values of the cornea and sclera.

Visit blanchardlab.com/products/onefit/.

Another Base Curve

Menicon has added a second base curve—8.4—for its Miru 1day Menicon Flat Pack daily disposable contact lenses, in addition to the existing 8.6 base curve. The 8.4 base curve was designed based on feedback from ODs seeking additional fitting options. The lenses are available in trial six-packs, 30-packs and 90-packs.

Visit www.meniconamerica.com.

More Scleral Approvals

Visionary Optics has received FDA clearance for its complete line of scleral contact lenses: Jupiter, Europa and Elara. Historically, FDA clearance for scleral lenses has been based solely on lens material. The approval allows doctors to choose from a variety of sclerals from multiple companies, based on their individual designs and case reports documenting efficacy, according to the company.

Visit www.visionary-optics.com.

Devices and Equipment

Epithelial Mapping via OCT

If you have Optovue's iVue or iFusion OCT system, you can now add corneal epithelial thickness mapping to the device, a first for the OCT category, says the manufacturer. Traditional measurements require high-frequency digital ultrasound and saline. Optovue says its new software, called epi-mapping, provides epithelial and stromal measurements in a fast, non-contact and easy exam. It is particularly helpful for keratoconic and dry eye patients, as well as to observe how the eye is healing after refractive and corneal surgery, the company says.

Visit optovue.com.

Lens Edging System

Coburn has introduced the HPE-410 Excelon Lens Edging System. Replacing the CPE-4000 model, the new HPE-410's design is fresh and modern, Coburn says. Its features were designed to keep up with optometrists' finishing demands. The machine comes with a stronger wheel with longer durability, and eliminates lens slippage with an adaptive lens chuck and position sensor, according to the company.

Visit www.coburntechnologies.com.

Portable Fundus Camera

Eyefficient is partnering with MediWorks to introduce a new handheld fundus camera, the FC160, in the United States. The manufacturer says the FC 160 is a sleek, portable, lightweight nonmydriatic camera with built-in wifi, Micro SD and Miracast connectivity that allows fast, easy dissemination of retinal images from remote locations. A 3.97-inch touch screen and five internal fixation targets allow doctors to capture images with ease.

Visit www.eyefficient.com.

Artificial Tear

New Ocular Lubricant

The Mentholatum Company now offers a new artificial tear called Rohto Dry-Aid Drops, designed to help relieve dry eye symptoms. The manufacturer calls it a non-blurring eye drop with a formula that provides long-lasting relief, helping to restore the natural tear film and improving dry eye signs and symptoms.

Visit www.rohtoeyedrops.com. ■

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Diagnostic **Quiz**

No Case Too Small

By Andrew S. Gurwood, OD

History

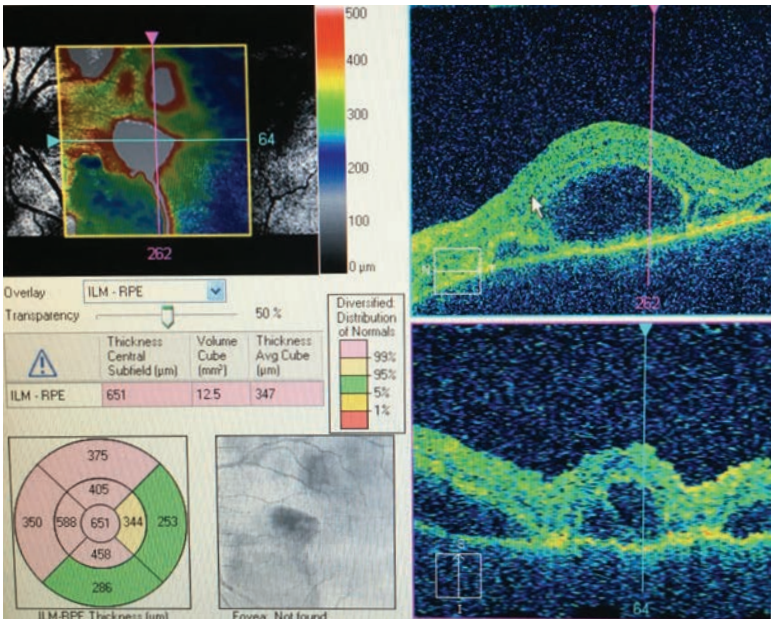
A 68-year-old female presented to the clinic for an ocular health check with a chief complaint of difficulty reading. Her ocular history was noncontributory. Her systemic history was remarkable for hypertension, for which she was compliant and properly medicated. She had no known allergies.

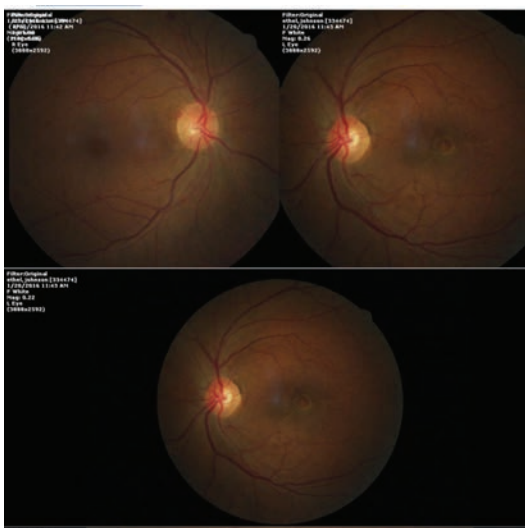
Diagnostic Data

Her best uncorrected visual acuities were measured at 20/25 OD and 20/50 OS at distance and near. Her external examination was within normal limits and there was no afferent pupillary defect. The refraction was negligibly different. Biomicroscopy found normal and healthy anterior segments with evidence of grade II+ nuclear cataracts. Goldmann intraocular pressures measured 14mm Hg OU. The pertinent dilated fundus findings are illustrated in the photograph and the optical coherence tomography (OCT).

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 do you believe is the patient's most likely prognosis? To find out, please visit us at www.reviewofoptometry.com. ■





The above OCT readout and the fundus images at left show a 68-year-old female patient who presented with trouble reading. Does anything on these images help to pinpoint her diagnosis?

Retina Quiz Answers *(from page 102):* 1) b; 2) c; 3) d; 4) a.

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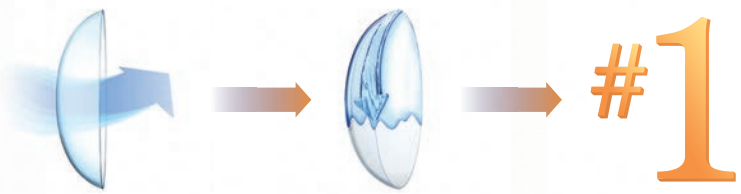

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