Earn 2 CE Credits — Guidelines on Glaucoma Progression, P. 78

REVIEW® OF OPTOMETRY

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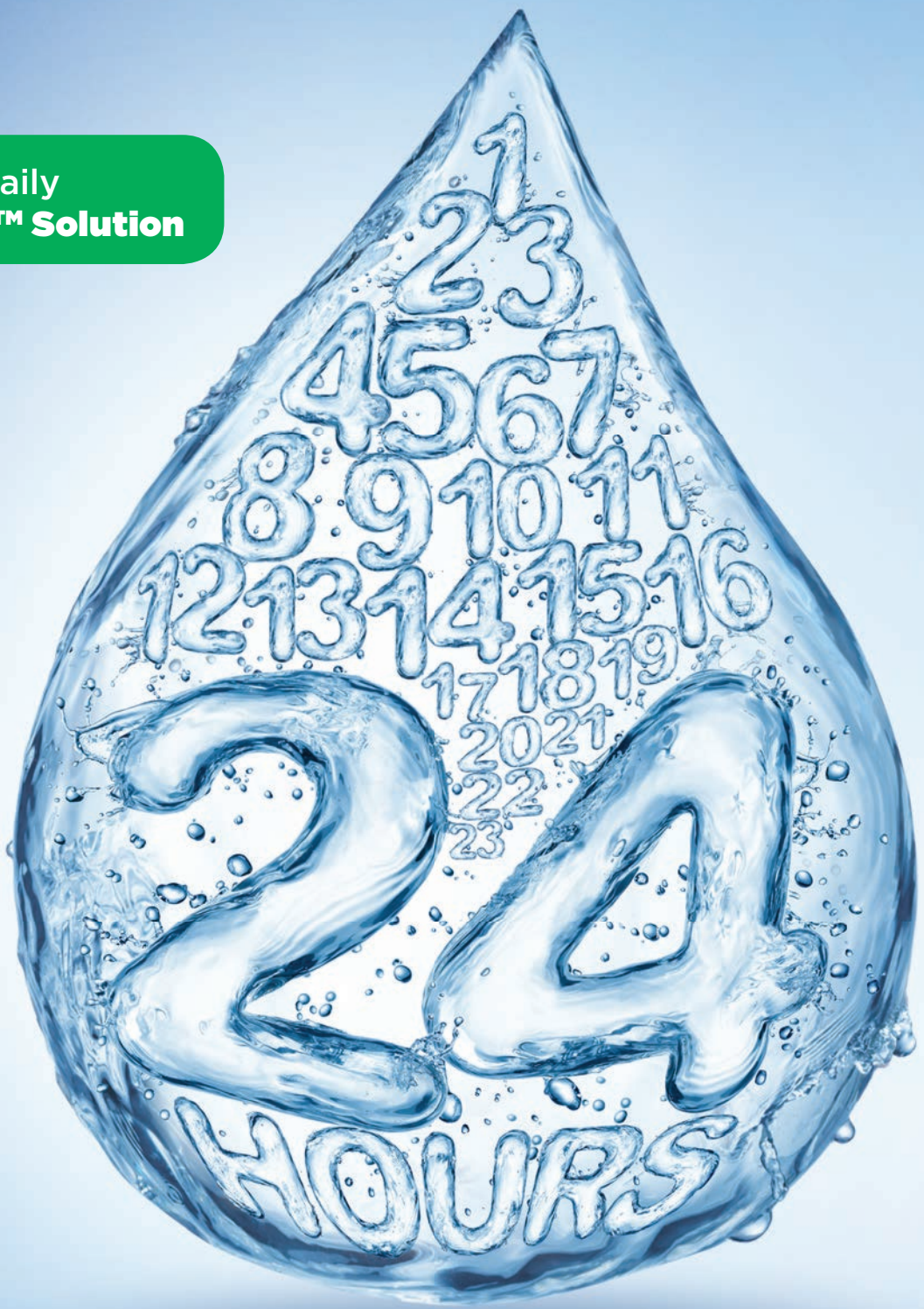
38th Annual

TECHNOLOGY REPORT

Once-Daily
PAZEO™ Solution



INDICATION AND DOSING

PAZEO™ Solution is indicated for the treatment of ocular itching associated with allergic conjunctivitis. The recommended dosage is to instill one drop in each affected eye once a day.

IMPORTANT SAFETY INFORMATION

As with any eye drop, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle to prevent contaminating the tip and solution. Keep bottle tightly closed when not in use.

Patients should not wear a contact lens if their eye is red. PAZEO™ Solution should not be used to treat contact lens-related irritation. The preservative in PAZEO™ Solution, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses and whose eyes are not red should be instructed to wait at least five minutes after instilling PAZEO™ Solution before they insert their contact lenses.

24 HOURS OF OCULAR ALLERGY ITCH RELIEF IN ONE DROP

Once-Daily **PAZEO™ Solution** for relief of ocular allergy itch:

- The first and only FDA-approved **once-daily** drop with demonstrated 24-hour ocular allergy itch relief[1]
- Statistically significantly improved relief of ocular itching compared to **PATADAY® (olopatadine hydrochloride ophthalmic solution) 0.2%** at 24 hours post dose (not statistically significantly different at 30-34 minutes)[1]
- Statistically significantly improved relief of ocular itching compared to vehicle through 24 hours post dose[1]

Study design: Two multicenter, randomized, double-masked, parallel-group, vehicle- and active-controlled studies in patients at least 18 years of age with allergic conjunctivitis using the conjunctival allergen challenge (CAC) model (N=547). Patients were randomized to receive study drug or vehicle, 1 drop per eye on each of 2-3 assessment days. On separate days, antigen challenge was performed at 27 (±1) minutes post dose to assess onset of action, at 16 hours post dose (Study 1 only), and at 24 hours post dose. Itching scores were evaluated using a half-unit scale from 0=none to 4=incapacitating itch, with data collected 3, 5, and 7 minutes after antigen instillation. The primary objectives were to demonstrate the superiority of **PAZEO™ Solution** for the treatment of ocular allergy itch. Study 1: **PAZEO™ Solution** vs vehicle at onset of action and 16 hours. Study 2: **PAZEO™ Solution** vs vehicle at onset of action; **PAZEO™ Solution** vs **PATADAY® Solution**, PATANOL® (olopatadine hydrochloride ophthalmic solution) 0.1%, and vehicle at 24 hours.[1-3]

PAZEO™ Solution: Safety Profile

- Well tolerated[1]
- The safety and effectiveness of **PAZEO™ Solution** have been established in patients two years of age and older[1]
- The most commonly reported adverse reactions, occurring in 2% to 5% of patients, were blurred vision, dry eye, superficial punctate keratitis, dysgeusia, and abnormal sensation in eye[1]

Once-daily dosing[1]

Give your patients 24 HOURS OF OCULAR ALLERGY ITCH RELIEF with once-daily PAZEO™ Solution[1]

IMPORTANT SAFETY INFORMATION (cont'd)

The most commonly reported adverse reactions in a clinical study occurred in 2%-5% of patients treated with either PAZEO™ Solution or vehicle. These events were blurred vision, dry eye, superficial punctate keratitis, dysgeusia, and abnormal sensation in eye.

For additional information on PAZEO™ Solution, please refer to the brief summary of the full Prescribing Information on the following page.

References: 1. PAZEO™ Solution Package Insert. **2.** Data on file, 2011. **3.** Data on file, 2013.

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 6/15 PAZ15099JAD

Pazeo™
(olopatadine hydrochloride ophthalmic solution) 0.7%

Pazeo™

(olopatadine hydrochloride ophthalmic solution) 0.7%

BRIEF SUMMARY

PAZEO (olopatadine hydrochloride ophthalmic solution) 0.7%. For topical ophthalmic administration.

The following is a brief summary only; see full prescribing information for complete product information.

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

Contamination of Tip and Solution

As with any eye drop, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle to prevent contaminating the tip and solution. Keep bottle tightly closed when not in use.

Contact Lens Use

Patients should not wear a contact lens if their eye is red.

The preservative in PAZEO solution, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses and whose eyes are not red, should be instructed to wait at least five minutes after instilling PAZEO before they insert their contact lenses.

ADVERSE REACTIONS

Clinical Trials Experience

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

In a randomized, double-masked, vehicle-controlled trial, patients at risk for developing allergic conjunctivitis received one drop of either PAZEO (N=330) or vehicle (N=169) in both eyes for 6 weeks. The mean age of the population was 32 years (range 2 to 74 years). Thirty-five percent were male. Fifty-three percent had brown iris color and 23% had blue iris color. The most commonly reported adverse reactions occurred in 2-5% of patients treated with either PAZEO or vehicle. These events were blurred vision, dry eye, superficial punctate keratitis, dysgeusia and abnormal sensation in eye.

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

There are no adequate or well-controlled studies with PAZEO in pregnant women. Olopatadine caused maternal toxicity and embryofetal toxicity in rats at levels 1,080 to 14,400 times the maximum recommended human ophthalmic dose (MRHOD). There was no toxicity in rat offspring at exposures estimated to be 45 to 150 times that at MRHOD. Olopatadine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Animal Data

In a rabbit embryofetal study, rabbits treated orally at 400 mg/kg/day during organogenesis showed a decrease in live fetuses. This dose is 14,400 times the MRHOD, on a mg/m^2 basis.

An oral dose of 600 mg/kg/day olopatadine (10,800 times the MRHOD) was shown to be maternally toxic in rats, producing death and reduced maternal body weight gain. When administered to rats throughout organogenesis, olopatadine produced cleft palate at 60 mg/kg/day (1080 times the MRHOD) and decreased embryofetal viability and reduced fetal weight in rats at 600 mg/kg/day. When administered to rats during late gestation and throughout the lactation period, olopatadine produced decreased neonatal survival at 60 mg/kg/day and reduced body weight gain in offspring at 4 mg/kg/day. A dose of 2 mg/kg/day olopatadine produced no toxicity in rat offspring. An oral dose of 1 mg/kg olopatadine in rats resulted in a range of systemic plasma area under the curve (AUC) levels that were 45 to 150 times higher than the observed human exposure [9.7 ng·hr/mL] following administration of the recommended human ophthalmic dose.

Nursing Mothers

Olopatadine has been identified in the milk of nursing rats following oral administration. Oral administration of olopatadine doses at or above 4 mg/kg/day throughout the lactation period produced decreased body weight gain in rat offspring; a dose of 2 mg/kg/day olopatadine produced no toxicity. An oral dose of 1 mg/kg olopatadine in rats resulted in a range of systemic plasma area under the curve (AUC) levels that were 45 to 150 times higher than the observed human exposure [9.7 ng·hr/mL] following administration of the recommended human ophthalmic dose. It is not known whether topical ocular administration could result in sufficient systemic absorption to produce detectable quantities in the human breast milk. Nevertheless, caution should be exercised when PAZEO is administered to a nursing mother.

Pediatric Use

The safety and effectiveness of PAZEO have been established in pediatric patients two years of age and older. Use of PAZEO in these pediatric patients is supported by evidence from adequate and well-controlled studies of PAZEO in adults and an adequate and well controlled study evaluating the safety of PAZEO in pediatric and adult patients.

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenicity

Olopatadine administered orally was not carcinogenic in mice and rats in doses up to 500 mg/kg/day and 200 mg/kg/day, respectively. Based on a 35 µL drop size and a 60 kg person, these doses are approximately 4,500 and 3,600 times the MRHOD, on a mg/m^2 basis.

Mutagenesis

No mutagenic potential was observed when olopatadine was tested in an *in vitro* bacterial reverse mutation (Ames) test, an *in vitro* mammalian chromosome aberration assay or an *in vivo* mouse micronucleus test.

Impairment of fertility

Olopatadine administered at an oral dose of 400 mg/kg/day (approximately 7,200 times the MRHOD) produced toxicity in male and female rats, and resulted in a decrease in the fertility index and reduced implantation rate. No effects on reproductive function were observed at 50 mg/kg/day (approximately 900 times the MRHOD).

PATIENT COUNSELING INFORMATION

• Risk of Contamination: Advise patients to not touch dropper tip to eyelids or surrounding areas, as this may contaminate the dropper tip and ophthalmic solution.

• Concomitant Use of Contact Lenses: Advise patients not to wear contact lenses if their eyes are red. Advise patients that PAZEO should not be used to treat contact lens-related irritation. Advise patients to remove contact lenses prior to instillation of PAZEO. The preservative in PAZEO solution, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 5 minutes following administration of PAZEO.

Patents: 8,791,154

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VOL. 152 NO. 9 ■ SEPTEMBER 15, 2015

IN THE NEWS

A recent study, published online ahead of print in *JAMA Ophthalmology*, looked at the link between trace metals and **glaucoma prevalence** in 2,680 South Korean patients 19 years and older. They found that participants with glaucoma had significantly **lower blood manganese levels** vs. patients without glaucoma. They also discovered patients with glaucoma had **higher blood mercury levels** vs. those without the disease.

A new study, recently published in the *Journal of Alzheimer's Disease*, showed a significant **link between macular pigment levels in the eye and various measures of cognitive performance**. Researchers looked at two groups of patients—those free of retinal disease but with low macular pigment and those with early AMD—and found that, even after controlling for age, gender, diet and education levels, the correlations between macular pigment and cognitive function remained statistically significant.

SkiVision, recently acquired by Jobson, joins the *Review of Optometry* meetings division. Held President's Day weekend in Snowmass Village, CO, SkiVision provides up to 20 hours of COPE-approved optometric education in a fun, informal setting.The team of **Murray Fingeret, OD, Leo Semes, OD, and Andrew Archila, OD,** will remain the driving force behind the event.

Dr. Fingeret hits the slopes during the annual event.

Autoimmune Uveitis Starts in the Gut

Intestinal bacteria masquerade as retinal antigens, prompting immune cell activation.

By Rebecca Hepp, Senior Associate Editor

New research from the National Eye Institute (NEI) suggests the retina-specific immune cells that are causally involved in autoimmune uveitis have their beginnings in the gut.

"Understanding what spurs autoimmune uveitis is fundamental to the development of safer long-term therapies and possibly even strategies for preventing it," Reiko Horai, PhD, a staff scientist at NEI and a lead author of the study, says in an NEI press release.

Because the eye is an immune-privileged site, researchers have struggled to understand how the immune cells become activated while their antigens are separated behind blood-tissue barriers. The new study found the cells receive the activation signal in the gut, an action that precedes the clinical onset of the disease.

Once they discovered the location of the immune cell activation, the investigators speculated that bacteria in the gut produce a molecule similar to the retinal antigens, thus prompting the immune cells to activate and seek the proteins in the eye. To support this theory, they activated retina-specific immune cells by exposing them to bacterial proteins extracted from mouse intestines. When they injected the activated cells into normal mice, they developed uveitis.

While the researchers note the findings have no impact on patient care yet, they hope it will help scientists one day identify the bacteria specifically involved in promoting autoimmune uveitis, possibly leading to targeted therapy options.

Horai R, Zárate-Bladés CR, Dillenburg-Pilla P, et al. Microbiota-Dependent Activation of an Autoreactive T Cell Receptor Provokes Autoimmunity in an Immunologically Privileged Site. Immunity. 2015;43:343-53.

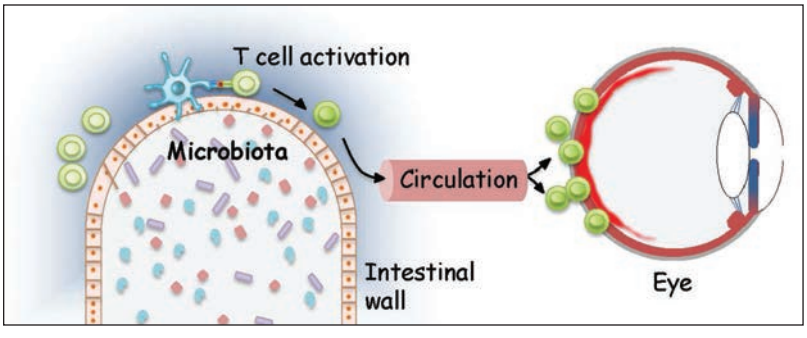

Image: National Eye Institute, part of NIH

Immune cells activated in the gut migrate to the eye, where they cause the inflammatory response associated with autoimmune uveitis.

AOA Urges FDA to Investigate Online Eye Exam Provider

The AOA recently sent a letter to the FDA requesting the investigation of alleged misleading and inaccurate claims made by the online eye exam administrator Opternative that its technology, which offers $40 eye exams by computer or smartphone, is FDA registered.

According to the AOA, the Chicago-based company launched its online platform in July and issued a subsequent press release making these false claims, which appear to convey that the FDA has in some way performed an assessment of this technology and may view it favorably. Additionally, Opternative's claims appear to indicate functionality beyond Class I (general controls), due to the intention to provide primary diagnosis or treatment decisions and perform patient-specific analysis. Such capabilities should qualify the mobile app as a medical device, which should not be on the market until FDA officials complete all essential oversight, the AOA's letter states.

The AOA says it embraces cutting-edge technology when it enhances quality patient care. "However, whenever and wherever technology is abused in a manner that undermines critical public and patient safety, or provides a false sense of security when there is undetected, unknown and early onset disease, the AOA remains committed to acting to ensure the public receives the care they expect and deserve," the AOA further states in its letter.

AOA President Steven A. Loomis, OD, says the AOA is firm in its position that there is no substitute for an in-person, comprehensive eye exam performed by a doctor of optometry, and a comprehensive eye exam is the only consistent and trustworthy method to detect not just refractive errors, but also the full range of eye health and vision conditions, many of which have no obvious signs or symptoms.

"We have a serious concern about separating the refraction from a comprehensive eye examination," Dr. Loomis says. "A refraction does not simply yield an eyeglass or contact lens prescription. A refraction is a diagnostic test that yields information about potential health concerns like diabetes, cataracts and retinal issues, to name only a few. These online 'exams' are completely insufficient to meet those needs. Of added concern is that patients are likely quite unaware of the deficiency of the product they have just purchased. That is why the AOA and its state affiliate associations are actively combating misleading claims made by these so-called 'online eye exams.'"

The AOA has already taken action against online eye exams, including the passage of Resolution 1987, "Potential Health Risks of Emerging Technologies in Eye Care," in the House of Delegates at Optometry's Meeting in June in Seattle, Wash. In addition, the AOA recently sent a letter to the president of the American Academy of Ophthalmology (AAO) to make a stand. "I've called on the AAO to rebuke the dangerously misleading product claims made by 'online eye exams' and to work alongside the AOA to safeguard public health and healthy vision," Dr. Loomis says.

First Guide Dog for Runners

Lt. Richard Hunter of San Francisco, who was diagnosed with retinitis pigmentosa while serving in the United States Marine Corps, is the first athlete to partner with a professionally trained running guide dog.

Lt. Hunter's partner Klinger, a two-year-old German Shepherd, is the first dog certified under Guiding Eyes for the Blind's Running Guides pilot program, which was established to support the underserved community of runners who are visually impaired, the Guiding Eyes website states.

Klinger and Lt. Hunter are currently training for the California International Marathon.

For more information, visit www.guidingeyes.org.

Photo: Guiding Eyes for the Blind.

Restasis®
(Cyclosporine Ophthalmic Emulsion) 0.05%

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

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

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

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

Indication and Usage

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

Important Safety Information

Contraindications

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

Warnings and Precautions

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

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

Adverse Reactions

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

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

Reference: 1. RESTASIS® Prescribing Information.

ALLERGAN

APC20AF14 141005

RESTASIS® (Cyclosporine Ophthalmic Emulsion) 0.05%

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

INDICATION AND USAGE

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

CONTRAINDICATIONS

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

WARNINGS AND PRECAUTIONS

Potential for Eye Injury and Contamination

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

Use with Contact Lenses

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

ADVERSE REACTIONS

Clinical Trials Experience

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

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

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

Post-marketing Experience

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects: Pregnancy Category C

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

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

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

Nursing Mothers

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

Pediatric Use

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

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility

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

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

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

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

PATIENT COUNSELING INFORMATION

Handling the Container

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

Use with Contact Lenses

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

Administration

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

Rx Only

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The Path to Vision Loss in AMD

Researchers at the Washington University School of Medicine in St. Louis have discovered a pathway that leads to the formation of atypical blood vessels, causing vision loss in patients with age-related macular degeneration.

The study, published online in the journal *Nature Communications*, looked at both engineered mice and eye tissue from patients treated in the 1980s and 1990s, when the removal of abnormal blood vessels was routinely performed on patients with wet AMD. The investigators found that, in both mice and human tissue, a signaling pathway involving the protein STAT3 was activating and altering immune cells, called M2 macrophages, known to contribute to the development of damaging blood vessels beneath the retina.

The researchers are hopeful the discovery could one day lead to targeted treatments, as some compounds can disrupt the actions of STAT3 in mice and keep the pathway from activating blood vessel growth. Those same compounds may also alter the course of macular degeneration in patients diagnosed with the condition, according to a Washington University press release.

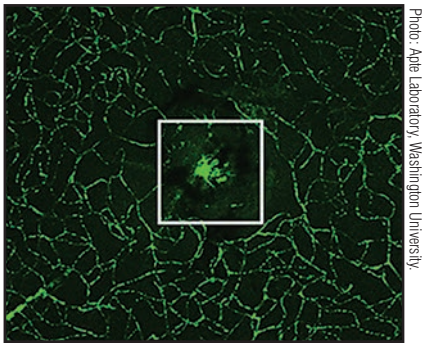

Photo: Apte Laboratory, Washington University.

Normal blood vessels surround a clump of new, abnormal vessels that has formed beneath the center of the retina.

"Now that we have a better idea of how these macrophages are activated at the molecular level, we may be able to use those drugs to halt or reverse the disease process," says principal investigator and retina specialist Rajendra S. Apte, MD, PhD.

"This study mirrors a new trend in research that looks at inflammation and the immune system as mediators of age-related macular degeneration, specifically wet, or neovascular, AMD and choroidal neovascular membrane (CNVM) formation," says Steven Ferrucci, OD, of the US Department of Veteran Affairs in North Hills, Calif., and a professor at the Southern California College of Optometry at Marshall B. Ketchum University in Los Angeles. "If the mediators of CNVM formation can be better understood, more targeted therapies may prove useful. Anti-VEGF therapy has been a great step in the right direction for AMD treatment, but monthly injection simply is not a sustainable treatment model. Newer therapies with different modes of action, synergistic medications, longer acting medication, alternative routes of administration, all need to be and are being investigated."

Nakamura R, Sene A, Santeford A, et al. IL10-driven STAT3 signalling in senescent macrophages promotes pathological eye angiogenesis. Nature Communications. 2015;6:7847.

Amblyopia's Effect on the Brain

A new study sheds light on how amblyopia affects the brain's visual pathways. Researchers from the University of Wisconsin-Madison used diffusion-weighted imaging to map three pathways known to carry visual information between the eyes and the brain. They discovered water diffusing more easily down the brain's visual pathways in patients with amblyopia.

The investigators speculate the conductive sheath of myelin around neurons becomes thinner, providing less insulation. With less myelin in the way, the water diffuses more easily.

This study may improve treatments for amblyopia and other disorders in which patients have trouble judging distance and location of objects, investigators hope.

"This diffusion-weighted imaging approach will help us understand whether, and how much, brain training treatments work," says Bas Rokers, a University of Wisconsin-Madison psychology professor, in a press release. "You can put patients in the scanner and see if your treatment actually has an effect." ■

Allen B, Spiegel DP, Thompson B, et al. Altered white matter in early visual pathways of humans with amblyopia. Vision Research. 2015;114:48.

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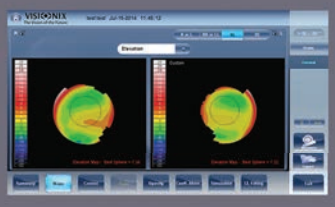

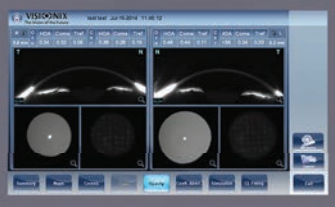

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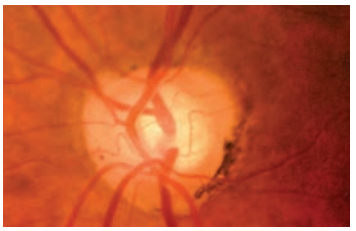
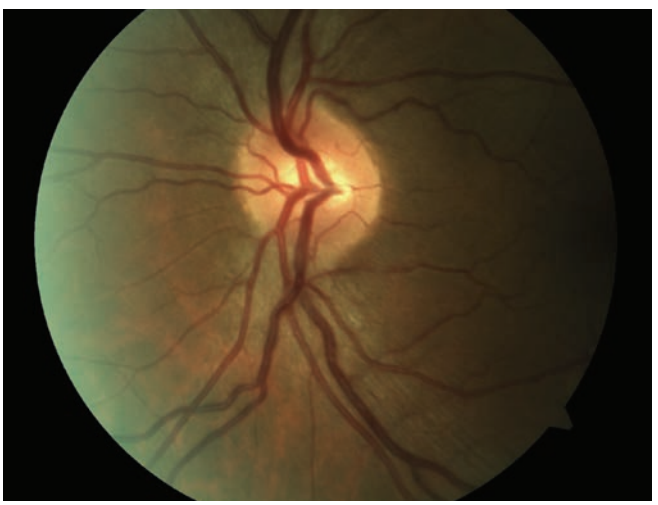
Contents

Review of Optometry September 2015

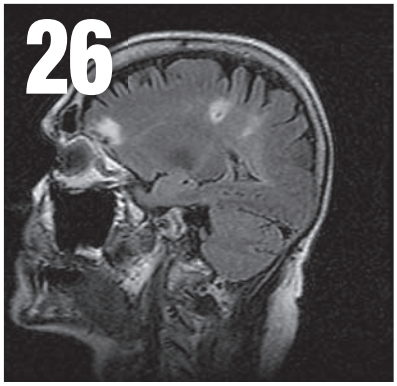
38th Annual TECHNOLOGY REPORT

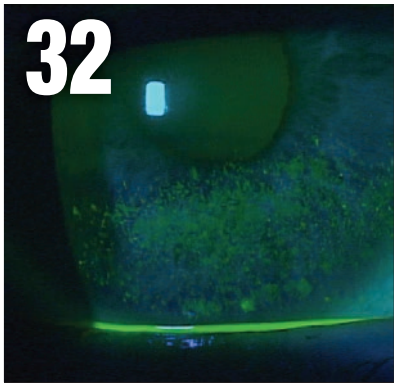


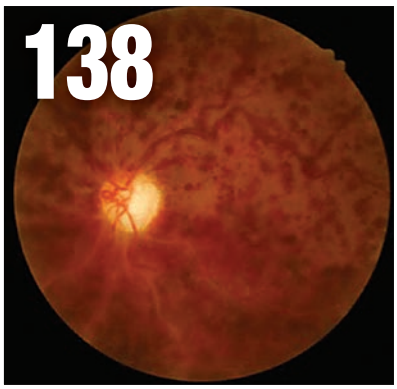


Departments

Review of Optometry September 2015







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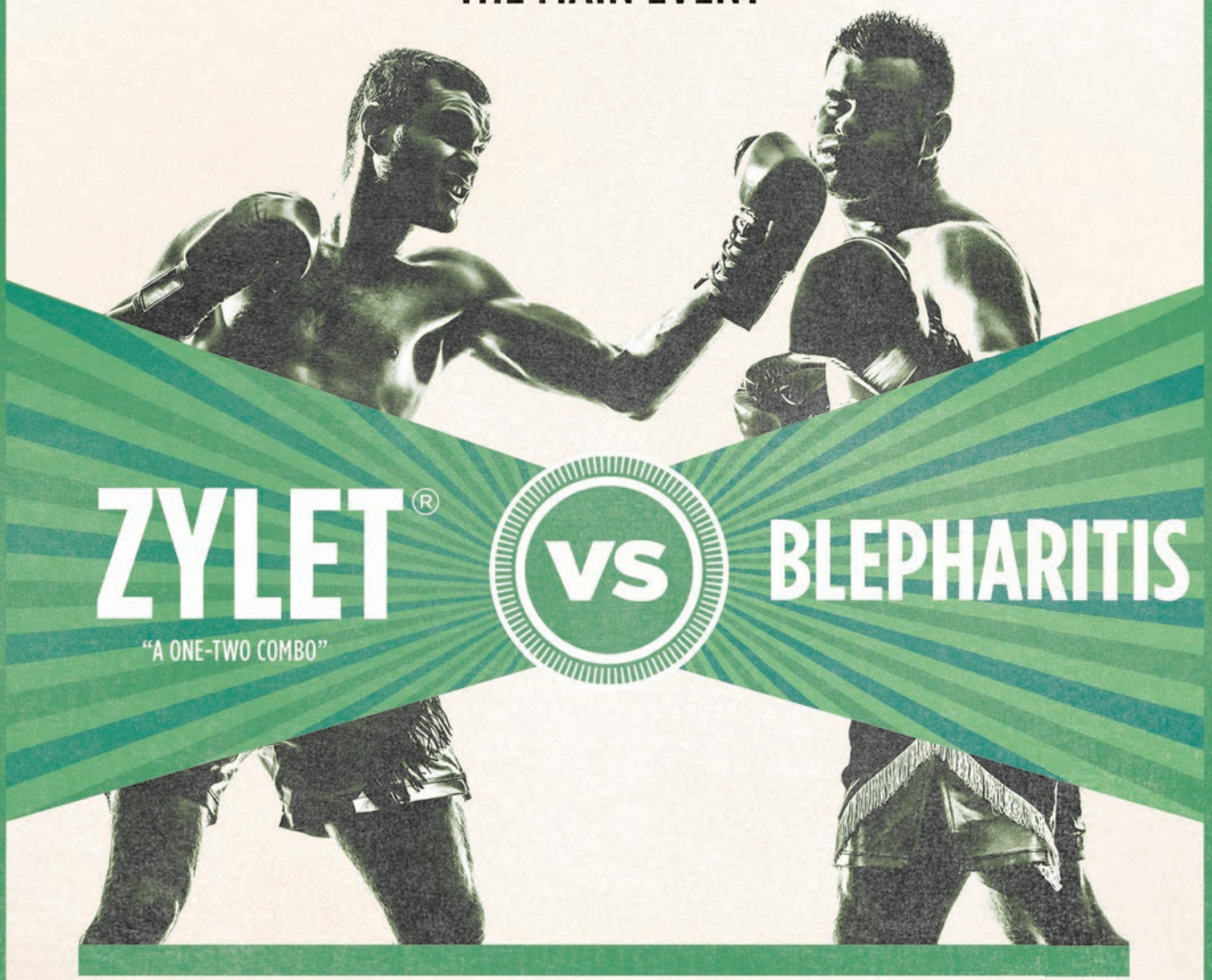

HELP PUT RELIEF IN YOUR CORNER

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.

Please see additional Indications and Usage information on adjacent page, including list of indicated organisms.

INDICATIONS AND USAGE (continued)

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 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. 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, burning and stinging upon instillation.

Please see Brief Summary of Prescribing Information on the following page.

With a one-two combo in the treatment of blepharitis and other steroid-responsive ocular conditions with the risk of bacterial infection, PRESCRIBE ZYLET® TODAY.

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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: Pregnancy Category C. Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb 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.

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BAUSCH & LOMB INCORPORATED

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Letters to the Editor

Delaying the diagnosis and treatment of a patient with a high ESR and GCA can result in rapid permanent vision loss of monocular or binocular vision.

A Prism of a Different Color

Editor's note: In June's "Focus on Refraction" column, authors Marc B. Taub, OD, MS, and Paul Harris, OD, reviewed the case of an 18-year-old experiencing blurry vision and intermittent diplopia. They recommended a reevaluation of the prescription and vision therapy. You can read this case at www.reviewofoptometry.com/content/c/55105/.

One question that I have about this very thorough evaluation of this patient: There is no mention of any keratometry readings. Did you take keratometry readings on this patient? Is the little amount of cylinder present equal to "stress cylinder" from accommodative dysfunction that also might be contributing to his visual problems?

As you are probably aware, at 18 years old this patient is susceptible to induced stress cylinder that should not be corrected if it does not correspond to his corneal readings.

—Steven Mordukowitz, OD, FAAO,
Bronx, NY

Drs. Taub and Harris respond:
Thank you so much for taking the time to write. We actually did not obtain the keratometry readings in this case, but they certainly would have been helpful for the precise reason you pointed out. In this case, we did subjectively assess the vision with and without the cylinder since and the patient reported clearer, more comfortable vision.

Since he was older and able to communicate the improvement effectively, we made the conscious decision to leave in the cylinder we found. As we reevaluate the patient once therapy has been completed, we do expect the refraction to change, and most likely, the cylinder will not be included in the next prescription.

Sixth Nerve Palsy Headaches

Editor's note: In July's "Neuro Clinic" column, authors Michael Trottini, OD, and Michael DelGiodice, OD, reviewed the case of a 92-year-old male experiencing sixth nerve palsy. They recommended a re-evaluation of the prescription and vision therapy. You can read this case at www.reviewofoptometry.com/content/c/55105/.

As both a professor emeritus and a clinician, I was troubled by the article "Sixth Nerve Palsy Prompts a Surprising Diagnosis." First, it should not have been a surprise! (I presume by the notation that measurements were taken at distance, since 2Δ ET by convention in primary gaze means a 2Δ alternating esotropia at distance).

Second, it is unusual for the patient to have a constant 2Δ alternating esotropia at distance. A left sixth nerve palsy usually presents with a 20Δ left esotropia (LET) (if the measurements were at near, then it should have been noted as LET. The prime denotes near testing). In my experience, sudden onset sixth nerve palsies almost always present as 20Δ esotrope at distance (plus or minus the original distance phoria) with a small esophoria at near (variation in the angle is dependent upon the original phoria). If the initial deviation is much less than 20Δ, then the patient must be watched since the disease might still be progressing. As a result, an incomplete sixth nerve palsy should be seen within a week and re-measured.

Third, the authors provided no relief for a patient presenting with diplopia. Sixth nerve palsies are best handled by applying a 20Δ Fresnel prism on the superior portion of the affected eye's lens (i.e., cut the prism and apply). This will eliminate the diplopia and can be easily removed upon resolution. Patching, which is often taught as a treatment, is a horrible way to eliminate diplopia, since it makes the patient nonfunctional (leading to a loss of depth perception and binocular field of view).

Leaving the patient diplopic is unacceptable. The last method, Botox, is unpredictable, but acceptable if the patient does not want a patch or Fresnel prism.

Fourth, and most important, these patients with a presumed isolated sixth nerve palsy should be immediately worked up. The following should be performed: visual fields, physical examination by PCP, complete blood count, blood sugar, HbA1C, ESR, C-reactive protein, Lyme titer ANA (depending on where you live), VDRL and rheumatoid factor test.

Isolated sixth nerve palsy rarely has a positive MRI, so that should be done if it does not resolve in six months. The problem is, if you don't order an MRI, the PCP probably will, right or wrong, which puts the referring optometrist in the position to justify to the patient why you didn't order the test. It is best to call the primary care physician and discuss whether or not to order an MRI. Delaying the diagnosis and treatment of a patient with a high ESR and GCA can result in rapid permanent loss of monocular or binocular

vision. Happily, your patient did not develop ischemic optic neuropathy. The tests needed to be ordered right away, not one month later!

In summary, rapid workup of these patients, including ESR, is essential to prevent visual loss. Diplopia in the elderly may be a result of GCA and requires an immediate evaluation. Application of Fresnel prism can keep these patients comfortable.

—Jeffrey Cooper, MS, OD, FAAO
New York, NY

Drs. Trottini and DelGiodice respond:
The diagnosis was a surprise, as GCA causes less than 4% of sixth nerve palsies.[1] We are aware of the connection, but this case was atypical because our patient didn't develop GCA symptoms until almost three months later (typically these patients have GCA symptoms upon presentation). Not to mention, our editors choose catchy titles to bring attention to the article.

We have seen a significant number of sixth nerve palsies both less than and greater than 20Δ, so we don't agree that sixth nerve palsies almost always present with a 20Δ esotropia. One study described clinical features of sixth nerve palsies, stating that out of 63 patients, the deviation ranged from 13.3pd to 34.3pd.[2] We also question the comment that "sixth nerve palsies are best handled by applying a 20Δ Fresnel prism" to alleviate diplopia. To give all sixth nerve palsies a 20Δ Fresnel is inappropriate; the prism should be determined on a case-by-case basis. To use our patient as an example, who only had a 2Δ esotropia in primary gaze, a 20Δ Fresnel would have significantly overcompensated and he still would have been diplopic.

Concerning the reccommendation that "patients with a presumed isolated sixth nerve palsy should be immediately worked up," this argument has been debated and well documented among optometrists, ophthalmologists and neuro-ophthalmologists; however, no general consensus or finite guidelines exist regarding initial testing. Depending on which study you read, the incidence of non-microvascular isolated nerve palsies ranges from 1% to 16%.[1,3,4] There was a great point-counterpoint article between Nicholas Volpe, MD, and Andrew Lee, MD, in a recent issue of the *Journal of Neuro-Ophthalmology* in which they debated initial neuro-imaging for isolated third, fourth and sixth nerve palsies.[5] Even two elite members of the neuro-ophthalmologic profession were not able to come to a consensus. So, to make the statement that patients with a presumed isolated sixth nerve palsy should be immediately worked up is more of an opinion rather than a fact.

It is our belief that the decision to run an MRI in these cases should be at the discretion of the eye care provider. If concerned about the PCP ordering it unnecessarily, share with that doctor your recommendation of initial observation. As the commenter points out, MRI shows a low yield and thus only recommends imaging after no resolution in six months; it cannot provide immediate laboratory studies with an even lower yield such as the RF, ANA and VDRL. A few case reports of sixth nerve palsies as the initial manifestation of lupus exist; however, the patients were 35- and 48-year-old females, which fits the epidemiologic profile of lupus far greater than that of a 92-year-old male.[6,7] Syphilis in a 92-year-old is extremely unlikely; also, we aren't aware of any case reports or connections between rheumatoid arthritis and sixth nerve palsies.

As for the ESR and CRP, the general recommendation is to obtain these tests in older individuals with suspected GCA. Otherwise, again, the yield is quite low. We did comment in our article that, because the incidence of GCA increases exponentially after age 80, and because of our experience managing this patient, we felt immediate testing of the ESR and CRP in this subset of patients may be helpful regardless of symptoms. Even with this recommendation, it is unknown whether or not our patient's ESR and CRP would have been elevated initially until the disease progressed when he started developing constitutional symptoms.

Lastly, we did communicate with this patient's internist to re-evaluate and continue managing his vascular issues. We also prescribed a temporary Fresnel prism to alleviate our patient's diplopia. Unfortunately, due to space constraints with a brief column we were not able to elaborate on every detail of the case. ■

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99 Problems

Only one contact lens wearer out of 100 is compliant. The rest abuse the privilege. **By Jack Persico, Editor-in-Chief**

It's probably not an exaggeration to call the state of contact lens patient compliance a disgrace. Most practitioners sense this anecdotally, as they're continually frustrated by patients who simply won't bother to care for lenses properly. Now, a report from the Centers for Disease Control all but confirms it.

A recent survey of 1,000 contact lens wearers by the CDC found that 99% reported at least one contact lens hygiene behavior associated with an increased risk for eye infection or inflammation.

Risky behaviors reported by patients included swimming (61%) or showering (85%) while wearing lenses, rinsing (36%) or storing (17%) them in tap water, sleeping (50%) or napping (87%) in daily wear lenses, extending the replacement interval for lenses (50%) or cases (82%), topping off solution in lens cases (55%) and failing to wash hands before inserting (4%) or removing (17%) lenses.

Truth be told, the survey design makes it almost impossible not to give patients a failing grade. If a respondent reported anything other than "never" for each of the above behaviors, they were counted as having "ever" engaged in risky behavior. In other words, there was no consideration for the frequency of compliance failure. There also was no weighting of the relative risks of behaviors; all were equally bad.

Still, there's no denying that these actions have consequences. "Nearly one third of contact lens wearers reported having experienced a previous contact lens-related red or painful eye requiring a doctor's visit," the report said.

Curiously, what's perceived as the safest modality of lens wear—daily disposables—didn't improve most of the scores on the risky behaviors. These patients were nearly as likely as other subgroups, or the contact lens population as a whole, to engage in most of the bad behaviors. And, in fact, they were actually *more* likely to rinse or store lenses in tap water and to top off solution when they do use them—which stands to reason, since part of the selling point of daily disposables is the ability to avoid solution costs. Maybe these patients take the convenience of daily disposables too far and could stand to be reminded that disposability doesn't absolve them of responsibility.

Extending the lens replacement interval beyond what's intended (and prescribed) seems especially hard for patients to give up. Even with 365 pairs of lenses at their disposal (no pun intended), these patients only shaved off 11% from the number likely to abuse the recommended replacement cycle, from 50% for all CL wearers to 39% for patients wearing daily disposable lenses.

Expect the Expected

Little of the above is surprising at this point. So, when educating your contact lens patients about lens care, expect the expected: They'll cheat. Better to recognize this than try to hold them to an unrealistic ideal. Remind them that *convenient* doesn't mean *carefree,* and give them a good understanding of the consequences of their actions—and 'inactions.' ■

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Howdy, Y'all!

I've moved on to bigger things—it's time to see what the Lone Star State has to offer an OD like me. **By Montgomery Vickers, OD**

There's this bumper sticker that says, "I wasn't born in Texas, but I got here as fast as I could!" After 35 years of wonderful small town solo optometry, I sold the practice and moved to Texas. Why start over now, and in Texas? There are many reasons:

1. I'm only licensed in West Virginia and Texas.
2. I can join the secessionist movement and work toward my lifelong goal of being the Emperor of my own country.
3. My West Virginia concealed carry permit is accepted here. By the way, I don't own a gun, just a permit, which I am not at all hesitant to show any villain who crosses my path.
4. Who wouldn't trade lush mountains and sparkling rivers for dust and tarantulas?
5. I forgot to read the fine print of my practice sale contracts. Man, they sure had a tough non-compete clause.
6. I hear they have two optometry schools around here, so now I can end up the old guy in the clinic as planned.
7. They say everything in Texas is bigger, and I have always wanted a giant slit lamp.
8. Six grandchildren in the state.

But, I didn't realize that getting to Texas would be such an adventure. Texas is a long ways away—from anywhere. In my household, there is a standing joke that when we took the family eight hours away to the beach, we spent the first night in Beckley, W.Va., one hour away, because we were sick of driving.

After two solid weeks of packing, I was whipped and ready for a nap, but I couldn't convince my wife, Renee, to let me chain my car to hers for a nice tow west.

So, with eyes bulging and a resting heart rate at 261bpm, we headed west. We stopped for an all-nighter in Nashville, but no one would sign me as the next Luke Bryan.

At the crack of 10am, we were back in gear and we made our way to lunch with Elvis in Memphis. You may recall that Elvis was once my patient. Well, he said he was an Elvis impersonator, and this was after Elvis' supposed passing, but I thought, 'what a perfect cover for Elvis … playing Elvis!'

I actually loved Graceland. Don't knock it until you try it.

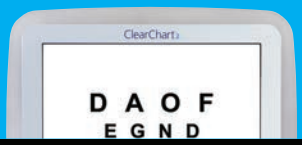
We were barely in the car to leave when the worst lightning storm I have ever seen hit. Me? I would have found a Holiday Inn. Renee? Well, she makes the Texas Rangers look like a Richard Simmons video. So, we plowed on at a blazing 10mph. It took us an hour to drive through it, and then the road was so straight I could tie down the wheel and catch a few winks.

In Arkansas, we hit another monster storm. I knew we were in trouble when I saw my old office knock a barn down east of Little Rock. But it's OK; we never had a sign, so nobody could find us anyway.

After much travail and many blessings, I safely made it to Dallas with the love of my life, my collection of nine guitars. Hmm? Who? Oh, sure, Renee made it too.

Now we are ready for our next optometric adventure. For the first time since working for Dr. Bodie in 1980, I am going to be an employee. *Chairside* will gallop along with an all-new perspective. I have already warned my new boss.

I am also excited to continue my Texas food quest. First stop? I think I saw a Taco Bell around the corner. And where does a tough ol' cowboy like me get a pedicure, anyway? ■



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Colored Shadows: An Emergency

Positive visual symptoms of amaurosis fugax are possible. Here's what to look for and how to diagnose. **By Michael Trottini, OD, and Michael DelGiodice, OD**

A 69-year-old white female presented as an emergency exam with a chief complaint of transient visual disturbance. She described these as "blue-colored shadows" that transiently and incompletely blocked the vision of the right eye. She experienced three separate episodes. Each lasted approximately five minutes and was isolated, as she denied symptoms of headache, dizziness, motor weakness, syncope or constitutional symptoms of temporal arteritis.

Upon questioning, she described prior episodes occurring over the past few years, which were attributed to ocular migraine. The remaining ocular history indicated high myopia in each eye. Her medical history was remarkable for essential hypertension and she was taking Bystolic (nebivolo, Forest Laboratories), Norvasc (amlodipine besylate, Pfizer) and triamterene-hydrochlorothiazide. Her family and social histories were unremarkable.

Her best-corrected visual acuity was 20/40 in each eye. Ocular motility was full with no limitation. Pupils were equal, round and reactive to light without afferent defect. Ocular alignment was normal with an orthophoric position in primary and lateral gazes. Additionally, she noted 10/10 color plates in each eye. IOP measured 18mm Hg OD and 19mm Hg OS. The anterior segment exam revealed mild nuclear sclerosis in each eye. Fundus exam showed normal vitreous and retinal structures. The optic nerves showed a cup-to-disc ratio of 0.20 with circumferential chorioretinal atrophy. The neuroretinal rim was pink, healthy, distinct and flat. Digital compression of the globe was negative for retinal-arterial collapse. Subsequently, we ordered a 30-2 threshold visual field test, which showed no localizing features of ocular or neurologic dysfunction.

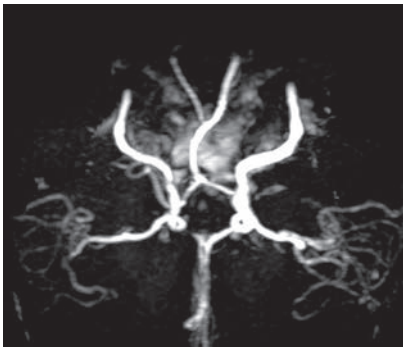

Fig. 1. MRA of the RICA showing stenosis.

Diagnosis

In light of the lack of clinical findings, we diagnosed her with isolated transient monocular vision loss (TMVL). The differential diagnoses included amaurosis fugax, impending retinal vascular occlusion and ischemic optic neuropathy, occipital seizure disorder, retinal migraine and acephalgic migraine.

Because the visual field was unremarkable, symptoms were isolated and digital compression did not reveal arterial collapse, our leading diagnosis was amaurosis fugax. We also considered both forms of migraine as a diagnosis of exclusion. Since she denied symptoms suggestive of cerebral TIA, we alerted her primary care physician and ordered an urgent carotid duplex along with complete blood count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), complete metabolic panel (CMP) and lipid panel. The carotid duplex showed severe stenosis of the right internal carotid artery (ICA) and mild stenosis of the left.

All serology was normal with the exception of moderately elevated lipids. Consequently, we scheduled her for urgent magnetic resonance angiography (MRA) of the neck and brain to detail the location of the stenosis within the carotid artery. We also ordered magnetic resonance imaging (MRI) to discount associated cerebral infarction. The MRA showed severe stenosis within the right ICA (*Figure 1*), along with periventricular white matter (WM) changes and two restricted diffusion foci located within the right posterior parietal-occipital junction, consistent with a subacute infarct.

Causes of WM lesions most commonly include: normal senescent changes, hypertension, focal cerebrovascular accidents, demyelination, migraine, vitamin B6 (pyridoxine) deficiency and infectious or inflammatory-related vasculitis.

Ultimately, we diagnosed her with amaurosis fugax and right hemispheric subacute CVA secondary to severe right ICA stenosis.

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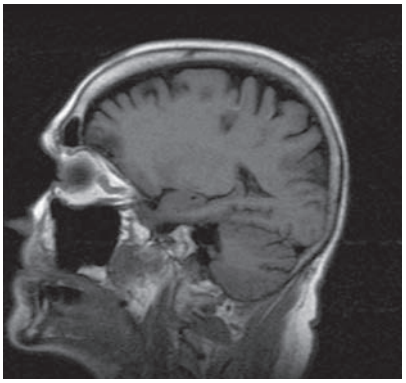

Two dark lesions within the parietal-occipital region that represent edema from ischemia.

Subsequently, she started 81mg of aspirin as prophylaxis and was referred to the vascular surgeon for medical and surgical intervention. The patient immediately started Plavix (clopidogrel bisulfate, Bristol-Myers Squibb) and underwent both a neurologic and cardiovascular work up prior to endarterectomy of the right ICA. She tolerated the procedure well and has been asymptomatic since.

Discussion

Amaurosis fugax is a term used to describe transient monocular vision loss (TMVL) secondary to vascular insufficiency due to atherosclerosis of the ipsilateral internal carotid artery. In 1990, the Amaurosis Fugax Study Group defined five causes of transient monocular blindness: embolic, hemodynamic, ocular, neurologic and idiopathic.[1]

While most patients with symptoms of amaurosis fugax describe negative visual phenomena (i.e., blurring, fogging, graying or dimming of vision), positive visual symptoms such as colored spots, flashes of light and fortification spectra can also occur, often presenting a challenge to the clinician.[2] Differentiating symptoms of benign migraine aura from more ominous causes such as vascular insufficiency or seizure disorders is crucial to timely diagnosis and treatment.

Migraine aura without headache (acephalgic migraine) is a subset of migrainous phenomena in the adult population. These include symptoms of scintillation, transient hemianopia, central scotomata, classic amaurosis fugax, diplopia, altitudinal field loss, tunnel vision and alterations in color perception.[3]

Acephalgic migraines typically develop slowly, over 10 to 20 minutes, and rarely occur longer than one hour. While migraine aura is a bilateral phenomenon, many describe the event as unilateral due to obstruction of their nasal field, confounding the diagnosis. The neuronal spread of electrical activity during a migraine coincides with one system being affected at a time in a continuous fashion within the distribution of the cerebral cortex.

Clinicians must also consider the more benign entity of retinal migraine, which often occurs unilaterally and may present in a similar fashion to amaurosis fugax. Retinal migraine, like acephalgic migraine, is a diagnosis of exclusion and should be considered only after discounting other pathologic causes.

Alternatively, seizures typically progress rapidly over the course of seconds, are repetitive and will occur in a single functional neurologic domain involving multiple symptoms simultaneously. Patients experiencing seizure activity are more apt to develop transient syncope and, in some instances, prolonged loss of consciousness.

This case represents an atypical presentation of carotid artery stenosis. It is important to have a low threshold for considering carotid artery stenosis in elderly patients complaining of symptoms masked as migraine aura.

TMVL

Research suggests the annual incidence of TMVL between the ages of 25 and 84 is 13.7/100,000 for men and 9.4/100,000 for women, with the greatest incidence occurring in the seventh decade of life.[4] Subsequently, the annual incidence of stroke following TMVL ranges from 2.0% to 2.8%.[4]

The most common causes of TMVL are ischemia and hypoperfusion, both of which may occur as a result of carotid artery stenosis, carotid or cardiac embolic events or giant cell arteritis. The lack of perfusion pressure causes a brief focal dysfunction followed by a return to normal function. Investigators found that ipsilateral carotid stenosis was responsible for 79% of TMVL symptoms and 57% for less specific complaints such as visual blurring or binocular scintillations.[5]

With carotid disease, chronic atheromatous plaque formation can result in thrombus formation, increased stenosis, ulceration and occlusion. Clinically, the problem is figuring out who will develop a subsequent cerebral infarct, permanent vision loss or both.

According to an early study, approximately 12% of patients with untreated ocular TIA will suffer a CVA within a year of the onset of symptoms and up to 35% within five years.[6] TIAs from carotid stenosis are associated with computed tomography (CT) confirmed ischemic CVA in 15% to 30% of patients. If a patient exhibits symptoms of both ocular and hemispheric TIA, up to 4% are at risk for permanent blindness.[6]

Diagnostic Testing

Given the risk of developing subsequent CVA, we recommend keeping a low threshold for diagnosis of amaurosis fugax when presented

For allergic conjunctivitis[1]

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INDICATION AND USAGE

BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% is a histamine H_1 receptor antagonist indicated for the treatment of itching associated with signs and symptoms of allergic conjunctivitis.

IMPORTANT RISK INFORMATION

BEPREVE® is contraindicated in patients with a history of hypersensitivity reactions to bepotastine or any of the other ingredients. BEPREVE® is for topical ophthalmic use only. To minimize risk of contamination, do not touch the dropper tip to any surface. Keep the bottle closed when not in use. BEPREVE® should not be used to treat contact lens–related irritation. Remove contact lenses prior to instillation of BEPREVE®.

The most common adverse reaction occurring in approximately 25% of patients was a mild taste following instillation. Other adverse reactions occurring in 2%-5% of patients were eye irritation, headache, and nasopharyngitis.

Please see the accompanying prescribing information for BEPREVE® on the following page.

Reference: 1. BEPREVE [package insert]. Tampa, FL: Bausch + Lomb, Inc; 2012.

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For product-related questions and concerns, call **1-800-323-0000** or visit **www.bepreve.com**.

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BEPREVE®
(bepotastine besilate ophthalmic solution) 1.5%

BEPREVE® (bepotastine besilate ophthalmic solution) 1.5%

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% safely and effectively. See full prescribing information for BEPREVE®.

BEPREVE® (bepotastine besilate ophthalmic solution) 1.5%
Initial U.S. Approval: 2009

----------RECENT MAJOR CHANGES----------
Contraindications (4) 06/2012

----------INDICATIONS AND USAGE----------
BEPREVE® is a histamine H1 receptor antagonist indicated for the treatment of itching associated with allergic conjunctivitis. (1)

----------DOSAGE AND ADMINISTRATION----------
Instill one drop into the affected eye(s) twice a day (BID). (2)

----------DOSAGE FORMS AND STRENGTHS--------
Solution containing bepotastine besilate, 1.5%. (3)

----------CONTRAINDICATIONS----------
Hypersensitivity to any component of this product. (4)

----------WARNINGS AND PRECAUTIONS----------
- To minimize the risk of contamination, do not touch dropper tip to any surface. Keep bottle tightly closed when not in use. (5.1)
- BEPREVE should not be used to treat contact lens-related irritation. (5.2)
- Remove contact lenses prior to instillation of BEPREVE. (5.2)

----------ADVERSE REACTIONS----------
The most common adverse reaction occurring in approximately 25% of patients was a mild taste following instillation. Other adverse reactions which occurred in 2-5% of subjects were eye irritation, headache, and nasopharyngitis. (6)

To report SUSPECTED ADVERSE REACTIONS, contact Bausch & Lomb Incorporated. at 1-800-323-0000, or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.
See 17 for PATIENT COUNSELING INFORMATION

Revised: 10/2012

FULL PRESCRIBING INFORMATION: CONTENTS*

*Sections or subsections omitted from the full prescribing information are not listed

FULL PRESCRIBING INFORMATION

1 INDICATIONS AND USAGE
BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% is a histamine H_1 receptor antagonist indicated for the treatment of itching associated with signs and symptoms of allergic conjunctivitis.

2 DOSAGE AND ADMINISTRATION
Instill one drop of BEPREVE into the affected eye(s) twice a day (BID).

3 DOSAGE FORMS AND STRENGTHS
Topical ophthalmic solution containing bepotastine besilate 1.5%.

4 CONTRAINDICATIONS
Bepreve is contraindicated in patients with a history of hypersensitivity reactions to bepotastine or any of the other ingredients *[see Adverse Reactions (6.2)]*.

5 WARNINGS AND PRECAUTIONS
5.1 Contamination of Tip and Solution
To minimize contaminating the dropper tip and solution, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle. Keep bottle tightly closed when not in use.

5.2 Contact Lens Use
Patients should be advised not to wear a contact lens if their eye is red. BEPREVE should not be used to treat contact lens-related irritation.

BEPREVE should not be instilled while wearing contact lenses. Remove contact lenses prior to instillation of BEPREVE. The preservative in BEPREVE, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted after 10 minutes following administration of BEPREVE.

5.3 Topical Ophthalmic Use Only
BEPREVE is for topical ophthalmic use only.

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

The most common reported adverse reaction occurring in approximately 25% of subjects was a mild taste following instillation. Other adverse reactions occurring in 2-5% of subjects were eye irritation, headache, and nasopharyngitis.

6.2 Post Marketing Experience
Hypersensitivity reactions have been reported rarely during the post-marketing use of BEPREVE. Because these reactions are reported voluntarily from a population of unknown size, it is not always possible to reliably estimate their frequency or establish a casual relationship to drug exposure. The hypersensitivity reactions include itching, body rash, and swelling of lips, tongue and/or throat.

8 USE IN SPECIFIC POPULATIONS
8.1 Pregnancy
Pregnancy Category C: Teratogenicity studies have been performed in animals. Bepotastine besilate was not found to be teratogenic in rats during organogenesis and fetal development at oral doses up to 200 mg/kg/day (representing a systemic concentration approximately 3,300 times that anticipated for topical ocular use in humans), but did show some potential for causing skeletal abnormalities at 1,000 mg/kg/day. There were no teratogenic effects seen in rabbits at oral doses up to 500 mg/kg/day given during organogenesis and fetal development (>13,000 times the dose in humans on a mg/kg basis). Evidence of infertility was seen in rats given oral bepotastine besilate 1,000 mg/kg/day; however, no evidence of infertility was observed in rats given 200 mg/kg/day (approximately 3,300 times the topical ocular use in humans). The concentration of radio-labeled bepotastine besilate was similar in fetal liver and maternal blood plasma following a single 3 mg/kg oral dose. The concentration in other fetal tissues was one-third to one-tenth the concentration in maternal blood plasma.

An increase in stillborns and decreased growth and development were observed in pups born from rats given oral doses of 1,000 mg/kg/day during perinatal and lactation periods. There were no observed effects in rats treated with 100 mg/kg/day.

There are no adequate and well-controlled studies of bepotastine besilate in pregnant women. Because animal reproduction studies are not always predictive of human response, BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

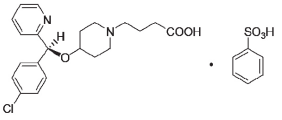
8.3 Nursing Mothers
Following a single 3 mg/kg oral dose of radiolabeled bepotastine besilate to nursing rats 11 days after delivery, the maximum concentration of radioactivity in milk was 0.40 mcg-eq/mL 1 hour after administration; at 48 hours after administration the concentration was below detection limits. The milk concentration was higher than the maternal blood plasma concentration at each time of measurement.

It is not known if bepotastine besilate is excreted in human milk. Caution should be exercised when BEPREVE (bepotastine besilate ophthalmic solution) 1.5% is administered to a nursing woman.

8.4 Pediatric Use
Safety and efficacy of BEPREVE (bepotastine besilate ophthalmic solution) 1.5% have not been established in pediatric patients under 2 years of age. Efficacy in pediatric patients under 10 years of age was extrapolated from clinical trials conducted in pediatric patients greater than 10 years of age and from adults.

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

11 DESCRIPTION
BEPREVE (bepotastine besilate ophthalmic solution) 1.5% is a sterile, topically administered drug for ophthalmic use. Each mL of BEPREVE contains 15 mg bepotastine besilate.
Bepotastine besilate is designated chemically as (+) -4-[[(S)-p-chloro-alpha -2-pyridylbenzyl]oxy]-1-piperidine butyric acid monobenzenesulfonate.
The chemical structure for bepotastine besilate is:



Bepotastine besilate is a white or pale yellowish crystalline powder. The molecular weight of bepotastine besilate is 547.06 daltons. BEPREVE® ophthalmic solution is supplied as a sterile, aqueous 1.5% solution, with a pH of 6.8.
The osmolality of BEPREVE (bepotastine besilate ophthalmic solution) 1.5% is approximately 290 mOsm/kg.
Each mL of BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% contains:
Active: Bepotastine besilate 15 mg (equivalent to 10.7 mg bepotastine)
Preservative: benzalkonium chloride 0.005%
Inactives: monobasic sodium phosphate dihydrate, sodium chloride, sodium hydroxide to adjust pH, and water for injection, USP.

12 CLINICAL PHARMACOLOGY
12.1 Mechanism of Action
Bepotastine is a topically active, direct H_1-receptor antagonist and an inhibitor of the release of histamine from mast cells.

12.3 Pharmacokinetics
Absorption: The extent of systemic exposure to bepotastine following topical ophthalmic administration of bepotastine besilate 1% and 1.5% ophthalmic solutions was evaluated in 12 healthy adults. Following one drop of 1% or 1.5% bepotastine besilate ophthalmic solution to both eyes four times daily (QID) for seven days, bepotastine plasma concentrations peaked at approximately one to two hours post-instillation. Maximum plasma concentration for the 1% and 1.5% strengths were 5.1 ± 2.5 ng/mL and 7.3 ± 1.9 ng/mL, respectively. Plasma concentration at 24 hours post-instillation were below the quantifiable limit (2 ng/mL) in 11/12 subjects in the two dose groups.

Distribution: The extent of protein binding of bepotastine is approximately 55% and independent of bepotastine concentration.

Metabolism: *In vitro* metabolism studies with human liver microsomes demonstrated that bepotastine is minimally metabolized by CYP450 isozymes.

In vitro studies demonstrated that bepotastine besilate does not inhibit the metabolism of various cytochrome P450 substrate via inhibition of CYP3A4, CYP2C9, and CYP2C19. The effect of bepotastine besilate on the metabolism of substrates of CYP1A2, CYP2C8, CYP2D6 was not studied. Bepotastine besilate has a low potential for drug interaction via inhibition of CYP3A4, CYP2C9, and CYP2C19.

Excretion: The main route of elimination of bepotastine besilate is urinary excretion (with approximately 75-90% excreted unchanged in urine).

13 NONCLINICAL TOXICOLOGY
13.1 Carcinogenesis, Mutagenesis and Impairment of Fertility
Long-term dietary studies in mice and rats were conducted to evaluate the carcinogenic potential of bepotastine besilate. Bepotastine besilate did not significantly induce neoplasms in mice receiving a nominal dose of up to 200 mg/kg/day for 21 months or rats receiving a nominal dose of up to 97 mg/kg/day for 24 months. These dose levels represent systemic exposures approximating 350 and 200 times that achieved with human topical ocular use. The no observable adverse effect levels for bepotastine besilate based on nominal dose levels in carcinogenicity tests were 18.7 to 19.9 mg/kg/day in mice and 9.6 to 9.8 mg/kg/day in rats (representing exposure margins of approximately 60 and 20 times the systemic exposure anticipated for topical ocular use in humans).

There was no evidence of genotoxicity in the Ames test, in CHO cells (chromosome aberrations), in mouse hepatocytes (unscheduled DNA synthesis), or in the mouse micronucleus test.

When oral bepotastine was administered to male and female rats at doses up to 1,000 mg/kg/day, there was a slight reduction in fertility index and surviving fetuses. Infertility was not seen in rats given 200 mg/kg/day oral bepotastine besilate (approximately 3,300 times the systemic concentration anticipated for topical ocular use in humans).

14 CLINICAL STUDIES
Clinical efficacy was evaluated in 2 conjunctival allergen challenge (CAC) studies (237 patients). BEPREVE (bepotastine besilate ophthalmic solution) 1.5% was more effective than its vehicle for relieving ocular itching induced by an ocular allergen challenge, both at a CAC 15 minutes post-dosing and a CAC 8 hours post dosing of BEPREVE.

The safety of BEPREVE was evaluated in a randomized clinical study of 861 subjects over a period of 6 weeks.

16 HOW SUPPLIED/STORAGE AND HANDLING
BEPREVE® (bepotastine besilate ophthalmic solution) 1.5% is supplied in a white low density polyethylene plastic squeeze bottle with a white controlled dropper tip and a white polypropylene cap in the following size:
5 mL (NDC 24208-629-02)
10 mL (NDC 24208-629-01)

STORAGE
Store at 15º – 25ºC (59º – 77ºF).

17 PATIENT COUNSELING INFORMATION
17.1 Topical Ophthalmic Use Only
For topical ophthalmic administration only.

17.2 Sterility of Dropper Tip
Patients should be advised to not touch dropper tip to any surface, as this may contaminate the contents.

17.3 Concomitant Use of Contact Lenses
Patients should be advised not to wear a contact lens if their eye is red. Patients should be advised that BEPREVE should not be used to treat contact lens-related irritation.

Patients should also be advised to remove contact lenses prior to instillation of BEPREVE. The preservative in BEPREVE, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted after 10 minutes following administration of BEPREVE.

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with TMVL. In adult patients with symptoms of acute TMVL without migrainous features, we begin our workup with a complete neuro-ophthalmic history and ophthalmologic exam. We palpate the temporal arteries and assess the neck and trapezius muscles for pain to rule out carotid dissection. If, during a normal exam, the full threshold visual field shows no localization or pattern suggestive of neurologic distress, discuss the case with the primary care physician and order a CMP, ESR, CRP, lipid panel and carotid artery duplex within 24 hours.

Urgent serology allows us to determine the presence of inflammatory disease and comorbid vascular abnormalities. Because of its bimodal properties, the carotid duplex will evaluate the velocity and direction of blood flow, as well as obtain an image of the carotid artery. With a normal duplex and absence of elevated ESR and CRP, we recommend evaluating for blood dyscrasias and obtaining a consultation for a neurologic evaluation. Alternatively, if stenosis is noted on the duplex, the patient should be scheduled for urgent MRA and MRI of the head and neck to obtain better visualization of the carotid artery for surgical intervention and evaluate the soft tissue of the brain for ischemia. In the absence of intracranial pathology, the patient should then be referred to a neurologist for a TIA workup prior to undergoing surgical intervention with a vascular surgeon. Lastly, the patient should undergo a cardiac evaluation, as there is a 33% risk of associated myocardial infarction.[7]

While patients who experience typical negative visual symptoms of amaurosis fugax have a greater risk of carotid artery stenosis, positive visual phenomena are less diagnostic. Given the findings of severe carotid artery stenosis, ulceration and subacute cerebral infarction, we believe that the positive visual phenomena our patient experienced indicated vascular insufficiency. While she had no complaints of somatosensory or motor weakness, isolated positive visual phenomena are a risk for carotid artery disease, and proper evaluation can reduce the risk of morbidity and mortality. ■

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A Grain of Salt?

Skeptics wonder if osmolarity testing lacks clinical value. Here's why they're wrong.

By Paul M. Karpecki, OD

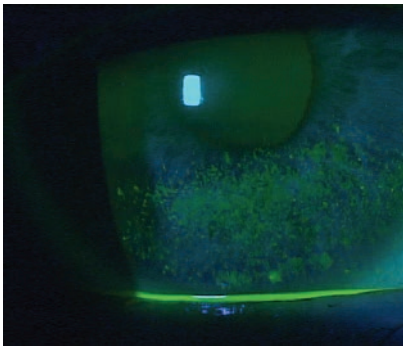
Darwin noted that those who are best able to adapt are the ones who survive. So it's no surprise that much of a clinician's success can be attributed to their ability to adapt to change; in optometry, this requires time, research, observation and evidence. When all of this leads to improved patient care and more effective management, the new way of thinking takes hold.

Two recent major changes from traditional thinking stand out. First is the realization that dry eye disease (DED) is often asymptomatic, and, second, osmolarity testing is not just a number used for patient compliance purposes; it can actually change the diagnosis and treatment plan.

For decades we've been taught that dry eye is a symptomatic disease and treatment will make your patients happy. Yet, research suggests that relying on symptoms to diagnose DED would produce an incorrect diagnosis over 40% of the time.[1-3] Imagine if you had to remake spectacles over 40% of the time—it'd be hard to build a practice on a process that error prone; it's the same with building an OSD or DED practice.

Signs vs. Symptoms

So why is there such a disparity between signs and symptoms? Research shows that we may not be asking all the right questions; for example, we rarely ask about blurred vision, yet it is one of the most consistent symptoms of DED in all severity levels.[4] Additionally, although tear fluid hyperosmolarity initially increases nerve activity of cold thermoreceptor endings in the cornea, leading to symptoms of dryness, chronic dry eye disease shows that symptoms dissipate as the disease progresses and corneal hypoesthesia develops.[5,6] If inflammation-induced hypersensitivity to polymodal or cold receptors occurs, patients may develop a neuropathic dry eye, which is extremely difficult to manage.[7] To make DED symptomatology even more complex, Sjögren's syndrome patients with longstanding aqueous tear deficiency and keratoconjunctivitis sicca (KCS) have reduced corneal sensitivity, yet they still complain of more irritation than other longstanding DED patients.[8]



Corneal staining is a late-stage disease indicator.

Because DED symptoms go through a cycle, a patient whose inflammation is not addressed (e.g., uses only OTC artificial tears) during the symptomatic phase of the disease may actually progress until the nerves are altered or downregulated, causing the patient to believe the disease has resolved. In actuality, it has likely progressed and the patient will only seek help when vision is significantly decreased due to advanced DED signs. These patients are not mentioning dryness, burning, grittiness or any typical dry eye symptoms, making it difficult to diagnose correctly.

With chronic inflammation, alterations in corneal nerve morphology develop, including thicker appearing stromal nerves (but no increase in nerve density) and nerve growth of cone-like structures often associated with dendritic antigen-presenting cells, thus implicating longstanding inflammation as a cause.[6-8] If it persists long enough, neuropathic pain, potentially involving the central and peripheral trigeminal sensory network, may develop.[9]

Additionally, symptoms that mimic dry eye disease can be caused by numerous conditions, including asthenopia from vertical imbalance, convergence insufficiency or fixation disparity, Salzmann's nodular degeneration, recurrent corneal erosion, giant papillary conjunctivitis, allergic conjunctivitis, bacterial or viral conjunctivitis, blepharitis, pinguecula, conjunctivochalasis, etc.[10-24] Many of these conditions involve symptoms of gritty, dry, burning eyes that give the impression of DED, based on a symptomatic approach, yet the causes and management are entirely different.

Osmolarity Testing

One test that may help make the DED diagnosis without relying on symptoms alone is osmolarity testing. Although there are over 2,000 studies

on osmolarity and the ocular surface, with the majority of them (>90%) supporting the technology in DED management, many doctors remain unsure of its applications and how, or even if, it benefits clinical care.[25]

Although most clinicians will state that it is mainly used to monitor disease progression and provide a number to assist patient compliance, I disagree. The primary purpose of tear osmolarity testing is to know if a patient has dry eye disease. Hyperosmolar status, whether through decreased tear production or an increased evaporative state, indicates reduced aqueous levels.[26] The test indicates whether or not the patient has a higher salt content than normal: as the volume of aqueous declines, the salt concentration in tears increases. When using osmolarity testing in an untreated patient, if you get a reading under 290mOsmol/L and each eye is within 8mOsmol/L of the other (e.g., 281 and 285), the patient doesn't have DED—end of story.[27] Don't put them on steroids, Restasis (cyclosporine ophthalmic emulsion, Allergan) or even artificial tears. Instead, look for other causes such as the various forms of conjunctivitis, conjunctivochalasis or eye alignment issues like vertical disparity or fixation/proprioceptive disparity between the eyes.

A recent patient with symptoms of dry eyes, grittiness and burning, which were worse while working on a computer or late in the day, tested positive for inferior corneal staining, a rapid tear break-up time and a small tear meniscus. She was put on topical steroids, Restasis and artificial tears, but reported no improvement after six months and discontinued all her drops. Four months later, she was observed in our clinic and osmolarity was measured at 287 and 289—no dry eye. *Demodex* blepharitis and vertical phorias were diagnosed. Both conditions were treated, and symptoms fully resolved. Without osmolarity testing, patients such as this one are often prescribed drops for months or years to treat DED. It is only after therapies fail do we decide they don't have DED. I'd rather perform osmolarity testing and know the answer in seconds on the first evaluation.

Treating DED

Knowing patients' osmolar status can also help with treatment options. Anecdotally, through a registry of hundreds of patients, we have determined that a patient with elevated osmolarity (i.e., >320) and any level of MGD is going to respond better to a tear that lowers osmolarity more than others, such as Blink (Abbott Medical Optics) or TheraTears (Akorn), and will typically choose it over a lipid-based tear.[28-30] By contrast, a patient with <310 osmolarity and mild to moderate MGD will often choose a lipid-based tear—such as Retaine MGD

(Ocusoft), Refresh Optive Advanced (Allergan), Systane Balance (Alcon), Soothe XP (Bausch + Lomb)—the majority of the time. Gauge the level of MGD to determine which direction to go with patients between 310 and 320.[31] Even my choice of artificial tears depends on osmolarity, MG expression and analysis.

Inflammation must be treated at all levels of DED, given the effects on symptoms and progression of the disease, but higher osmolarity measurements require more aggressive therapy. If you use topical corticosteroids and Restasis, you must alter treatment if osmolarity is not improved at two months. If osmolarity does improve but symptoms do not, you can feel confident that it's the right treatment and the patient will eventually show improvement in symptoms.[32] Proper patient education about the benefits of the right treatment will keep patients engaged in their care. It also helps with compliance when patients see a quantifiable improvement. Without osmolarity, as DED management stands now, a patient would either stay on the drop unnecessarily—even if it is doing nothing—or stop prematurely because the symptoms haven't improved, even though it's the correct treatment and just needs more time. Neither is ideal.

I have had many cases where osmolarity completely changed my dry eye management plan. Recently, I saw a patient with osmolarity of 287 and 284, with all the classic symptoms of dry eye disease. The osmolarity number prompted me to look elsewhere for the cause of discomfort. I everted the eyelid and there was a large concretion, which I only noticed on double eversion because it was at the tarsal plate margin edge. After its removal, the patient's symptoms resolved completely. He'd been treated for DED with artificial tears, topical steroids, Restasis and doxycycline for over eight months. The osmolarity readings increased my suspicion of a diagnosis other than DED.

Keep in mind that a normal osmolarity reading could also mean a DED patient that is well treated and the disease is controlled or homeostasis maintained.[26] Also, osmolarity testing should not be used in isolation; it must be combined with other findings. No one test works 100% of the time. Osmolarity may be the most accurate predictor of DED or severity of disease, but when you start combining tests (e.g., osmolarity + MG expression + corneal staining + symptoms, etc.), you begin to see a significant improvement in sensitivity and specificity of the disease diagnosis and management plan.[27,33] ■

Dr. Karpecki has a financial relationship with AcuFocus, AMO, Alcon Labs, Allergan, Akorn, Bausch + Lomb/Valeant, BioTissue, Bruder

Healthcare, Cambium Pharmaceuticals, Eleven Biotherapeutics, Eyemaginations, Essilor, Fera Pharmaceuticals, Focus Laboratories, iCare USA, Ocusoft, Freedom Meditech, Konan Medical, Beaver-Visitech, Eye Solutions, Reichert, Shire Pharmaceuticals, RySurg, Science Based Health, SightRisk, TearLab, TearScience, TLC Vision, Topcon and Vmax.

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‘Assume’ Leads to ‘Doom’

A patient presents with a simple itchy eye. Is that all it is? Or should you consider more consequential scenarios? **Edited by Paul C. Ajamian, OD**

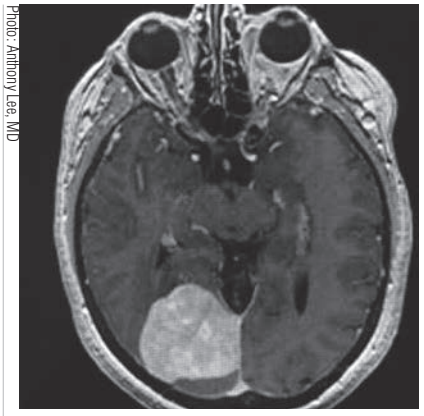
Q I had a patient recently who came in complaining of an itchy eye. She had an eye exam eight months ago. How far should I go with the workup?

A Though this patient was recently seen by another eye practice, she is new to you, warranting a full new patient examination, according to David J. Baptiste, OD, an optometrist working in Fort Worth, Texas. This consists of a comprehensive work-up, including vitals (i.e., blood pressure), retinal photos and a visual field screening test. The patient reported self-medicating with over-the-counter allergy remedies to relieve the itching, which had persisted for more than six months in her left eye. She was not wearing any refractive correction other than over-the-counter reading glasses.

While the Texas allergy season lasts the whole year round, Dr. Baptiste notes, the fact that the itching was unilateral is atypical for an allergic eye presentation. The patient’s unaided VA was 20/20 OU. Additionally, the external examination revealed swollen upper and lower lid margins as well as crusting at the base of the patient’s lashes—leading to the diagnosis of blepharitis.

Review of the entrance test results from the screening visual field testing revealed a mild defect in the lower left visual field. While possibly a false positive, it is not good practice to ignore such a seemingly mild finding. For this reason, a second test was administered 30 minutes later to confirm results, according to Dr. Baptiste. The re-test revealed a similar but more pronounced field defect. When questioned, the patient admitted to some visual impairment while driving. She specifically reported that she had difficulty merging into traffic and not always seeing cars on her left side as she entered the roadway. Based on these warning signs, Dr. Baptiste recommended the patient see a neurologist immediately. He went a step further and called to make the appointment, not leaving the referral to chance.

Photo: Anthony Lee, MD



A scan of the patient’s meningioma.

The neurologist she saw ordered an MRI and identified the tumor as a meningioma attached to the falx cerebri and tentorium cerebelli, roughly 4cm in diameter and pressing on the occipital lobe. Though the tumor was benign, meningioma growth can impact speech, motor skills and ultimately, breathing and heart function, depending on their location. The neurologist told Dr. Baptiste that he believed the tumor had been growing for roughly 15 to 20 years, and would have been inoperable within another five to 10 years.

The patient was scheduled for surgery three weeks after the initial eye examination and, ultimately, the procedure was successful. The patient recovered and returned to work six weeks later. No tumor regrowth was found at the one-year follow-up appointment. The patient remains extremely grateful for Dr. Baptiste’s life-saving catch.

So what can we, as practitioners, learn from this case? First, even if a patient recently saw another doctor, the moment she steps through your door you are now responsible for her vision and visual health. Be thorough, and don’t exhibit the mentality that “they must have done that at the other office so I don’t need to.” Assumptions like this can lead to disaster.

Second, while it is not practical to order an MRI every time a patient misses a point or two on a visual field, if there are a cluster of points missed, either on an FDT or formal automated field, it should be repeated. If the defect persists, a consult with a neurologist should be considered.

Third, do not leave it up to the patient to make the appointment, as many ultimately will. Direct your staff to make the appointment and document the time and date of the exam. If the patient fails to make the visit, any subsequent issues are on her. ■

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For Many Eyes, Many Answers

Cristina M. Schnider, OD, MBA, FAAO

Just as skin care products have been developed for different skin types and hair care products for different types of hair, contact lenses are available to address a range of vision, health, comfort and lifestyle needs. **No contact lens – no matter how great – will meet the needs of every patient**. In addition, there is a tremendous opportunity to create a real connection with a patient by personalizing the conversation around the lens recommendation, and tying it to a need that really resonates with the patient.

Each day, we take into account our patients' attitudes and lifestyle, ocular physiology, and signs and symptoms to determine which available contact lens brand is best suited to that patient. That's the art and science of contact lens prescribing. But many of us miss the opportunity to transmit all that analysis in a simple, concise and meaningful way to the patient. **Taking a few extra minutes to convey this more personalized approach to prescribing can result in a higher potential to delight patients and increase the value they place on our products and services**.

A recent study[1] involving 501 contact lens wearers and considerers, representative of the population ages 18-50, identified distinct groups of patients based on key differences in their needs and attitudes. This research also showed that with a few simple questions and observations, we can quickly identify needs that patients may otherwise not explicitly mention and select a lens that best meets those needs. For example, agreement with the statement, "I spend more than 8 hours in front of a digital screen" is highly correlated with the "demanding environments" group, while patients in the "healthy intense wear" group are more likely than those in other groups to agree that "My eye health is so important to me that I always go to someone I can really trust for the best products." Other categories include those who are prone to discomfort (prone to symptoms of lens-related dryness and irritation) and those who are appearance conscious (Table 1). **While there is potential for overlap, by combining what we hear and see in the eye examination, we can better discern the dominant need, provide a compelling rationale for our choice of lenses, and engage in a richer and more personal dialogue with the patient**.

	Questions for Patients Around Attitude, Symptoms and Lifestyle	Clinical Signs and Ocular Health Needs	Contact Lens Recommendation
Discomfort Prone	■ Do your eyes frequently feel dry, scratchy, or irritated during the day? ■ Do you feel that your eyes are sensitive? ■ Do you suffer from seasonal allergies affecting your eyes?	■ Bulbar and/or palpebral conjunctival redness ■ Need for UV blocking†*	A lens that helps keep moisture in and irritation out such as 1-DAY ACUVUE® MOIST Brand Contact Lenses Family including Sphere, ASTIGMATISM and NEW MULTIFOCAL lenses
Demanding Environments	■ Do you spend significant time in demanding environments such as air-conditioned places, or do you spend long periods using digital devices? ■ Do your eyes feel tired after a busy day filled with a variety of activities?	■ Tear film quality issues ■ Ocular surface appears dry or dull when viewing tear film reflex ■ Need for UV blocking†*	A lens that is designed to help stabilize the tear film such as ACUVUE OASYS® Brand Contact Lenses Family including Sphere, ASTIGMATISM
Healthy Intense Wear	■ Do you want or need to routinely wear contact lenses ≥14 hours/day and ≥5 days/week? ■ Do you place a priority on the long-term health of your eyes? ■ Do you want the highest levels of UV blocking available in a contact lens?	■ Presence of corneal staining ■ Limbal redness ■ Need for UV blocking†*	A lens that is designed to help keep eyes in their natural state all day and over time[2] such as 1-DAY ACUVUE® TruEye® Brand Contact Lenses
Appearance Conscious	■ Would you like to accentuate the natural appearance of your eyes? ■ Would you like your eyes to look naturally whiter and brighter?	■ Indistinct and faded limbal ring ■ Flat or small iris appearance ■ Need for UV blocking†*	A lens that is designed to accentuate eye appearance in a natural looking way such as 1-DAY ACUVUE® DEFINE® Brand Contact Lenses

Table 1: Contact Lens Wearer Attributes, by Need Group

Sponsored by Johnson & Johnson Vision Care, Inc. Dr. Schnider is an employee of Johnson & Johnson Vision Care, Inc. Drs. Closson, Czaja, and Shah are paid consultants of Johnson & Johnson Vision Care, Inc.

Dr. Schnider: *What is your approach to contact lens prescribing?*

Dr. Closson: Patients come to us for our expertise and our knowledge of what is best for their needs. They don't always know what all the possibilities are, so my goal is always to prescribe the lens that best meets their individual needs.

Dr. Shah: That's right. When I uncover those hidden needs by listening and observing and find the right solution, they'll say things like, "You've given me my life back!" or "I can't wait to tell all my friends about this!"

Dr. Schnider: *What do you need to know to tailor your lens selection to each patient's needs?*

Dr. Closson: For new CL wearers, I want to know their expectations, their lifestyle and hobbies. For existing CL wearers, I ask a lot of questions about comfort because I know that discomfort is the leading reason for contact lens dropout. I dig into comfort not only in the beginning and end of the day but also during moments throughout the day. I listen for specific symptoms of discomfort and how those are related to their lifestyle: Is she a frequent business traveler? Is he really into sports or camping?

Dr. Shah: Lately I've also been looking for clues that a patient might be interested in eye-enhancing contact lenses. Sometimes I'll ask a successful wearer, "Hey, do you want to see something new and different?" I'll let them try out 1-DAY ACUVUE® DEFINE® Brand, and talk briefly about how it enhances the limbal ring, creating more depth and definition. The effects are subtle, but it really makes eyes look more radiant.

Dr. Czaja: It's really about listening to my patients first. And then combining that with what I see during the eye exam. That's what drives me to the best choice for that patient in particular; and when you do it that way and explain it, the patient will know you have heard them and that you know them.

Dr. Closson: And when the patient feels they have been heard, that their needs have been taken care of, you have established a special bond with that patient who will then talk about you to their families and friends. This is why I find prescribing contact lenses so rewarding – it changes people's lives and inspires loyalty and referrals.

Dr. Schnider: *Why is it important to make a brand recommendation?*

Dr. Shah: When we fit a patient, we take a lot into consideration. We do a follow-up to ensure that it's a healthy fit with good centration, coverage, movement; that there is good tear exchange and no impact to the ocular surface. We ensure that if they are using solutions, those solutions are compatible with not only the ocular surface, but with the contact lens itself. So we do a lot when we fit a patient into contact lenses. These are not just a commodity. Even within a modality, not all lenses are the same.

Dr. Closson: My exam is a full evaluation of the patient's visual and ocular health needs and the lens I ultimately choose for them is a reflection of that. It's not just a base curve and prescription that are interchangeable. I am choosing other aspects of the lens – material, wetting agent, UV protection, replacement cycle, modulus, etc. – for very specific reasons.

Reference

1. Roussopoulou E, Rose M. Contact lens wearers' attitudes and needs: Similarities and differences. BCLA 2015
2. Morgan PB, Chamberlain P, Moody K, Maldonado-Codina C. Ocular physiology and comfort in neophyte subjects fitted with daily disposable silicone hydrogel contact lenses. Cont Lens Anterior Eye. 2013;36(3):118-125. Study conducted over 365 days.

ROUND TABLE PANELISTS

Christi Closson, OD
Cornelius, OR

John P. Czaja, OD
Merrillville, IN

Arti Shah, OD, FAAO
Los Angeles, CA

DOCTOR/PATIENT CONVERSATION

Dr. Czaja shares how he recently discussed his recommendation with an IT professional who is an outdoor sport enthusiast.

"I know you said your contact lenses were 'just fine,' but I'm seeing some tear film quality issues. This is not surprising given the time you spend in front of your PC and other digital devices that make you blink five times less than usual. I've got a lens called ACUVUE OASYS® Brand that is designed to help keep the tear film stable. It also features UV blocking†* which would be another benefit to you since you spend so much time outdoors. Of course, you should still use sunscreen, wrap-around sunglasses and a hat. How does that sound?"

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

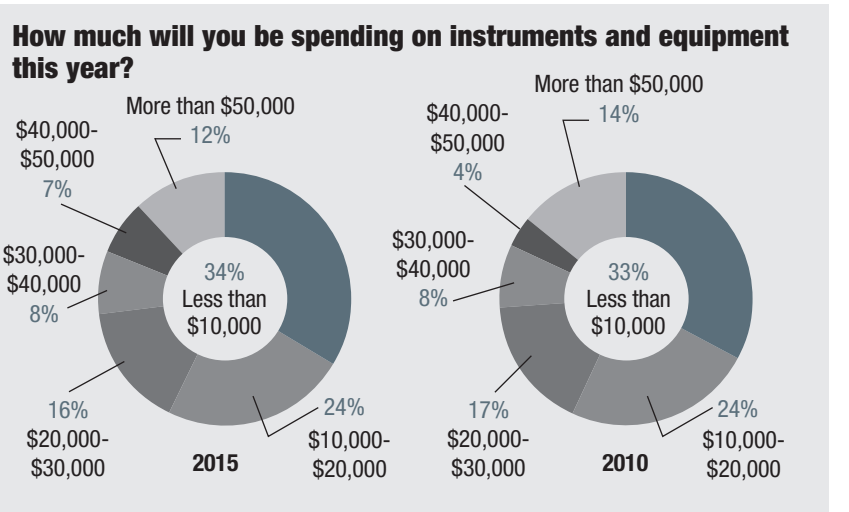
***WARNING**: UV-absorbing contact lenses are NOT substitutes for protective UV-absorbing eyewear, such as UV-absorbing goggles or sunglasses, because they do not completely cover the eye and surrounding area. You should continue to use UV-absorbing eyewear as directed. **NOTE**: Long-term exposure to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

An OD's Tech Tool WISH LIST

Improving patient care is the top priority when purchasing new technology—but some other benefits are making upgrades worth the price.

By Rebecca Hepp, Senior Associate Editor

Despite the recent explosion of technological advances in the optometric world, optometrists' must-haves haven't changed much. Digital fundus cameras and EHR software topped the list of recent purchases for the past five years, according to *Review of Optometry*'s annual diagnostic technology survey. Take a look at what more than 300 of your colleagues spent their money on last year—and how the new tools are impacting their practices.



Top Purchases

EHRs squeaked ahead of digital fundus cameras by just 1% with 30% of respondents upgrading to a new one this year (*see, "What's New in Your Office? What's Still on the Wish List?"*). That comes as no surprise, of course, given the impending regulations regarding Meaningful Use and ICD-10 just around the corner. And considering 70% of this year's respondents perform a dilated fundus exam at least once a year for patients with eye diseases such as glaucoma or diabetes, a digital fundus camera is a safe bet too. In fact, the top 10 recently purchased items are the same from last year, if a little shuffled. Last year, the top 10 purchases were:

1 and 2. Digital fundus camera, EHR (both 30% of respondents)

3. SD-OCT (24%)

4 and 5. Corneal topographer, patient callback/reminder system (both 21%)

6 and 7. Automated refraction system, Tonometer (both 20%)

8 and 9. Perimeter/visual field analyzer, OCT (both 19%)

10. Widefield scanning laser

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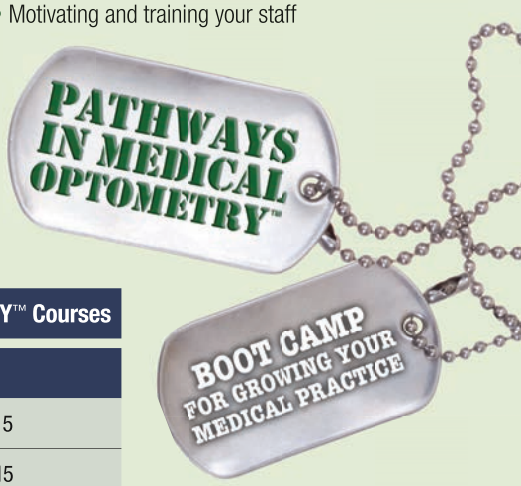

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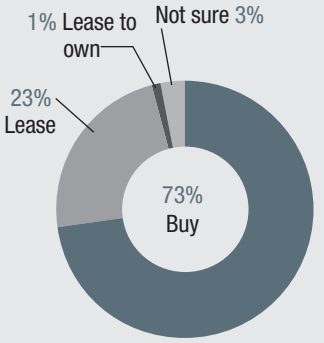
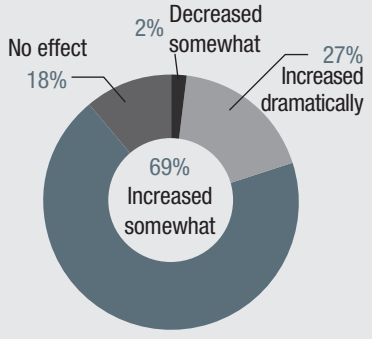


Has this new technology increased your profitability?



What's New in Your Office? What's Still on the Wish List?

ophthalmoscope (Optomap) (18%)

But technology trends have morphed some over the years. Even though patient callback/reminder systems have been in the top 10 in recent years, they weren't even an option five years ago. This year, they rank number three in recently purchased.

"Our new patient communication tool has helped make contacting patients more efficient," says Rosa Suarez-Reyna, OD, of San Antonio, Texas. "It's a lot less work for staff now."

OD Wish List

The wish list, however, is another story, and responses were all over the board this year. While nearly half (47%) of the survey takers were hankering for a new digital fundus camera in 2010, the most desired piece of equipment this year is an OCT (15%)—and that's the largest percentage of respondents. Other technologies that have doctors excited include automated refraction systems (13%), digital anterior segment cameras (12%) and, of course, digital fundus cameras (13%) and EHR systems (12%). Surprisingly, 21% don't

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April Jasper, OD
West Palm Beach, FL

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Ian Benjamin Gaddie
OD, FAAO
Louisville, KY

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Dori M. Carlson, OD
Park River, ND

"The Xfraction WOW factor with my patients is huge! They really notice and appreciate the new high-tech and integrated experience...making them more likely to come back in the future."

Nathan Bonilla-Warford
OD, FAAO
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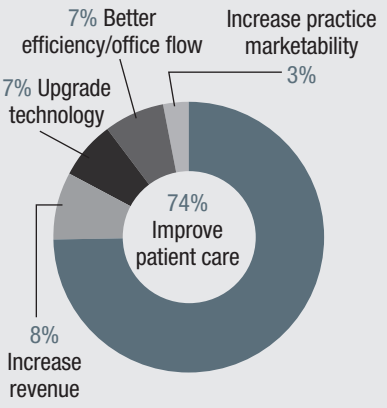
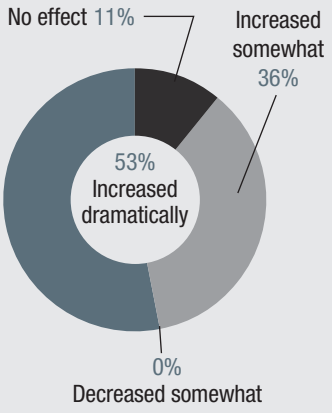
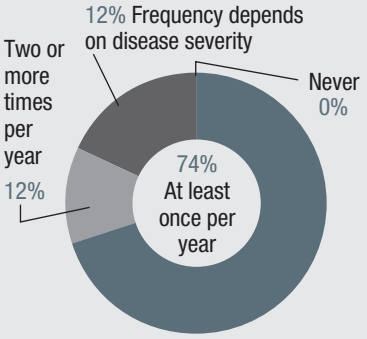
What is your most important factor for buying new technology?



Has this new technology improved patient care?



For patients with diabetic eye diseases or glaucoma, for example, do you perform fundus examinations?



have anything on their technology wish lists. Of course, nearly three-quarters of respondents who don't have plans for upcoming purchases had recently invested in more than one new instrument for their office—only one respondent has neither purchased nor plans to purchase new technology in the next few years.

Why Buy

The desire to boost patient care came in as the number one reason optometrists are upgrading their technology. Respondents seem thrilled with the outcomes too. Nearly 90% of survey takers said the new technology improved patient care, and more than half said it was a dramatic improvement to patient care. Many doctors purchased a digital fundus camera to help with patient education—with impressive results. Mariem Rodriguez Añasco, an OD in Puerto Rico, recently invested in a digital fundus camera, and offers an example of its impact: "The resolution and the photos are so sharp, and the patient loved it because she could see her retina. I was able explain everything about what's going on, and the patient said 'Oh my, I have never seen my retina before!' In that moment, I felt so proud!"

New OCTs are drastically improving patient care as well. "I was constantly having to refer patients to an ophthalmology practice for OCT," says Jennifer Keady, OD, of Burns, Ore., who recently purchased an OCT of her own. "It has made a huge difference keeping patients in office—financially and in patient satisfaction."

Tina Jang, OD, who practices in Ontario, Canada, recently took a financial hit to purchase several new tools—including a perimeter/visual field analyzer, tonometer, wide field scanning laser ophthalmoscope and an SD-OCT—and says she couldn't be happier with the decision. "Investment at the time was a bit overwhelming," she says. "However, the patient sees the difference of our exams, and [we have] improved our patients' care dramatically, which has led to patient referrals. Patients have complete confidence in our office to do a thorough eye exam when they come to see us."

Money Matters

Although patient care is priority number one, it's certainly a plus to have a boost in revenue, too. Only 8% of survey takers sought an increase in revenue as their main goal when purchasing new tools, yet 80% of those who invested in new technology saw an increase in revenue anyway. In fact, a whopping 78% of respondents who purchased something in the last year had an increase in revenue and patient care.

It's lucky so many investments pay off with more money coming in. Budgets for new equipment haven't changed a bit over the years. Most practitioners (68%) have $20,000 or less to spend on new technology this year, compared with 67% in 2010. While 73% of survey takers say they will buy their next piece of equipment, 24% are avoiding some of the financial strain by leasing. For some, it's a fantastic opportunity to get the technology you need without the worry.

"Leasing the Optomap [retinal imaging device] was one of the best investments we have made," one survey respondent says. "It

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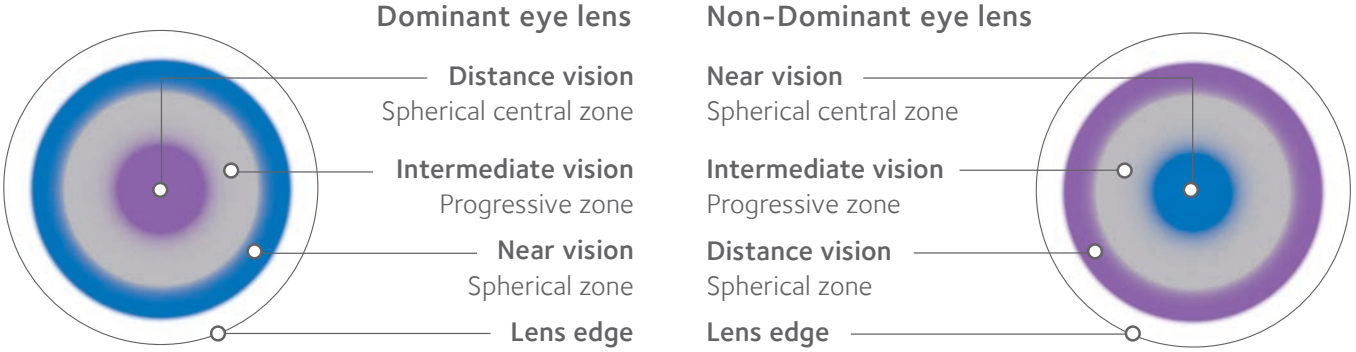

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Rave Reviews

Here are other technology purchases optometrists are thirlled they made:

• **Automated Refraction System.** "I didn't think I needed a digital refracting system until I got one," says Leslie Falcon, OD, of Chandler, Ariz. "I love the efficiency and the wow factor. Patients have told their friends about it. Kids think they look like Wall-E. The only problem is now I want one for my other exam lane."

• **Electrodiagnostic System (i.e., VEP, pERG).** "It has given us an objective measure of the nerve functioning of various areas of the optic nerve, unlike visual fields, which are subjective," says Rodney Fong, OD, who practices in Honolulu. "Patients love the high-tech aspect, and the procedure reimburses well through third party insurance."

• **OCT.** "I purchased OCT to help with glaucoma and retinal disease," says Richard Stegen, OD, of West Lebanon, NH. "It has impacted our patients and bottom line tremendously. This was by far the best tech purchase I have made to this point."

• **Slit Lamp.** "We purchased a smartphone slit lamp adapter for anterior segment photos," says Thad Wells, OD, of Russellville, Ky. "It's great for patient education and monitoring disease processes, in addition to modest profitability."

• **Tonometer.** "Patients hate the 'puff' and refer people here who have anxiety over that one simple test," says Kristin O'Brien, OD, of Denver, Colo., who purchased a new tonometer last year. "It has increased our patient's perception of us being a progressive office that does things better and differently."

has increased patient care, as we are able to see and document more in less time, which is good for our patients' ongoing health care. We find it so valuable [that] we have included it in all exams and raised our exam fee. Patients are impressed with our technology and comment all the time." ■

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How to SHOP for New Technology

Upgrading your practice's equipment can be overwhelming and expensive—but necessary. Here are some tips to ensure success.

By Rebecca Hepp, Senior Associate Editor

Technology can be both a blessing and a curse for your practice: it enables you to provide exceptional patient care, yet the push to obtain the latest and greatest gadgets can put a huge strain on your budget and your patience. If it's time to invest in new technology or upgrade your current equipment, you might be reeling from your options.

This article walks you through some of the main questions you should ask yourself when considering new equipment, as well as tips for making the shopping and purchasing experience a positive one.

Conference exhibit halls provide an excellent opportunity to see many technologies in action and get a feel for which ones will fit your practice.

What to Buy

The first step is always to decide what you truly need. While replacing a broken phoropter or slit lamp is a no-brainer, other choices aren't so obvious. Andrew S. Gurwood, OD, a professor and attending optometric physician at the Eye Institute of the Pennsylvania College of Optometry at Salus University, Philadelphia, breaks the decision down into two categories: need and economy.

"*Need* for a new instrument implies that the new instrument provides data you can't currently capture and require to practice, or will make practice easier," Dr. Gurwood says. "Need, with regard to upgrading an instrument you already have, implies that the instrument you want to upgrade is inconsistent,

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unreliable, is in disrepair or lacking new advancements which permit the user some advantage in practice," such as software that assists interpretation.

While need is usually easy to determine, *economy* often is not. It's a careful balance between what you can afford and the revenue new equipment could generate for the practice, Dr. Gurwood says. He suggests optometrists carefully consider if the cost is outweighed by the time it will save them or the reimbursements it will generate. He gives two examples:

- **Good purchase:** An auto lensometer. "There is no reimbursement, but it eliminates a tedious task that is done on almost every patient, making its value-for-dollar high," Dr. Gurwood suggests.
- **Bad purchase:** A B-scan. "This is a terrific instrument to have; but, unless you are purchasing it for the tax advantage at an average cost of $27,000 per unit, it makes little sense if your history of need over a five-year period is one time," he says.

Alan Glazier, OD, founder of Shady Grove Eye and Vision Care in Rockville, Md., and the "ODs on Facebook" page, agrees that practitioners must carefully balance need with economy, especially since not all diagnostic equipment is revenue generating.

"'Not billable' doesn't mean there isn't a business benefit, as these devices often save time, enabling more exams and add to the technical sophistication—and reputation of—the office, but there is no direct income from them," Dr. Glazier says. "All of these products need to be paid for, so it is important to understand what financial value they bring to the practice."

Photo: Justin Bazan, OD.

Nothing wows patients more than seeing nearly the entire inside of their eye. That reaction means a deeper understanding of what we are doing, and it hopefully translates into a greater desire for routine annual care.

Of course, specialists will have to budget for the specific instruments they will need to successfully run a specialist practice, Dr. Gurwood says. A retina specialist, for instance, would have greater justification for upgrading from time-domain to spectral-domain OCT than a general optometrist.

Who to Buy From

Choosing the right vendor can make all the difference in your buying and implementation experience. Drs. Glazier and Gurwood agree that reputation for service should be your number one consideration when you are deciding between vendors.

"Machines break—it's a fact," Dr. Gurwood says. "What matters is the company's response to your needs, permitting quick and accurate diagnosis of the problem and a reliable and timely solution that will permit uninterrupted, seamless ongoing business."

So make sure the vendor you choose has exceptional customer service and helpful policies such as "providing a loaner when the instrument is being repaired or providing additional training for new staffers or if the current staff isn't achieving results," Dr. Gurwood recommends.

With technology changing at such a rapid pace, a good working relationship with your vendor is a must. Whether it's software upgrades, integration concerns, hardware failures or staff training issues, you want to know your vendor will be there to help. And don't think you are on the path of technological advancement alone.

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Bill Potter, OD, chief of Optometry and Contact Lens Services at Millennium Eye Care in West Freehold, NJ, says consulting with colleagues who have recently purchased the equipment you are looking to buy can give you exceptionally useful information on their purchasing and implementing experiences. "Online forums like 'ODs on Facebook' can make this even easier," he adds.

How To Finance

The biggest hurdle, by far, is managing to finance these upgrades. To ensure you are making the best financial decision—and saving as much as you can in the process—work closely with your practice's accountant, Dr. Glazier says. A professional can make sure it's the right time to buy for tax purposes, help you research and compare vendor rates and estimate revenue generation. "You should earn more from the machine per month than you pay in a lease payment, and use conservative numbers in your calculations," Dr. Glazier advises.

But practitioners have more than one option when financing big purchases, according to Dr. Gurwood. He suggests considering a planned replacement schedule.

"Planned replacement recognizes that new and improved models of something will be released every three to five years. 'Taxing' one's practice a small percentage of the reimbursements from a given instrument with the intention of replacing it based on a predetermined schedule permits cost averaging," Dr. Gurwood explains. Cost averaging spreads the cost of the new device over time, making it far more manageable, he says. "This is a method used by municipalities to replace fire apparatus or the police fleet. Instead of realizing a $400,000 expense in one year, the 'planned' replacement time can be placed on the calendar and saved for many years, making it a painless per-day process."

But buying new equipment isn't always the best way to go. If you find yourself ogling the latest model OCT—every year—leasing instead of buying is a great option. Just like trading in your car every two or three years for the newest

7 Tips for Shopping Success

By Corinne Z. Wohl, MHSA, administrator at Delaware Ophthalmology Consultants

Wooed by new technology, it's enticing to add, replace and upgrade equipment regularly. But saying yes is the easy part. Once you determine what new technology will help you provide the best patient care, your office manager can play a key role to help ensure the process goes as smoothly as possible. Follow these steps to make the most of your purchasing experience:

1. Once you have selected the right equipment, call three doctors with practices of a similar size who already use this equipment and ask about their installation experiences. Is there anything they would do differently that they would share with you?
2. Before you make a purchase, do a physical assessment. Does it fit in the elevator or stairwell for delivery? Once installed in the lane, can a patient in a wheelchair still access it? Can your office meet the new electrical needs?
3. Consider integration with your existing equipment. If you use an EHR, will this equipment work best if integrated via wireless or cable connections? Do you need a stronger Wi-Fi signal upgrade? Does your EHR company already have a relationship with this vendor or do you need to involve them in this transition pre-purchase? What are the costs for these changes?
4. Conduct a financial analysis. Prepare a return on investment (ROI) analysis on each new purchase or upgrade. This will tell you how many tests you need to perform to pay for the new equipment and eventually make a profit. If you are purchasing the testing equipment with the assumption that it's reimbursable, it's extremely important to confirm with your participating insurance companies that they do reimburse for the CPT codes that cover these tests. Vendors anxious to sell their equipment are quick to announce that it is a covered service, but not all insurers get on board at equal speeds. If the technology isn't for reimbursable testing, how will it streamline office functioning? You can turn that answer into a simple mathematical equivalent.
5. If the insurers are not paying for this new testing, prepare a written fact sheet for patients to explain why paying for it out-of-pocket is worthwhile. This will help you discuss the new testing with your patients.
6. With a reasonable ROI anticipated, contact your bank to arrange for financing. Also contact your accountant to advise on whether it's best to finance through the bank or self finance.
7. Once you determine purchasing the equipment makes clinical and financial sense, the next step is training. Negotiate training hours in the initial purchase contract. For highly technical equipment, such as an EHR system, your office manager may have to decrease the appointment schedule for a period to allow you and your staff to become adept.

Overall, adding new technology to your office can be satisfying, as long as you take all the right steps to ensure personal and patient satisfaction, along with financial success.

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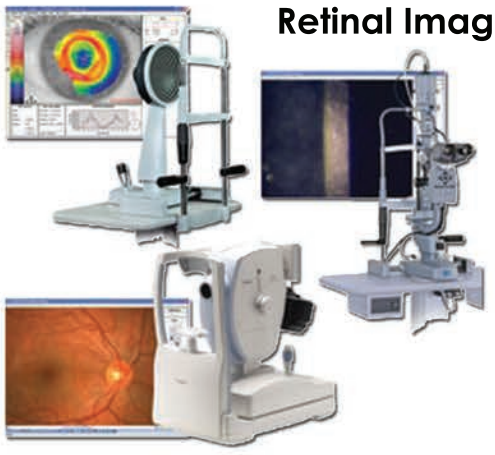

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model, leasing allows you to upgrade to the latest and greatest with little hassle.

"You would tend to lease if you are worried about Moore's Law of technology making the device obsolete in 18 months. If we are worried that the next OCT technology will render our current spectral-domain instrument obsolete, we may do a lease," Dr. Potter says. However, "we purchased some optical manufacturing equipment, when we were confident that the machine was durable and the technology would not leapfrog."

Financially, leasing can have just as many pros and cons as buying. "While leasing may have a lower payment," Dr. Potter says, "instrument values can plunge even faster than those of a car, so the lease rate may not be extremely different from the purchase."

"It becomes as much an accounting decision as a professional one, as depreciation rates vary, and every practice has different cash flow problems and needs," he adds. With all of these balls in the air, it's often best to decide if the need to upgrade warrants a lease, and then decide on a financing option.

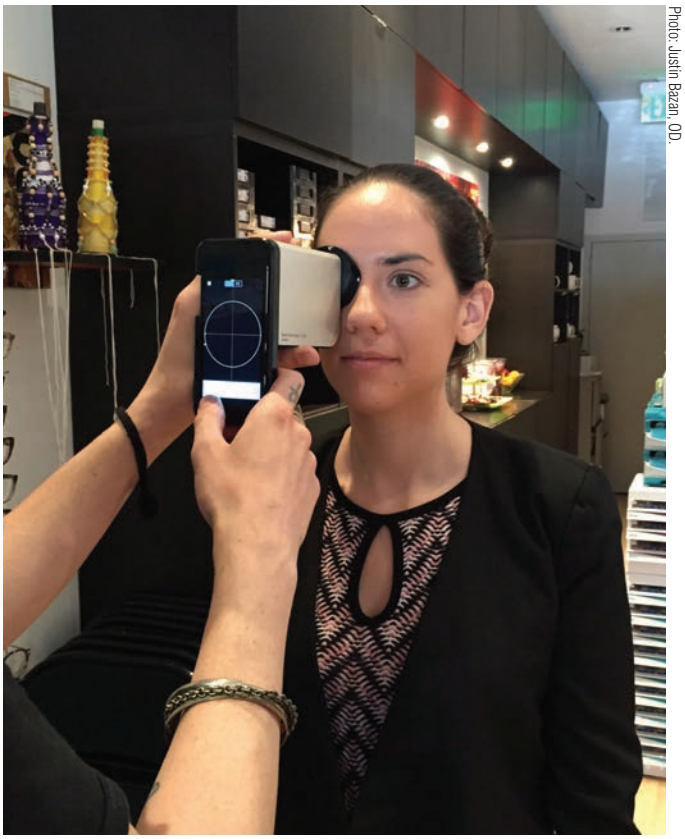


Photo: Justin Bazan, OD.

A smartphone-based autorefractor, such as the SVOne (Smart Vision Labs), eliminates patient anxeity behind a table-top autorefractor. It also helps alleviate pretest bottleneck, often speeding up subjective refraction to mere seconds.

When To Buy

If you are in the market for new technology, a conference exhibit hall might be the perfect place for you. "Conference exhibit halls usually are great places to test different equipment, check the look, feel and performance, and make comparisons all at once," Dr. Glazier says.

While the exhibits give you the chance to "kick the tires," so to speak, the vendors also often have lectures that demonstrate the benefits and applications of their technology for your practice, Dr. Gurwood says.

However, Dr. Potter cautions that exhibits tend to be unusually polished and produce optimistic presentations, so be sure to speak with the product representatives independently to make sure they have the expertise necessary to foster a positive relationship with you and your staff.

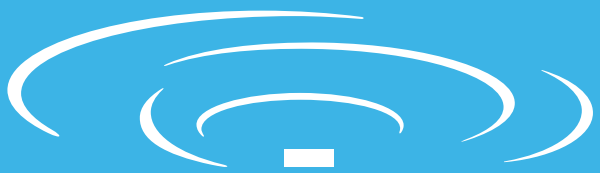
"Interviewing the demonstrating company's representative can be all-telling. No one has all the answers, but absence of expertise and failure to follow-up on a question can be death knells for the doctor-vendor relationship," he says. "Simplistically, the device has to be flawless on the exhibit floor. I recently witnessed the total failure of a testing device on an exhibit hall floor. The representatives were floored and without answers." Needless to say, those who witnessed the presentation disaster steered clear of that vendor for their future purchases.

But if everything goes well and the vendor's presentation gave you the peace of mind you needed to make an informed decision, "there is usually a conference discount if you purchase at the conference," Dr. Glazier adds. Vendors have sales quotas to hit, giving you leverage—use it!

How to Train Staff

No matter how or when you upgrade your technology, staff training is going to be an integral part of the process. Staff needs to understand why you are upgrading your equipment and how it will impact patient care, says Jason Miller, OD, a faculty member at Ohio State University and practitioner at Eye Care Professionals of Powell, Ohio.

Getting your staff on board will



LACRISERT®

(hydroxypropyl cellulose ophthalmic insert)

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ATON PHARMA Lawrenceville NJ 08648 USA

STERILE OPHTHALMIC INSERT

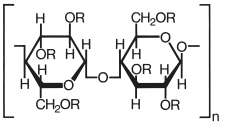
LACRISERT®

(HYDROXYPROPYL CELLULOSE OPHTHALMIC INSERT)

DESCRIPTION

LACRISERT* (hydroxypropyl cellulose ophthalmic insert) is a sterile, translucent, rod-shaped, water soluble, ophthalmic insert made of hydroxypropyl cellulose, for administration into the inferior cul-de-sac of the eye.

The chemical name for hydroxypropyl cellulose is cellulose, 2-hydroxypropyl ether. It is an ether of cellulose in which hydroxypropyl groups (-$CH_2CHOHCH_3$) are attached to the hydroxyls present in the anhydroglucose rings of cellulose by ether linkages. A representative structure of the monomer is:



$$R = CH_2CHCH_3 \quad (OH)$$

The molecular weight is typically 1×10^6.

Hydroxypropyl cellulose is an off-white, odorless, tasteless powder. It is soluble in water below 38°C, and in many polar organic solvents such as ethanol, propylene glycol, dioxane, methanol, isopropyl alcohol (95%), dimethyl sulfoxide, and dimethyl formamide.

Each LACRISERT is 5 mg of hydroxypropyl cellulose. LACRISERT contains no preservatives or other ingredients. It is about 1.27 mm in diameter by about 3.5 mm long.

LACRISERT is supplied in packages of 60 units, together with illustrated instructions and a special applicator for removing LACRISERT from the unit dose blister and inserting it into the eye. A spare applicator is included in each package.

CLINICAL PHARMACOLOGY

Pharmacodynamics

LACRISERT acts to stabilize and thicken the precorneal tear film and prolong the tear film breakup time which is usually accelerated in patients with dry eye states. LACRISERT also acts to lubricate and protect the eye.

LACRISERT usually reduces the signs and symptoms resulting from moderate to severe dry eye syndromes, such as conjunctival hyperemia, corneal and conjunctival staining with rose bengal, exudation, itching, burning, foreign body sensation, smarting, photophobia, dryness and blurred or cloudy vision. Progressive visual deterioration which occurs in some patients may be retarded, halted, or sometimes reversed.

In a multicenter crossover study the 5 mg LACRISERT administered once a day during the waking hours was compared to artificial tears used four or more times daily. There was a prolongation of tear film breakup time and a decrease in foreign body sensation associated with dry eye syndrome in patients during treatment with inserts as compared to artificial tears; these findings were statistically significantly different between the treatment groups. Improvement, as measured by amelioration of symptoms, by slit lamp examination and by rose bengal staining of the cornea and conjunctiva, was greater in most patients with moderate to severe symptoms during treatment with LACRISERT. Patient comfort was usually better with LACRISERT than with artificial tears solution, and most patients preferred LACRISERT.

In most patients treated with LACRISERT for over one year, improvement was observed as evidenced by amelioration of symptoms generally associated with keratoconjunctivitis sicca such as burning, tearing, foreign body sensation, itching, photophobia and blurred or cloudy vision.

During studies in healthy volunteers, a thickened precorneal tear film was usually observed through the slit-lamp while LACRISERT was present in the conjunctival sac.

Pharmacokinetics and Metabolism

Hydroxypropyl cellulose is a physiologically inert substance. In a study of rats fed hydroxypropyl cellulose or unmodified cellulose at levels up to 5% of their diet, it was found that the two were biologically equivalent in that neither was metabolized.

Studies conducted in rats fed ^{14}C-labeled hydroxypropyl cellulose demonstrated that when orally administered, hydroxypropyl cellulose is not absorbed from the gastrointestinal tract and is quantitatively excreted in the feces.

Dissolution studies in rabbits showed that hydroxypropyl cellulose inserts became softer within 1 hour after they were placed in the conjunctival sac. Most of the inserts dissolved completely in 14 to 18 hours; with a single exception, all had disappeared by 24 hours after insertion. Similar dissolution of the inserts was observed during prolonged administration (up to 54 weeks).

INDICATIONS AND USAGE

LACRISERT is indicated in patients with moderate to severe dry eye syndromes, including keratoconjunctivitis sicca. LACRISERT is indicated especially in patients who remain symptomatic after an adequate trial of therapy with artificial tear solutions.

LACRISERT is also indicated for patients with:

- Exposure keratitis
- Decreased corneal sensitivity
- Recurrent corneal erosions

CONTRAINDICATIONS

LACRISERT is contraindicated in patients who are hypersensitive to hydroxypropyl cellulose.

WARNINGS

Instructions for inserting and removing LACRISERT should be carefully followed.

PRECAUTIONS

General

If improperly placed, LACRISERT may result in corneal abrasion (see DOSAGE AND ADMINISTRATION).

Information for Patients

Patients should be advised to follow the instructions for using LACRISERT which accompany the package.

Because this product may produce transient blurring of vision, patients should be instructed to exercise caution when operating hazardous machinery or driving a motor vehicle.

Drug Interactions

Application of hydroxypropyl cellulose ophthalmic inserts to the eyes of unanesthetized rabbits immediately prior to or two hours before instilling pilocarpine, proparacaine HCl (0.5%), or phenylephrine (5%) did not markedly alter the magnitude and/or duration of the miotic, local corneal anesthetic, or mydriatic activity, respectively, of these agents. Under various treatment schedules, the anti- inflammatory effect of ocularly instilled dexamethasone (0.1%) in unanesthetized rabbits with primary uveitis was not affected by the presence of hydroxypropyl cellulose inserts.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Feeding of hydroxypropyl cellulose to rats at levels up to 5% of their diet produced no gross or histopathologic changes or other deleterious effects.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

ADVERSE REACTIONS

The following adverse reactions have been reported in patients treated with LACRISERT, but were in most instances mild and transient:

- Transient blurring of vision (See PRECAUTIONS)
- Ocular discomfort or irritation
- Matting or stickiness of eyelashes
- Photophobia
- Hypersensitivity
- Edema of the eyelids
- Hyperemia

DOSAGE AND ADMINISTRATION

One LACRISERT ophthalmic insert in each eye once daily is usually sufficient to relieve the symptoms associated with moderate to severe dry eye syndromes. Individual patients may require more flexibility in the use of LACRISERT; some patients may require twice daily use for optimal results.

Clinical experience with LACRISERT indicates that in some patients several weeks may be required before satisfactory improvement of symptoms is achieved.

LACRISERT is inserted into the inferior cul-de-sac of the eye beneath the base of the tarsus, not in apposition to the cornea, nor beneath the eyelid at the level of the tarsal plate. If not properly positioned, it will be expelled into the interpalpebral fissure, and may cause symptoms of a foreign body. Illustrated instructions are included in each package. While in the licensed practitioner's office, the patient should read the instructions, then practice insertion and removal of LACRISERT until proficiency is achieved.

NOTE: Occasionally LACRISERT is inadvertently expelled from the eye, especially in patients with shallow conjunctival fornices. The patient should be cautioned against rubbing the eye(s) containing LACRISERT, especially upon awakening, so as not to dislodge or expel the insert. If required, another LACRISERT ophthalmic insert may be inserted. If experience indicates that transient blurred vision develops in an individual patient, the patient may want to remove LACRISERT a few hours after insertion to avoid this. Another LACRISERT ophthalmic insert maybe inserted if needed.

If LACRISERT causes worsening of symptoms, the patient should be instructed to inspect the conjunctival sac to make certain LACRISERT is in the proper location, deep in the inferior cul-de-sac of the eye beneath the base of the tarsus. If these symptoms persist, LACRISERT should be removed and the patient should contact the practitioner.

HOW SUPPLIED

LACRISERT, a sterile, translucent, rod-shaped, water-soluble, ophthalmic insert made of hydroxypropyl cellulose, 5 mg, is supplied as follows:

NDC 25010-805-68 in packages containing 60 unit doses (each wrapped in an aluminum blister), two reusable applicators, and a plastic storage container to store the applicators after use.

Storage

Store below 30°C (86°F).

Distributed by:
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Lawrenceville, NJ 08648, USA

Manufactured by:
DPT Lakewood, Inc.
Lakewood, NJ 08701 USA

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be key to a smooth integration, he adds. He suggests the emergence of retinal imaging as an example. "It is a great tool to be able to show patients their retinal photos and potential diseases found in the retina," he explains. "If staff doesn't understand why they need to recommend this technology during the pre-exam, utilization will not be very high."

Luckily, this is where your careful vendor selection pays off. While you should make sure your new instrument is easy to use, you should also make sure the vendor takes care of installation, orientation and staff training, Dr. Gurwood says. Even though you probably won't be the one running the tests most of the time, you still need to be trained on performing them—and of course interpreting the data.

At the end of the day, no matter how high-tech your new equipment, it all boils down to proper staff education. "Utter failure results if staff is poorly trained," Dr. Potter says.

Why Patients Are the First Concern

While you are juggling financial, space and staff concerns, never lose sight of why you wanted the new technology to begin with: to improve patient care. All upgrades and new purchases should positively impact your patients' experience in your office—but sometimes that goal can get lost in the minutia of the buying process.

"The newest technology is always great and may provide a 'wow factor' with your patients," Dr. Miller says. "But if it adds more patient or staff time, it may backfire."

That new OCT may be fantastic, but shuffling patients to and from the only pretesting area you could squeeze it in could quickly create a scheduling and workflow nightmare. You want to offer your patients the best in diagnostic testing, but you don't want them waiting in line for it.

The bottleneck a single pretesting area creates "is frustrating to patients and staff and can result in less efficiency, less income and cause customer loyalty and service issues," Dr. Glazier says. He recommends purchasing more diagnostic equipment—when finances allow it—for a second pretesting area to improve your office's patient flow, thereby increasing efficiency.

Make the Most of a Technology Exhibit

By Bill Potter, OD

Here are eight questions you should ask a manufacturer's sales representative during a conference to ensure you have the right information to make a smart purchase:

1. How is this instrument different or better than prior models? Is a new version planned soon?
2. Is the instrument used in any university centers or eye hospitals?
3. What's the trend in insurance reimbursement? (This is easily verifiable, so watch for exaggerations here!)
4. What doctors are on board as consultants and researchers? Are any of them local, and can I talk to them?
5. Is there progression analysis? (if applicable)
6. What are the chances, statistically, of a false positive/false negative?
7. How does the test result influence patient care?
8. What is the service like in my area? Where is the representative located?

New equipment is your chance to improve your office's workflow, so take note of technology causing bottlenecks and add it to the short list for replacement. Dr. Miller mentions his office's patient education videos as a prime example.

"The patient educational videos we used to have for floaters, glaucoma, presbyopia and so on were always useful, but were a challenge to integrate into normal patient flow because the programs ran on the same computer as my EHR," Dr. Miller says. "Many of the new educational video systems now run on a tablet, and now we can input exam data while the patient watches the short video on a tablet, thus making us more efficient in the exam lane."

Another patient-centric consideration is the data the instrument produces. Are the printouts easy to present to the patient? As an example, Dr. Potter says he can "show you a printout on a patient with a vitreoretinal adhesion, whose description is extremely abstract without a good graphic and a model eye." Most new equipment will have good printouts, he says, but just make sure this is the case before you take on the hefty payments.

Your new technology should be worth every penny you spend. While many upgrades pay for themselves in reimbursements, the more significant value lies in the technology's ability to transform how you care for your patients. So go ahead and browse every vendor's booth at your next conference, and ask questions. Just remember who, what, when, why and how to go about being a smart shopper, and your patients—and practice—will be thrilled with your savvy purchases. ■

The Anatomy Of an OCT Scan

OCTs—and their printouts—can be tough to understand. Here's a how-to on differentiating OCT models and reading their scans.

By Jarett Mazzarella, OD, and Justin Cole, OD

Optical coherence tomography (OCT) has evolved over the last several years from a device used in only a few practices to one found in many primary eye care clinics. The technological growth in ocular imaging has allowed for profound advancements in the clinician's ability to recognize subtle abnormalities, differentiate stability from progression in ocular pathology and identify disease stages.

Clinicians have four prominent commercially available spectral-domain (SD) OCT models to choose from: Spectralis SD-OCT (Heidelberg Engineering), 3D OCT-2000 (Topcon Medical Systems), Avanti RTVue XR (Optovue), and Cirrus HD SD-OCT 5000 (Carl Zeiss Meditec). This article outlines the basics of OCT imaging and walks you through how to analyze retinal nerve fiber layer (RNFL) thickness, macular thickness and ganglion cell complex using each model.

OCT Basics

While the earliest time-domain (TD) OCT technology could only acquire 400 scans per second, current SD-OCT models, depending on the manufacturer, can capture between 26,000 and 70,000 axial-scans per second (*Table 1*).[1,2] This improvement is beneficial to the clinician because it minimizes image artifacts, makes 3D imaging possible and increases image resolution.[1,3]

Today's SD-OCT can also provide an axial resolution of 3µm to 6µm within tissues, compared with previous TD-OCT technology, which had a maximum resolution of 10µm.[3,4] The increased speed

Table 1. Comparison of Four Commercially Available SD-OCT Models

Model (Manufacturer)	Cirrus HD-OCT 5000 (Carl Zeiss Meditec)[2]	3D OCT-2000 (Topcon Medical Systems)[5]	Spectralis SD-OCT (Heidelberg Engineering)[6]	Avanti RTVue XR (Optovue)[7]
Scanning Speed (A-scans per second)	27,000-68,000*	27,000	40,000	70,000
Axial Resolution (µm in tissue)	5	5-6 (longitudinal)	3.9 (optical)	5 (depth)
Minimum Pupil Diameter (mm)	2	>2.5	2.5	2.5
Focus Adjustment (diopters)	+20 to -20 (internal)	+12 to -13 (in fundus photo)	+24 to -24	+20 to -15

*Cirrus OCT (5000 model with version 6.5 software) acquires 68,000 scans per second with fundus photos

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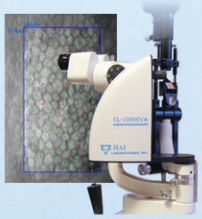
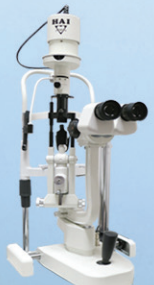


HAI CL-1000eva Endothelium Viewing Attachment

HAI SL-5000bx Basic Slit Lamp

HAI SL-5000s Standard Slit Lamp



HAI SL-5000p Plus Slit Lamp

Table 2. Comparison of Imaging Modes and Capabilities by Model

	Cirrus HD-OCT 5000[2]	3D OCT-2000[5]	Spectralis SD-OCT[6]	Avanti RTVue XR[7]
Imaging Modes	SD-OCT, confocal scanning laser ophthalmoscope (cSLO)	SD-OCT, near IR color fundus photos	SD-OCT, IR fundus photo with cSLO	SD-OCT
Fundus Photos Scanning Modes	IR	Color, FA, FAF, red-free	IR, blue laser FAF, red-free photos, FA, ICG angiography, panning camera	Live IR
Image Field of View	Fundus photo, 36x30 degrees	Fundus photo, 30-degree digital zoom	Fundus photo, 30x30 degrees (optional 55 degree lens)	OCT, 40 degree wide-field reference scan

and resolution provide an enhanced ability to visualize retinal layers.

With eye tracking capability and image registration, artifacts are much less common than in prior technology. These models also have increased fundus camera resolution and focus options for detailed image capture compared with prior TD-OCT (*Table 2*). OCT's ability to define particular layers of the retina, known as "segmentation," as well as depth localization in tissue, also aids in identifying points of interest within the scans. Two more benefits of new OCT software are 3D reconstruction of the retinal image and structural comparison functions, which compare a lesion within an area of the OCT image to the associated location visible on a retinal photograph. Current models allow image acquisition with miotic pupils, which is advantageous for patients who struggle with dilation due to age or other factors.

OCT Models

The Cirrus HD-OCT's current software features additional layouts that enable the clinician to see nerve and macular findings on a single printout and formulate functional and structural relationships between visual fields and OCT data, respectively.[3] An upcoming software algorithm is expected to add a widefield analysis of the posterior pole, including the optic nerve head (ONH), RNFL and ganglion cell analysis (GCA), in one printout.[3]

The 3D OCT-2000 uses a high resolution, 16.2 megapixel fundus camera as well as a color touchscreen.[5] The software provides the clinician with same-time image viewing of the fundus photo and OCT. The software also locates the OCT line scan in the fundus photo for easy comparison between imaging modalities.[5]

Table 3. Scan Acquisition and Analyzing Options by Model

Model	Cirrus HD-OCT 5000[2]	3D OCT-2000[5]	Spectralis SD-OCT[6]	Avanti RTVue XR[7]
Scanning Range	Retina/nerve, anterior segment	Retina/nerve, anterior segment	Retina/nerve	Retina/nerve, cornea, angle
Scan Acquisition: Macula	Macular cube (512x128, 200x200), HD five-line raster	3D scan, radial scan, seven-line raster scan	Line, star, volume	3D cube (320x320), widefield imaging (12mmx9mm), line, HD-line, crossline, HD-crossline 3D macula, radial slices, multiple patterns
Scan Acquisition: Nerve	Optic disc cube (200x200)	3D scan, circle scan	Circle, volume	RNFL 3.45
Scan Acquisition: Anterior Segment	five-line raster, cornea, manual angle	Radial corneal scan, line angle scan	Not on standard model	Cornea line, cornea crossline, angle, raster, 3D cornea
Posterior Segment Analysis	Macula: macular thickness, macular change, ganglion cell, RPE change Nerve: RNFL thickness, guided progression 3D imaging	Glaucoma and macula report (12mmx9mm wide scan) Macula: 3D macula report, macular drusen analysis Nerve: 3D disc report, RNFL trend analysis, glaucoma analysis	Macula: Real time, fast, dense, detail, posterior pole, seven lines Nerve: Fast, dense, posterior pole, nerve head circle	Widefield enface mapping Combined RNFL and ganglion cell change report Macula: Retinal trend analysis, ganglion cell complex, retinal overview report, multilayers enface report Nerve: Retinal nerve fiber and optic disc, optic disc structure analysis
Anterior Segment Analysis	Cornea: Manual thickness Angle: Manual angle measurement	Cornea: Thickness map, curvature and radius Angle: Manual angle measurement	Not on standard model	Cornea: Corneal power, pachymetry map, epithelium thickness map Angle: Automated measurement

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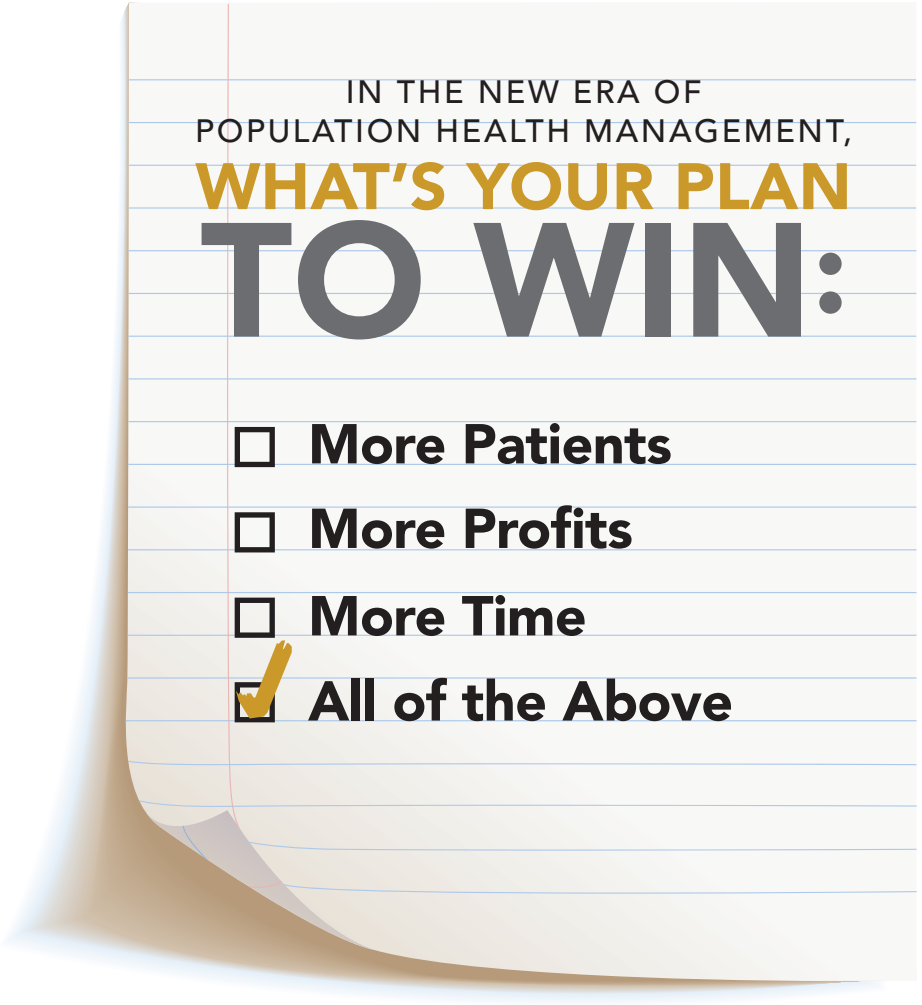

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The Spectralis SD-OCT's imaging modalities include fluorescein angiography (FA), red-free images, indocyanine green (ICG) angiography, infrared photography and ultra-widefield angiography. The new OCT2 module is an upgrade option that offers a 70,000Hz to 85,000Hz scan rate.[6]

The Avanti RTVue XR software has motion correction and can evaluate multiple layers of the peripheral retina simultaneously. Alternative models available to the clinician include the iVue, which is a more compact instrument. It has similar specifications as its larger counterpart, but may be beneficial to doctors who need to use the device in multiple practice locations or outside the office, as in nursing home care.[7]

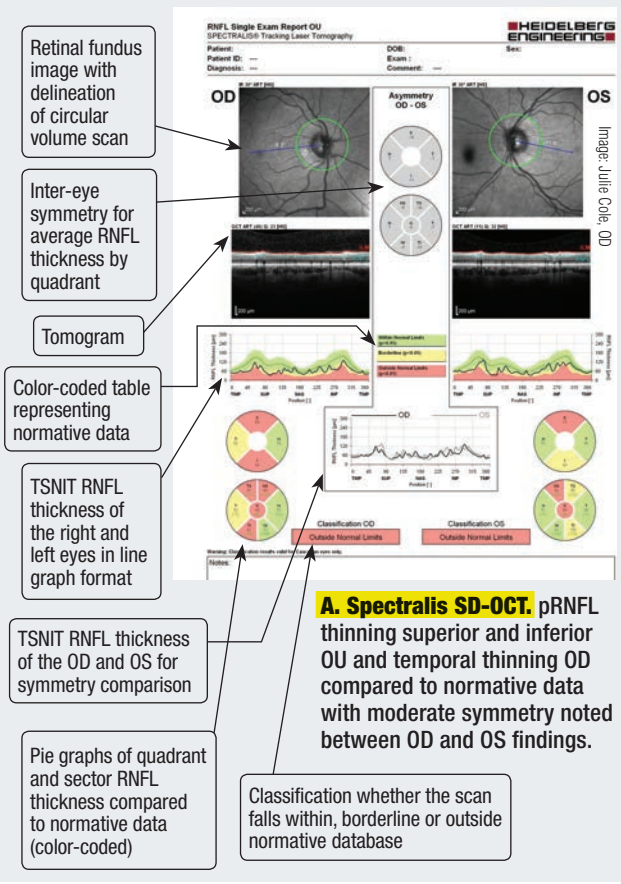
All instruments possess a retinal thickness scanning mode as well as an optic nerve and RNFL acquisition and analysis (*Table 3*). All OCT models allow for the change of the size, length and width of the line scans, depending on the pathology imaged. Current OCT systems also possess recognition software and progression or change functions to accurately track and monitor progression over time based on each model's normative data (*Table 4*).

SD-OCT Limitations

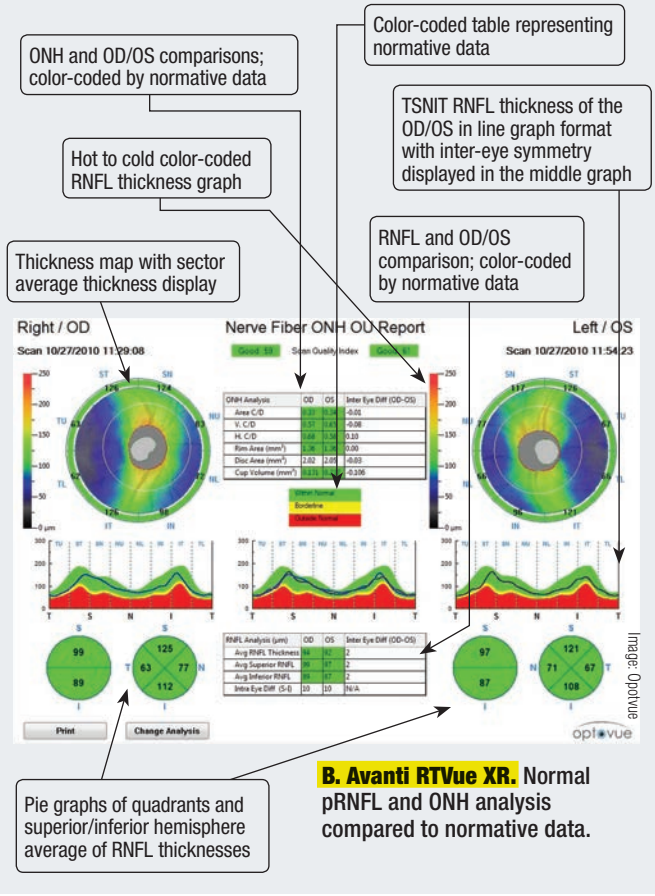
Recent literature suggests that, although reproducible OCT findings are common among healthy individuals, variable results can occur when retinal pathology is present due to the differences in acquisition and boundary identification between machines.[8-11]

Along with variability in retinal presentations, clinicians can encounter ambiguous findings of non-glaucomatous optic neuropathies and normal optic nerve anatomy, which can lead to false positive and false negative OCT results. Researchers have determined that nerve diameters greater than 4.0mm^2 can falsely affect the accuracy of nerve fiber analysis results.[3,12] Accuracy is also limited by high refractive error and axial length. Longer eyes can artificially cause thinner peripapillary retinal

Figures A-D. RNFL Thickness Analysis By Manufacturer



A. Spectralis SD-OCT. pRNFL thinning superior and inferior OU and temporal thinning OD compared to normative data with moderate symmetry noted between OD and OS findings.



B. Avanti RTVue XR. Normal pRNFL and ONH analysis compared to normative data.

nerve fiber layer (pRNFL) measurements, while shorter eyes may falsely represent higher pRNFL values due to camera magnification.[13]

Repeatability of RNFL and retinal thickness measurements over time must also be considered when monitoring for progression of an ocular disease state. Researchers determined that SD-OCT has an inter-visit tolerance limit of 95% for average pRNFL—equivalent to approximately 4µm.[14] Cross sectional studies suggest a normal age-related decline of RNFL is 0.2µm per year, and the rate of decline may be higher in eyes with greater baseline retinal nerve fiber layer thickness.[13,15] These considerations must be taken into account when evaluating OCT data and managing progressive ocular conditions.

Studies suggest that clinicians should be cautious when comparing thickness measurements from one brand of OCT with another in the

Common OCT Abbreviations and Terminology

A-Scan/B-Scan: Axial images allowing for 3D representation/line scan of longitudinal images

Deviation Map: Graph comparing patients' deviation to normative age-matched database

EDTRS: Early Treatment Diabetic Retinopathy Study

En face OCT: View of retina or optic nerve as a clinician would view during funduscopy

GCL/GCA/GCC: Ganglion cell layer/ganglion cell analysis/ganglion cell complex

GCL-IPL: Ganglion cell layer-inner plexiform layer

Line Scan: Scan through a tissue which can be adjusted to orientation

ONH: Optic nerve head

Raster: Scan generally consisting of five lines that can have various spacing and orientation (customizable)

RNFL/pRNFL: Retinal nerve fiber layer/peripapillary retinal nerve fiber layer

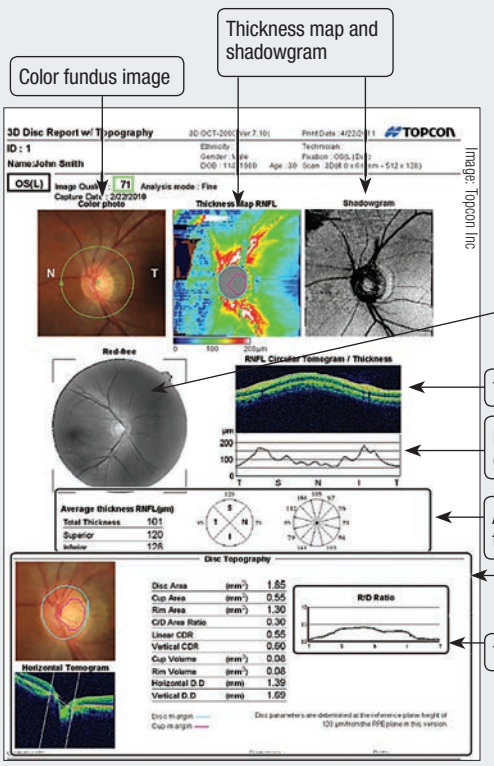
SD-OCT: Spectral-domain optical coherence tomography

Thickness Map: Graph comparing retinal thickness to normative age-matched database

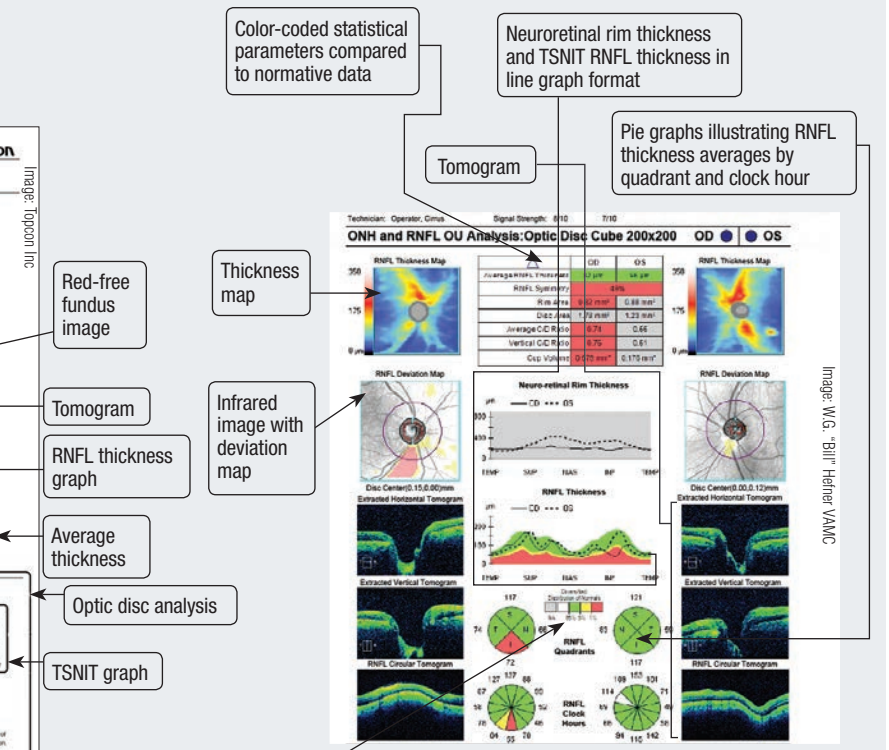
Tomogram: A two-dimensional image of a slice through a tissue (i.e., retinal tissue)

TSNIT: Linear graph of concentric nerve fiber layer thickness in respect to normative database

Volume Scan: 3D representation formed from the vertical & horizontal line scans, representing a block or cube of retinal tissue



C. 3D OCT-2000. A tilted nerve OS with topographical summary and no apparent pRNFL thinning or ONH statistical outliers.



D. HD-OCT 5000. pRNFL and cup-to-disc asymmetry OD greater than OS with inferior pRNFL thinning OD compared to normative data.

Distribution of normal: green correlates to a 95% normal distribution; yellow indicates less than 5% but greater than 1% normal distribution; red is associated with less than a 1% normal distribution, meaning that less than 1% of normal patients will have this measurement compared to Zeiss' defined normative data

same generation due to differences in algorithm acquisitions between machines.[14,16] This often leads to a difficult decision for a provider looking to make a change from an OCT model of one company to another company's when baseline data has already been obtained on established patients.

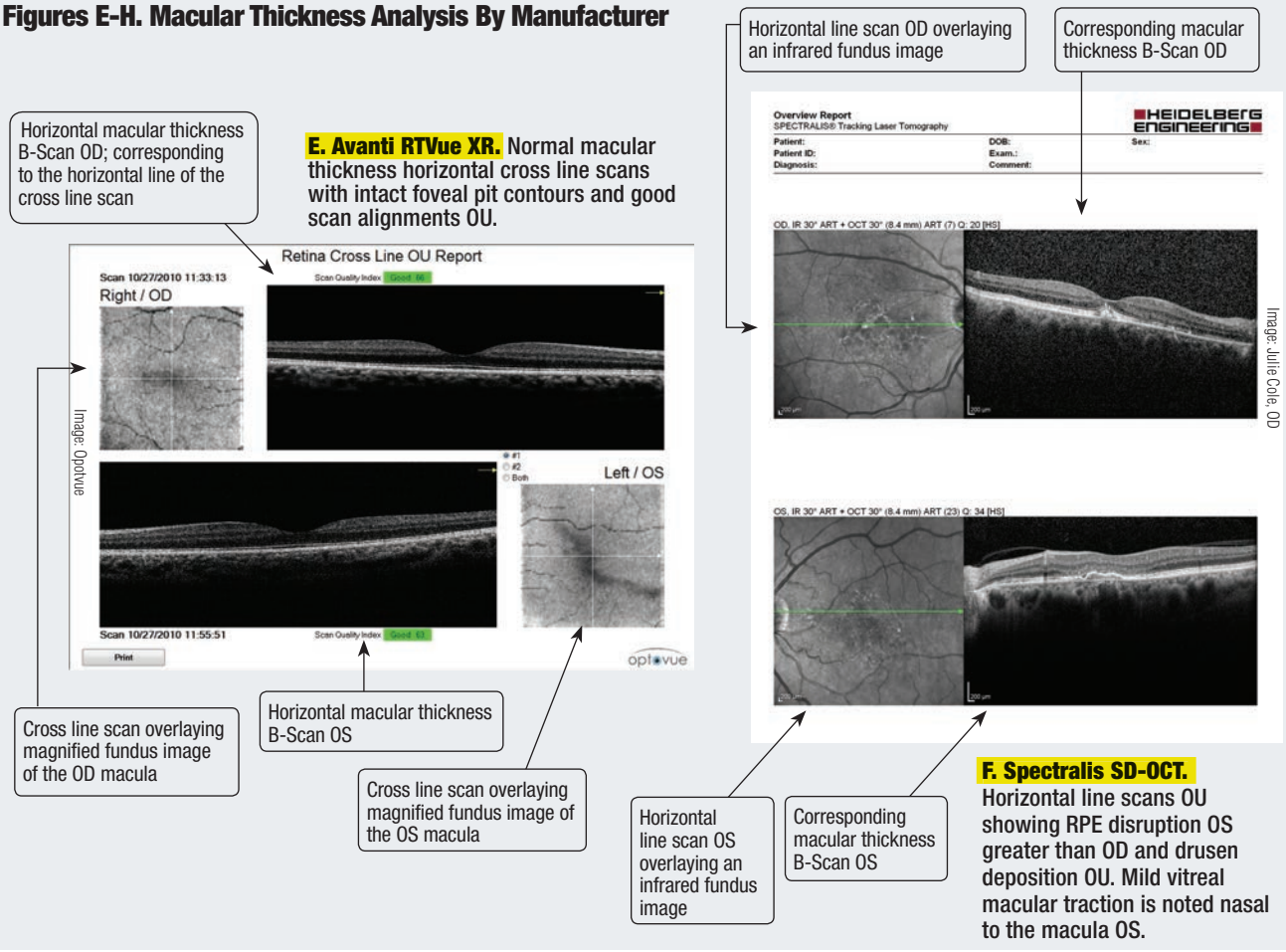
Although SD-OCT technology gives highly reproducible results in optimal patients, the technology can still be confounded by poor signal strength. Common causes include media opacity, ocular surface disease, reduced tear film quality, miotic pupils and image or motion artifacts. Software algorithm errors, including improper identification of anatomical boundaries and scan misalignment, can lead to decreased reliability and repeatability in testing.[17,18] Poor patient cooperation secondary to ocular saccades, blink artifacts and conditions such as tremors and nystagmus can result in poor image capture, even by a seasoned operator.

Despite these variables, OCTs are a valuable tool in clinical practice, especially given the limitations of other, more subjective ocular ancillary tests. For example, fundus photography can have low inter-observer agreement, especially when evaluating diffuse retinal nerve fiber loss. Alternatively, in visual field testing, reduced reliability can occur due to the subjectivity of the patient's responses. Also, the sensitivity of visual field testing in diagnosing early disease processes needs to be considered, as significant retinal ganglion cell (RGC) loss often occurs before visual field defects are evident.[19]

Comparing OCT Scans

While all four SD-OCT models are comparable in many ways, each one presents the information differently. *Figures A-K* are comparisons of RNFL thickness analysis, macular thickness analysis and ganglion cell complex analysis as represented by each model.

Figures E-H. Macular Thickness Analysis By Manufacturer



The Future of OCT

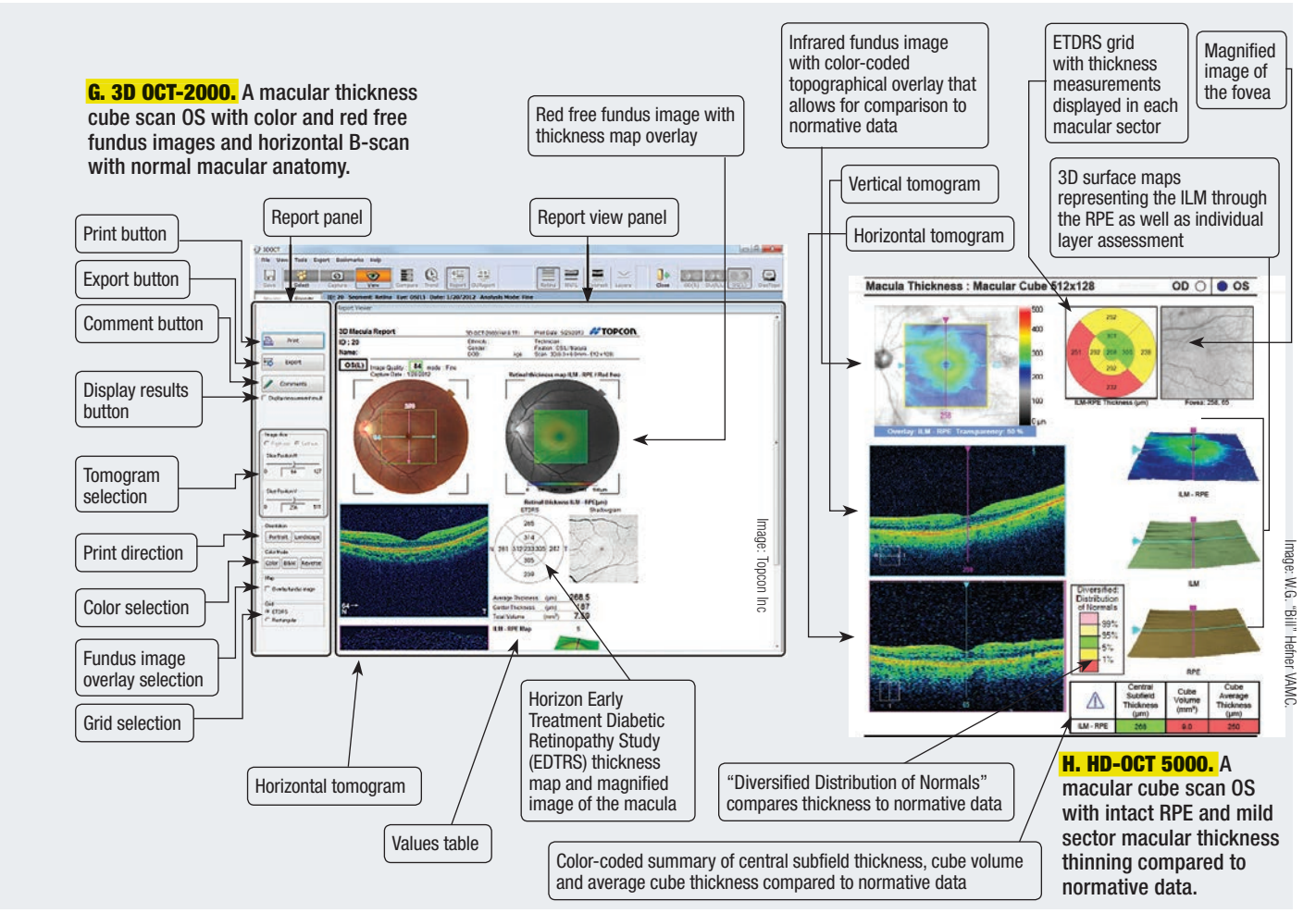
A significant challenge with OCT technology is keeping up with advances without losing previous data during system and software upgrades. Clinicians may have to establish an entirely new baseline when transferring care to a newer model OCT—a challenge clinicians faced with the transition from TD-OCT to SD-OCT. Time will tell if swept source (SS) OCT technology, the newest iteration, will lead to similar consequences.

SS-OCT has improved acquisition speed, volume and depth of ocular tissue measurements compared with SD-OCT technology.[2] This technology has the potential to provide excellent image resolution from the posterior hyaloid face through the choroid without the need for multiple image averaging or loss of internal retinal layer image quality when viewing deeper retinal or choroidal structures, as is the case of enhanced depth imaging functions with SD-OCT. The first commercially available SS-OCT, the DRI OCT Triton (Topcon Europe Medical BV) has a scan speed of 100,000 A-scans per second and uses a 1050nm wavelength to pass through cataracts and retinal hemorrhages.[20]

OCT has also been adapted to surgical suites for intraoperative use, and portable models have been incorporated into neonatal ophthalmologic care.[21] Handheld OCT models have been used for screening purposes in primary care settings to evaluate potential pathologies of the eye, ear and skin.[22]

OCT is also being used in cardiology, dermatology and respiratory medicine, and is under investigation as a possible tool to monitor cancer progression and response to therapeutic intervention.[23-26]

The combination of Doppler and OCT—referred to as phase variable OCT—and other similar technologies collectively referred to as OCT-angiography (OCT-A), can give a representation of retinal blood flow and provides the ability to define capillary networks in tissue.[2] The



G. 3D OCT-2000. A macular thickness cube scan OS with color and red free fundus images and horizontal B-scan with normal macular anatomy.

H. HD-OCT 5000. A macular cube scan OS with intact RPE and mild sector macular thickness thinning compared to normative data.

Figures I-K. Ganglion Cell Complex Thickness Analysis By Manufacturer

I. Avanti RTVue XR. A combined GCC and RNFL thickness progression map OD demonstrates thinning of the RNFL and GCC compared with normative data with no statistically significant progression over serial scans.

GCC thickness map showing thickness change over multiple visits

OD RNFL thickness map showing change over multiple visits

Graphs showing RNFL and GCC thickness, respectively, on the y-axis over age on the x-axis. The graphs demonstrate rate of change over multiple visits with best fit line and prediction capability

Color-coded statistical analysis of the GCC and RNFL parameters compared to normative data

TSNIT graph with different color lines representing each visit and corresponding to RNFL thickness in each peripapillary quadrant

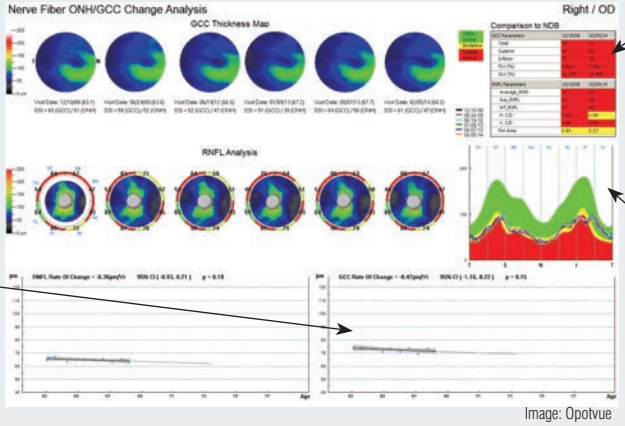


Image: Opotvue

K. 3D OCT-2000. Combined RNFL and GCL thickness report with progression and predictive software capability over successive scans. Early thinning is noted OD and variable thinning is depicted OS on the RNFL significance map over serial scans OU. No statistically significant thinning or progression is noted on the ganglion cell scans OU over successive images.

Color-coded RNFL thickness maps over multiple visits

Significance map indicating areas of deviation from normative data overlaying a fundus image

Thickness map of the GCL-IPL OD/OS, overlaying an infrared fundus image

GCL-IPL thickness averages for each sector

Color-coded distribution of normals based on normative data

Combined change analysis for RNFL and GCL OD/OS

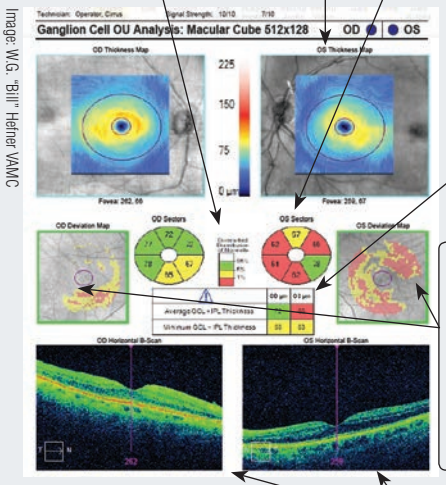


Image: W.G. "Bill" Hefner VAMC

Table of values for average and minimal thickness OD/OS

OD and OS deviation maps overlaying magnified images of the macula color-coded compared to normative data

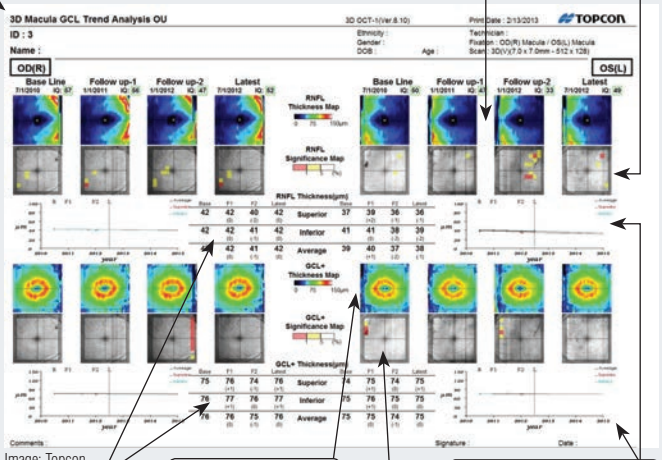


Image: Topcon

J. HD-OCT 5000. Asymmetric GCL-IPL thinning OU. Asymmetry is noted in the superior to inferior hemispheres of the ellipse OD and overall depression is depicted OS leading to significant asymmetry OS to OD. Average thickness is reduced OS and minimum thickness is reduced OU compared to normative data.

Horizontal B-scans OD/OS through the foveal pit indicating good scan alignment

Tables showing thickness measurements OD/OS for RNFL and GCL over multiple visits with progression indicated by comparison to baseline data

Color-coded GCL thickness map over multiple visits

Graphs showing RNFL thickness on the y-axis and years on the x-axis, demonstrating thickness rate of change over time

Significance map indicating areas of deviation from normative data overlaying a fundus image

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advantage of OCT-A is that no dye injection is needed, which eliminates the risk of allergic or adverse reactions and provides more visible capillary plexuses.[27] Additionally, it is not impeded by dye leakage as compared to FA testing.[27] The technology also can provide a 3D image of the retina and choroid, visualizing superficial and deep vessels, which is an advantage compared to FA and ICG imaging.[28]

The future of OCT may lie in the coupling of other imaging modalities with OCT to better evaluate structures at a molecular level.[2] The continually evolving nature of OCT technology has made it a crucial and integral part of early diagnosis of ocular disease, as well as a reliable and noninvasive way to identify disease progression. We look forward to future advancements that will augment and refine our use of OCT technology in the primary eye care setting. ■

Drs. Mazzarella and Cole are staff optometrists at the W.G. "Bill" Hefner VAMC in Salisbury, NC.

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Table 4. Comparison of SD-OCT Normative Databases by Manufacturer for US Models

Model	Carl Zeiss Meditec[1]	Topcon Medical Systems[2]	Heidelberg Engineering[3]	Optovue[4]
Number of Subjects	284	182	201	480
Ages (years)	19 to 84	19 to 84	18 to 78	18 to 84
Gender (M/F)	133 M 149 F	Disc: 54 M / 92 F Macula: 112 F / 61 M	111 M 90 F	N/A
Ethnicity	43% Caucasian 24% Asian 18% African American 12% Hispanic 1% Indian 6% Mixed Ethnicity	64% Caucasian 21% African American 15% Hispanic	Caucasian	33% Caucasian 22% Asian 20% African American 12% Hispanics 12% Indian 1% Other
Anatomy Evaluated	pRNFL thickness Optic nerve Parameters GCL + IPL thickness Macular thickness	Optic disc Macula	RNFL thickness	RNFL thickness Ganglion cell complex Macular thickness
Study Location(s)	United States, China	United States	Germany	11 Clinical sites worldwide

1. Carl Zeiss Meditec I. Cirrus HD-OCT 2015.
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4. Optivue I. 510(k) Summary Optovue, Inc. RTVue with Normative Database. Available from: www.accessdata.fda.gov/cdrh_docs/pdf10/K101505.pdf.

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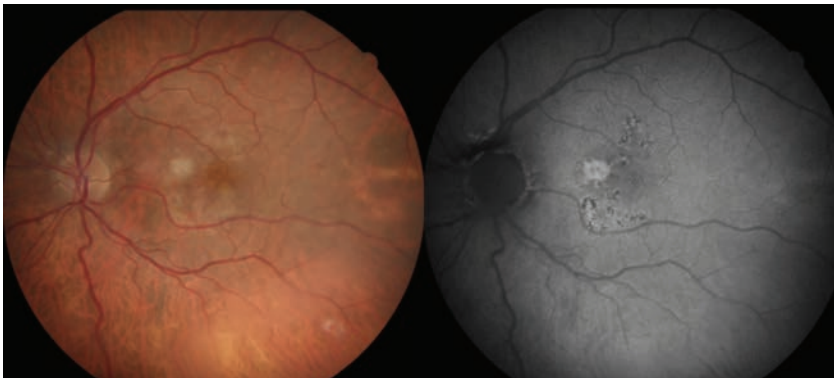
PEER-REVIEWED PRA ARTICLE

A Clinical Guide to FUNDUS AUTOFLUORESCENCE

Lipofuscin reveals a previously unseen world of potential disease indicators. Here's how to navigate it.

By Kevin Mercado, OD, and Amber Louprasong, OD

With its relative ease of use, noninvasive technique and ability to aid in the diagnosis and management of a variety of retinal disorders, fundus autofluorescence (FAF) has increasingly become a mainstream imaging modality. Although FAF technology has been referenced in medical research for more than 40 years, advances over the last decade have made it increasingly relevant as an ophthalmic imaging technique suitable for daily practice.[1] FAF provides a view of the disease processes from a metabolic perspective. Research has shown it to be effective in the early detection of retinal disease and the potential predictive makers for progression.[1] It has also helped us understand certain pathophysiological mechanisms. Although an excellent clinical tool, FAF can be used in conjunction with conventional fundus photography, optical coherence tomography (OCT), fluorescein angiography and other imaging modalities to provide an even more complete clinical picture.



Figs. 1a and 1b. At left, fundus photo shows early-stage dry AMD. At right, FAF image shows large soft drusen associated with increased fundus autofluorescence in a patchy FAF pattern. There are also more defined areas of hyper-AF corresponding to mixed drusen in a focal increased pattern superior and inferior to the fovea.

Lipofuscin

The retinal pigment epithelium (RPE) plays an important role in the normal functioning of the retina and is essential for vision. Lipofuscin, a byproduct of phagocytosed photoreceptor outer segments, is

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If this product is used for 10 days or longer, intraocular pressure should be monitored. The initial prescription and renewal of the medication order beyond 14 days should be made by a physician only after reexamination of the patient with the aid of magnification. Fungal infections of the cornea may develop with prolonged use of corticosteroids.

Ocular adverse reactions occurring in 5-15% of patients treated with loteprednol etabonate ophthalmic suspension (0.2%-0.5%) in clinical studies included abnormal vision/blurring, burning on instillation, chemosis, discharge, dry eyes, epiphora, foreign body sensation, itching, infection, and photophobia.

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

References: 1. ALREX [package insert]. Tampa, FL: Bausch & Lomb Incorporated; 2013. **2.** Dell SJ, Lowry GM, Northcutt JA, Howes J, Novack GD, Hart K. A randomized, double-masked, placebo-controlled parallel study of 0.2% loteprednol etabonate in patients with seasonal allergic conjunctivitis. *J Allergy Clin Immunol.* 1998;102(2):251-255. **3.** Shulman DG, Lothringer LL, Rubin JM, et al. A randomized, double-masked, placebo-controlled parallel study of loteprednol etabonate 0.2% in patients with seasonal allergic conjunctivitis. *Ophthalmology.* 1999;106(2):362-369.

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CONTRAINDICATIONS
ALREX, 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. ALREX is also contraindicated in individuals with known or suspected hypersensitivity to any of the ingredients of this preparation and to other corticosteroids.

WARNINGS
Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision, and in posterior subcapsular cataract formation. Steroids should be used with caution in the presence of glaucoma.

Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection.

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

PRECAUTIONS
General: For ophthalmic use only. The initial prescription and renewal of the medication order beyond 14 days 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.

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

If this product is used for 10 days or longer, intraocular pressure should be monitored.

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.

Information for Patients: 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 redness or itching becomes aggravated, the patient should be advised to consult a physician.

Patients should be advised not to wear a contact lens if their eye is red. ALREX should not be used to treat contact lens related irritation. The preservative in ALREX, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses **and whose eyes are not red,** should be instructed to wait at least ten minutes after instilling ALREX before they insert their contact lenses.

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, (1500 and 750 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender.

Pregnancy: Teratogenic effects: Pregnancy Category C. Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (85 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 (15 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 postimplantation 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 (15 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. ALREX Ophthalmic Suspension 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 ALREX is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients have not been established.

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

Ocular adverse reactions occurring in 5-15% of patients treated with loteprednol etabonate ophthalmic suspension (0.2% - 0.5%) in clinical studies included abnormal vision/blurring, burning on instillation, chemosis, discharge, dry eyes, epiphora, foreign body sensation, itching, injection, and photophobia. Other ocular adverse reactions occurring in less than 5% of patients include conjunctivitis, corneal abnormalities, eyelid erythema, keratoconjunctivitis, ocular irritation/pain/discomfort, papillae, and uveitis. Some of these events were similar to the underlying ocular disease being studied.

Non-ocular adverse reactions occurred in less than 15% of patients. These include headache, rhinitis and pharyngitis.

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. Among the smaller group of patients who were studied with ALREX, the incidence of clinically significant increases in IOP (≥10 mm Hg) was 1% (1/133) with ALREX and 1% (1/135) with placebo.

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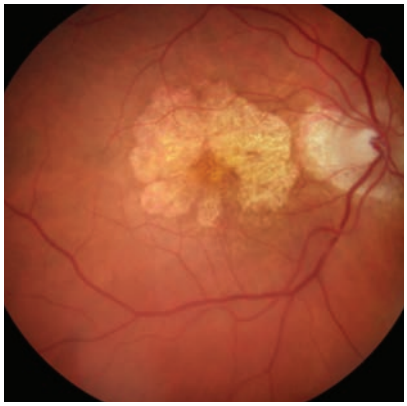
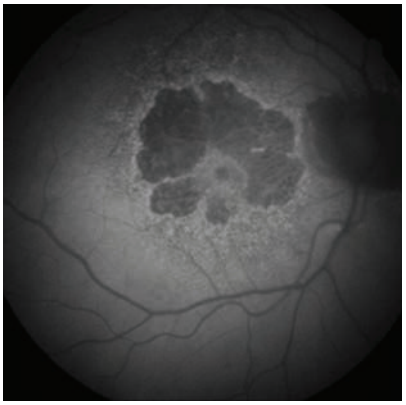
found in a number of aging and diseased tissues, including the RPE. Lipofuscin will accumulate in the RPE when its ability to break down pigmented outer segments of photoreceptors is compromised. While this occurs naturally with age, excess accumulation is considered pathologic and may occur with RPE cell dysfunction or abnormal metabolic load on the RPE.[1] This makes the presence of lipofuscin a potential early indicator of degenerative retinal diseases. FAF allows us to take advantage of the autofluorescent properties of lipofuscin when exposed to short-to-medium-wavelengths of light (short 500nm to medium 750nm).[1-3,5,9] This lets us visualize RPE changes that are affected by lipofuscin.





Figs. 2a and 2b. At left, fundus photograph of late dry AMD and geographic atrophy. At right, FAF image shows the intensity of the atrophic areas surrounding the macular area, markedly decreased due to loss of lipofuscin and clearly delineated. While the surrounding retina appears mostly normal on the fundus photograph, FAF gives additional information by showing increased FAF intensity in a fine granular pattern extending beyond the geographic atrophy (GA) junctional zone. This pattern has been shown to increase the likelihood of GA progression.

FAF Images

The images produced are similar in appearance to those of fluorescein angiography images; however, unlike FA, FAF is not dependent on circulation as is fluorescein angiography. The intensity of the FAF signal corresponds to the accumulation or absence/reduction of lipofuscin and is compared to the surrounding retina. In a healthy retina, the posterior pole will have a diffuse gray appearance—a mildly hyper-fluorescent signal due to normal levels of lipofuscin in RPE cells. The optic nerve head is dark due to the absence of RPE, retinal vessels appear dark due to signal absorption from blood and the fovea appears dark due to absorption from macular luteal pigment.[3,9] By evaluating this pattern of lipofuscin distribution in the RPE, we can begin to visualize disease processes. Any deviation from normal results should be evaluated, and that cause determined.

When interpreting FAF images, the image quality and amount of FAF intensity from one patient to the next may vary due to several variables, such as age, media opacity and pupil size. The type of FAF device used—scanning laser ophthalmoscope (SLO) vs. fundus camera-based system—will also affect the image.[2,3] These factors are important to recognize, as FAF intensity is relative to variables such as these, as well as each individual patient. Absolute FAF values therefore cannot be precisely determined between patients.

FAF in Disease

Reduced FAF signals may be indicative of RPE atrophy, cell loss or death, hyper-pigmentation, subretinal fluid or new hemorrhages. Focal areas of increased FAF signal may be caused by drusen, older hemorrhages and excess concentrations of lipofuscin in the RPE. Increased levels of autofluorescence typically indicates abnormal metabolism. Changes that are visible with FAF are often not visible clinically or with other imaging techniques such as conventional fundus photography. This means that not only can FAF be used to highlight areas of diseased retina and abnormalities that may otherwise not be visible, but it may serve as a marker of disease progression, with potential prognostic value.[3,4,6]

Early Macular Degeneration

In its early stages, dry age-related macular degeneration (AMD) may show a wide variety of FAF patterns that are not always visible funduscopically. These patterns were categorized into eight phenotypes and described by the Fundus Autofluorescence in Age-related Macular Degeneration (FAM) study group.[3]

As AMD progresses, changes may be seen with FAF imaging before the appearance of visible lesions. For example, images demonstrating several defined areas of markedly increased FAF (focal FAF pattern) have been shown to represent a higher risk of progression to geographic atrophy.[3] Images with multiple large (>200µm in diameter) areas of increased FAF

that correspond to soft drusen and hyperplasia (patchy pattern) have been found to represent a higher risk of conversion to wet AMD.[3] (*Figures 1a and 1b*).

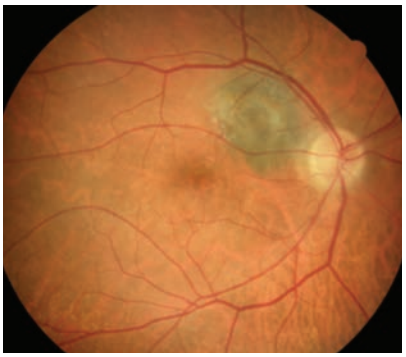
Late Macular Degeneration

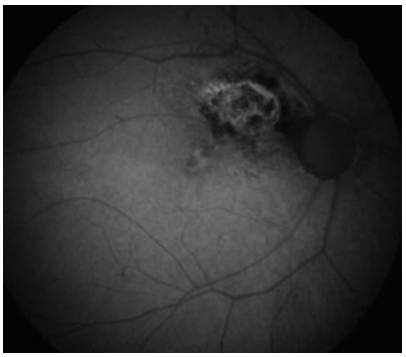
We know that geographic atrophy (GA) is an important cause of severe, irreversible vision loss in patients with AMD. When lipofuscin is absent due to RPE cell loss and death, as in the case with GA, the FAF signal drastically decreases. FAF allows a more dramatic visualization of areas of atrophy than standard fundus photography. It is the area surrounding the GA, described as the junctional zone, that may serve to help predict risk of progression.

The FAM study group found that, in general, the more hyperautofluorescent the FAF signal is at the junctional zone, the more likely there will be GA progression. This is especially true when the autofluorescent signal expands in a diffuse pattern beyond the area of GA (*Figures 2a and 2b*). Interestingly, the FAF patterns described by the FAM study group demonstrate a stronger impact on GA progression than other risk factors—including age, history of smoking, atrophy size at baseline and systemic disease.[3,6] In terms of the patient, being able to identify risk of future vision loss is functionally relevant for procurring low vision aids and maintaining quality of life.

Choroidal Nevi and Melanoma

FAF also helps to differentiate acute from chronic intraocular lesions. Benign lesions, such as choroidal nevi, are found in up to 7% of white patients, but at times may be difficult to distinguish from a small melanoma.[9,10] Choroidal melanoma represents a serious and even life-threatening malignancy with approximately 2,000 new cases in the United States each year.[9]





Figs. 3a and 3b. At left, fundus photo shows a choroidal nevus with high risk for conversion. At right, orange pigment provides an intense signal and is easily visible in this FAF image. Areas of overlying RPE atrophy with low intensity are also evident.

When evaluating a choroidal lesion with FAF, be mindful of both the intrinsic autofluorescence of the lesion as well as its extrinsic properties, specifically overlying RPE. Generally speaking, pigmented choroidal nevi and melanoma have relatively low intrinsic autofluorescence, while the overlying RPE is often more telling, considering RPE hyperplasia and atrophy have a much more dramatic hypo-autofluorescent appearance and are more indicative of a choroidal nevus.

Drusen, also suggestive of chronic nevi, will typically display a slightly increased FAF pattern. In contrast, melanoma and high-risk nevi often demonstrate a marked amount of intense extrinsic hyperautofluorescence due to orange pigment and to a lesser extent, subretinal fluid.[8,9]

FAF imaging is ideal for detecting orange pigment, which is sometimes subtle or near-invisible funduscopically, especially in deep or amelanotic lesions (*Figures 3a and 3b*).[9]

Retinal Telangiectasia

Investigators have found that Type 2 idiopathic macular telangiectasia (IMT) can be seen using FAF via changes in the fovea, even in the early stages of the disease.[11] While the exact mechanism of IMT itself is not completely understood, the FAF changes are likely the result of pathological changes in macular pigment distribution or changes in the RPE lipofuscin and melanin composition. In early IMT, FAF abnormalities at the macula may precede changes shown by other imaging modalities. In later stages of IMT, low areas of FAF surrounded by increased FAF may signal signs of likely progression.[11]

Retinal Dystrophies

Pattern dystrophies, Best's disease, Stargardt's disease and other vitelliform dystrophies are all conditions that may be better visualized with the aid of FAF imaging.[12,13] Hyperautofluorescence will vary depending on lipofuscin concentration, while areas of RPE atrophy (which lack lipofuscin) are easy to identify because of their low intensity. Appearance of FAF is limited in its ability to help differentiate between various forms of retinal dystrophies, especially in late stages, but is helpful for monitoring retinal changes for follow-up in addition to with genetic testing and counseling.[12]

In retinitis pigmentosa, FAF may

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show a hyper-autofluorescence parafoveal ring that will tend to constrict over time, as will a patient's visual function.[14,15] With the advent of widefield FAF images, researchers have found that peripheral areas of hypo-autofluorescence correlate with visual field loss and progression indicated by surrounding hyper-autofluorescence similar to GA in AMD.[15]

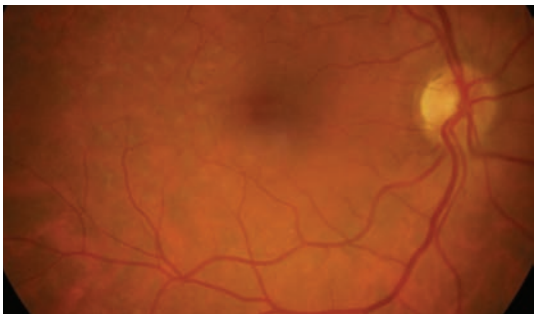
Other FAF Applications

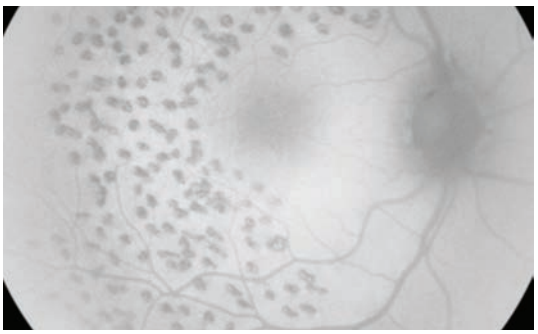
In addition to traditional 10-2 threshold visual field testing, FAF is among the objective tools that may be used as part of the screening process for hydroxychloroquine maculopathy.

A ring of hyper-autofluorescence along with parafoveal dark areas will be visible. This is especially true in moderate and later stages of hydroxychloroquine maculopathy.[16] Researchers speculate as to the sensitivity of FAF in early stages of hydroxychloroquine toxicity for some patients, especially compared with spectral domain OCT and mfERG, and it is always important to check for toxic hydroxychloroquine dosing during your screening.[7]

Postoperative laser sites are often easier to visualize when using FAF. The extent of subtle focal laser or PRP atrophy is well defined using FAF and may help confirm treatment areas. Consider that not all patients are aware of their exact past ocular surgical histories, including what type of surgery or which eye, and FAF may provide a dramatic picture of a laser-treated retina (*Figure 4a and 4b*).

FAF is a great clinical tool for detecting and monitoring a variety of retinal conditions and has become a readily available imaging technique. In some cases, it's not the availability of FAF limiting its use, but some uncertainty in its interpretation, as demonstrated in a recent hydroxychloroquone screening study.[7]





Figs. 4a and 4b. Above, this fundus photo of a patient who has undergone past panretinal photocoagulation shows subtle hints of previous laser treatment temporal to the macular area. Below, the FAF image clearly defines the extent of the previous surgery. Note that on FAF, there is a diffuse hyper-AF signal to the image due to media opacity (cataract).

FAF is more than just another picture. It provides a metabolic map of the retina, giving us detailed insight to disease processes that may otherwise remain unseen. It allows us to manage a variety of conditions at earlier stages than ever before and gives us the chance to improve long-term visual outcomes for our patients. ■

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Guidelines on Glaucoma PROGRESSION

Monitoring patients is key to providing appropriate treatment. **By Michael Cymbor, OD**

As optometrists, much of our energy is devoted to the diagnosis of glaucoma, and rightfully so. It is, after all, the second leading cause of blindness in the United States and the world.[1] Glaucoma is a degenerative optic neuropathy characterized by a progressive loss of retinal ganglion cells, leading to a loss of visual function. In today's optometric environment, with so many available treatment options, we are charged with doing more than simply prescribing a prostaglandin and wishing the patient well. The detection and monitoring of glaucomatous progression is critical both to our patient's health and our discipline.

In 2004, our practice diagnosed a 61-year-old white male patient with ocular hypertension, based on intraocular pressure (IOP) of 23mm Hg OD and 25mm Hg OS. From 2004 to 2011, his peak IOP was measured at 25mm Hg OD and 29mm Hg OS, while his mean IOP was 19.8mm Hg OD and 21.6mm Hg OS. His pachymetry is 502µm OD and 518µm OS. From 2004 to 2011, his visual fields were clean and his cup-to-disc ratios were 0.6/0.7 OD and 0.5/0.5 OS (*Figures 1 and 2*). His nerve fiber layer OCT showed some possible thinning around 8:00 in the right eye but was stable from 2010 to 2011, while his ganglion cell complex (GCC) showed an inferior defect in the right eye (*Figure 3*). Retinal tomography started to show infratemporal changes in 2011. In 2012, his glaucoma hemifield test results in the right eye went from "borderline" to "outside normal limits," and his visual field GPA1 (event analysis) went from "no progression

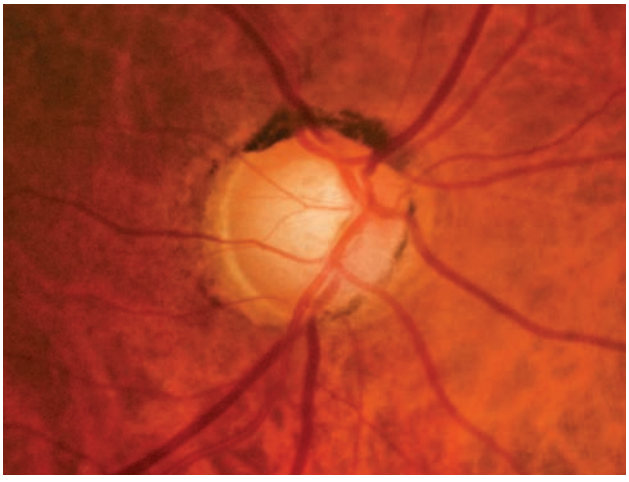

Fig. 1. Using modern analysis technologies, we were able to trace this patient's progression with enough time to delay development. Two decades ago, optometrists evaluated glaucomatous progression primarily using visual field tests.

Release Date: September 2015
Expiration Date: September 1, 2018
Goal Statement: A vital aspect of caring for patients with glaucoma is tracking the disease's development. This course reviews how to perform a comprehensive glaucoma evaluation, including IOP measurements and visual fields tests, as well as how to respond to glaucomatous changes.
Faculty/Editorial Board: Michael Cymbor, OD

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Disclosure Statement: Dr. Cymbor is on the speaker's bureau for Optovue and is an advisor for Eye IC.

detected" to "possible progression" (*Figure 4*). In 2012, we started treatment with Lumigan 0.01% OU.

Determining Progression

Two decades ago, optometrists were evaluating glaucomatous progression primarily using visual field tests and, to a lesser extent, photography. One decade ago, we were employing a combination of visual fields and structural testing of the optic nerve head/retinal nerve fiber layer (RNFL), such as confocal laser scanning tomography (HRT), scanning laser polarimetry (GDx) or optical coherence tomography (OCT). How are we evaluating risk factors and determining glaucomatous progression today? This article will highlight current concepts in managing glaucomatous progression.

Pressure Points

IOP remains the most prominent and consistent risk factor for glaucomatous progression. This was confirmed in the Ocular Hypertensive Treatment Study (OHTS), Early Manifest Glaucoma Trial (EMGT), Advanced Glaucoma Intervention Study (AGIS) and the Collaborative Normal Tension Glaucoma Study (CNTG), which all show lowering IOP delays or prevents glaucomatous progression or conversion.[2-5]

Peak IOP might be the most important factor in determining progression, but mean IOP and IOP fluctuation are also important.[6] However, there is one confounding factor—investigators estimate that 67% of peak IOP occurs outside of typical office hours.[7] This can be quite confusing when dealing with a patient who is progressing more rapidly than expected.

Checking Pressure

The most common method of obtaining IOP is Goldmann tonometry. Goldmann is relatively inexpensive and straightforward to perform.

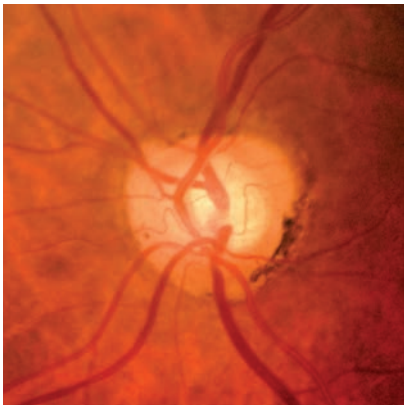

Fig. 2. The fact that this 61-year-old patient progressed within one year in the right eye shows us he is at significant risk for future progression.

However, the inaccuracy and imprecision of the instrument are well documented.[8] For example, Goldmann tonometry may be inaccurate with thick or thin corneas, patients who've undergone corneal surgeries, corneal edema or high corneal astigmatism.[9] There may be inconsistent readings between doctors and between technicians. Eyelid squeezing and breath holding may influence the readings.[9] Despite these challenges, Goldmann tonometry is universally accepted as the primary method to gather IOP in glaucoma management because of cost, convenience and general practice patterns.

Noncontact tonometry (NCT) is another method of obtaining IOP. The NCT creates a burst of air, and the tonometer measures the cornea's resistance. Generally, researchers have found good agreement between NCT and Goldmann.[10] However, the relationship between increasing and decreasing corneal thickness and increasing and decreasing IOP can be magnified with NCT.[11]

The Tono-Pen, a descendant of the Mackay-Marg tonometer, is a handheld tonometer option. However, early models were limited by inadequate sampling rates. For instance, a study comparing Goldmann with the Tono-Pen XL (Reichert) concluded the Tono-Pen XL should not be used as a substitute for Goldmann in the management of patients with glaucoma or ocular hypertension.[12] These sampling rates have improved for the newest device of this type called the Avia (Reichert); however, another study questions whether the Tono-Pen Avia can be used interchangeably with Goldmann in monitoring glaucoma patients.[13]

The Pascal tonometer (Zeimer) uses a high sensitivity pressure transducer that measures the countervail of the cornea when one gram of pressure is applied. Although the Pascal is less studied than some of the other methods, investigators think it may reduce operator bias by directly giving a pressure number.[14] It also can report the ocular pulse amplitude, which investigators suspect may be lower in glaucoma patients compared with non-glaucoma patients.[14]

The iCare tonometer (iCare) is based on a rebound measuring principle, in which a light weight probe is used to make momentary contact with the cornea. The instrument measures probe deceleration and contact time. The iCare is well tolerated and its accuracy is thought to correlate well with Goldmann.[15]

Another method of obtaining IOP is the Ocular Response Analizer (ORA) (Reichert). The ORA uses a much longer pulse than NCT technology.[16] Investigators believe the ORA correlates well with Goldmann but is less influenced by corneal properties.[17]

Whatever method is chosen, IOP measurements for each patient should be consistent from visit to visit so as to minimize apples-to-oranges IOP comparisons.

According to a recent publication, no perfect tonometer exists and clinicians must choose which to use, balancing accuracy, precision, convenience and cost.[18]

The Future

The problem with IOP measurements is that they provide an extremely small number of data points by which we make vital treatment decisions. This should make all glaucoma clinicians feel somewhat ill-equipped.

Twenty-four hour IOP monitoring has the potential to significantly disrupt the way we manage glaucoma. In fact, at last year's Glaucoma 360 conference, Robert Weinreb, MD, said "the most disruptive event for glaucoma practitioners, and the most transformative event for glaucoma patients within the next five years, is 24-hour IOP monitoring."[19]

One recent contact lens sensor 24-hour IOP study found that contact lens sensor measurements may be useful for the detection of sleep-induced IOP changes.[20] We may soon see a parade of 24-hour studies elucidating what is happening with our patients' IOPs in those vast times between pressure checks.

Serial Photography

Serial optic disc and nerve fiber layer photography are established methods of monitoring glaucomatous structural progression and are arguably still considered the standard of care.[21] Optic nerve progression can include new or increased neuroretinal rim thinning, notching, excavation and nerve fiber layer defects.[22] Serial optic nerve photography can also detect optic nerve hemorrhages and B zone peripapillary atrophy changes, which are both known risk factors for optic nerve progression.[23,24] Serial photography can be used to evaluate optic nerve photos taken from different cameras so clinicians often have years of images.

But this method can be time consuming and may require a high degree of skill. Optic nerve and nerve fiber layer assessment can be qualitative and subjective. Intraobserver and interobserver agreement among glaucoma specialists is only slight to fair, and agreement among general ophthalmologists is poor.[25-29] It can be challenging to detect diffuse glaucomatous progression of the nerve fiber layer and neuroretinal rim employing photography.

At least one technology attempts to improve the process of using serial photography to detect glaucomatous progression—a technique called flicker comparison.[30,31] This involves taking two images, obtained at different times, and rapidly projecting them one atop the other. In the flickered images, unchanged areas appear stable, whereas changes in the optic nerve appear as movement. This concept is similar to the process of animation in that slight differences in sequential pictures give the illusion of movement when alternated or "flickered" at a certain rate. This is a commercially available, cloud-based software that can use any digital optic nerve photo. Flicker comparison has the potential to improve accuracy and may be more sensitive than standard serial photography at detecting disc hemorrhages, peripapillary progression and retinal blood vessel positional shifts.[32-35]

Other Imaging Technologies

Because of the weaknesses in serial photography mentioned above, there is a need for objective and quantitative imaging analysis. The two main methods today are HRT and OCT.

• ***HRT.*** Confocal laser scanning tomography can accurately detect glaucomatous structural changes to the optic nerve, often before field changes.[36-42] This technology is generally understood to have the most well-developed and most tested progression analysis among all optical imaging devices.[43]

Years of confocal progression research culminates in the ancillary OHTS study, which compared the rate of structural change in ocular hypertensive eyes that do or do not develop primary open-angle glaucoma.[44] This study is significant because the median length of follow-up was 11 years for non-POAG converting patients and 5.6 years for POAG converters. This represents the longest follow-up of the largest group of ocular hypertensive subjects using optical imaging instruments. Eye care specialists will not see anything close to those follow-up periods with Fourier-domain OCT technology for many years to come.

The study's authors made the following conclusions:[44]

(1) The rate of neuroretinal rim loss is five times faster in eyes in which POAG developed compared with eyes in which it did not.

(2) Eyes that developed POAG changed at a rate of 0.99%/year vs. 0.18% for eyes that did not.

(3) The fastest neuroretinal rim change is in the inferotemporal sector.

(4) There was a weak association with central corneal thickness (CCT) and the rate of neuroretinal rim loss.

This study verifies that measuring the rate of structural change using HRT technology provides useful information for the clinical management of ocular hypertensive patients.[44] It also clarifies something HRT users have long known: that glaucomatous structural change, as determined by HRT technology, often precedes visual field loss.[44]

Despite the impressive credentials, advancements in confocal tomography have slowed in favor of the time-domain (TD-OCT) technology.

• ***OCT.*** TD-OCT started promisingly, but weaknesses soon emerged,

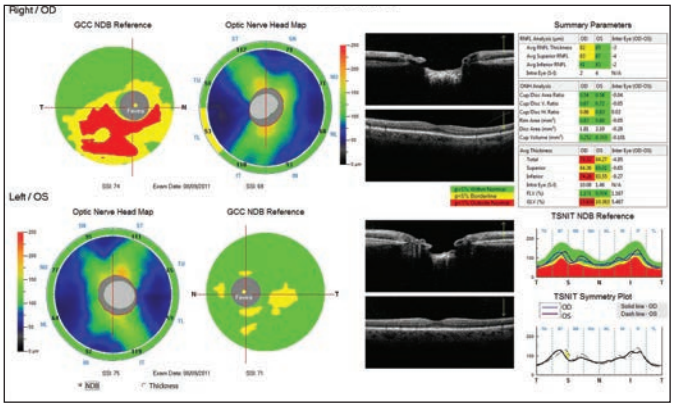

Fig. 3. This OCT shows our patient's nerve fiber layer displays thinning around 8:00 while his ganglion cell complex showed an inferior defect, both in the right eye.

including slow scanning speed, artifacts, limited axial resolution and inadequate reproducibility. In addition, TD-OCT RNFL progression was hampered by a lack of image registration with scan misalignment, significantly altering results.[45] All of these issues have been improved with spectral-domain OCT (SD-OCT).[46,47]

Four of the current commercially available SD-OCT devices in the United States are:

- The Spectralis (Heidelberg Engineering).
- The Cirrus (Carl Zeiss Meditec).
- The Avanti (Optovue).
- The 3D OCT-2000 (Topcon).

The Spectralis SD-OCT obtains OCT scans with a speed of 40,000 A-scans/second. The device has an axial resolution of 3.9µm, with a transverse resolution of 14µm and a scan depth of 1.9mm.[48] The Cirrus operates at 27,000 A-scans/second, has an axial resolution of 5µm, with a transverse resolution of 15µm and a scan depth of 3mm.[49] The Avanti SD-OCT scans at a speed of 70,000 A-scans/second, has an axial resolution of 5µm (3µm digital), with a transverse resolution of 15µm and a scan depth of 3mm.[50] The 3D OCT-2000 scans at a speed of 27,000 A-scans/second. The device has an axial resolution of 5µm to 6µm, with a transverse resolution of less than 20µm and a scan depth of 2.3mm.[51]

The principles of imaging acquisition are similar for each of these devices and involve the use of a scanning laser diode to provide high-resolution images of neuroretinal tissue through the detection of the echo optical signal measured by the spectral/Fourier domain technique.[52]

Previous studies demonstrated that measurements taken from these instruments show good clinical–histological correlations.[53-55]

SD-OCT is more sensitive than TD-OCT in detecting glaucomatous progression in the retinal nerve fiber layer.[56] The most common pattern of RNFL progression, a study shows, is widening of RNFL defects (85.7%), followed by development of new defects (17.9%) and deepening of RNFL defects (7.1%).[56] The inferotemporal meridian was the most common location where RNFL change was detected. Other studies have confirmed the accuracy of using SD-OCT to monitor changes in the RNFL.[57-60]

Doctors generally accept that glaucoma causes thinning to retinal tissue within the macular area and that macular scans are complementary to RNFL scans.[61-63] Some studies seem to indicate that macular scans in glaucoma may even detect damage prior to RNFL OCT and visual fields.[64,65] The practice of using the macular region to monitor glaucomatous progression is still emerging.

While SD-OCT is enjoying widespread acceptance, challenges in using the technology for the monitoring of glaucomatous progression remain, in that we are just beginning to see published studies on the validation of these statistical progression analyses. No evidence supports the use of one device over another in detection and management of glaucomatous progression.[66] Even though many clinicians have employed SD-OCT technology for years, SD-OCT research for the management of glaucoma progression is still in its infancy.

Visual Fields

The main goal in the management of glaucoma is to preserve visual function. The primary method of measuring visual function is the automated visual field. Automated visual fields enjoy widespread use and are found in the most eye care providers' offices.[67,68] Visual field limitations include being subjective, time-consuming and sometimes unreliable.

There are several ways to improve reliability, which will then help to more accurately assess progression.[43] These include:

(1) Explain to the patient the purpose of the test.

(2) The technician should be in close proximity to the patient to give advice and encouragement.

(3) If possible, a 1:1 technician to patient ratio should be kept.

(4) The technician should give feedback to the doctor about the patient's performance.

(5) The technician should explain to the patient that they are not expected to see all the lights.

Between 2001 and 2009, visual field usage decreased by 44%, while other forms of testing increased.[69] While eye care providers are performing less visual field testing now than in the past, some authorities

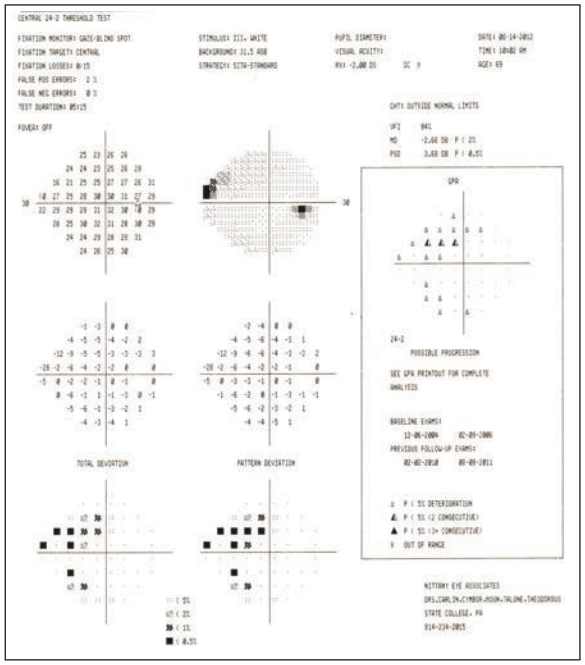

Fig. 4. Our patient's HRT3 showed infratemporal changes in 2011. In 2012, his glaucoma hemifield test results OD went from "borderline" to "outside normal limits," and this visual field GPA1 (event analysis) went from "no progression detected" to "possible progression." These technologies, combined, helped us determine a course of early treatment.

are recommending running more.[43] The World Glaucoma Association's 2011 Consensus statement recommends at least two visual fields in the first six months and at least two more in the next 18. This equals four fields in the first two years and possibly six if the patient is at risk for rapid progression. Rapid progression is defined as 2dB/year or greater.[43]

Many doctors employ the 24-2 testing pattern for monitoring glaucoma, while some prefer the 30-2. Both testing strategies have test points that are six degrees apart. The OHTS found that 12% of the patients that reached a visual field endpoint on the 30-2 strategy would not have been found on the 24-2 testing strategy.[70] Conversely, one study found more glaucomatous damage on the 10-2 test than on the 30-2 test in approximately 10% of their patients with suspected or early glaucoma.[71] This is because there are only four central points tested in both the 30-2 and 24-2. Early glaucomatous damage is easily missed with these two testing strategies. Also, some macular ganglion cells may be preserved until later in the disease process.[72]

Arcuate defects of the RNFL produce a range of glaucomatous VF defects. Sometimes these defects are best identified on the 30-2 test, some on the 24-2 and some on 10-2. Each of these tests can miss damage that others pick up. Clearly, eye care practitioners are looking for a better testing strategy that more often correctly identifies glaucomatous visual field defects.

In the past, the process of finding visual field glaucomatous progression was limited to serial visual field analysis. This process is cumbersome, and the agreement between glaucoma specialists is poor.[73] Better methods of determining glaucomatous visual progression are crucial, as many of our patients will experience progression if given enough time. In the EMGT, 59% of well controlled glaucoma patients progressed.[74] Fortunately, we now have visual field software analysis to help sift through numerous visual fields, including: glaucoma progression analysis (GPA1) and visual field index (GPA2 or VFI) from Carl Zeiss Meditec and the Octopus (Haag-Streit) cluster trend analysis with event analysis (CTA) and corrected cluster trend analysis (CCTA).

Visual field progression software comes in event analysis and trend analysis. After establishing a baseline, event analysis looks at every visual field to see if a reduction is more than just variability on a point-by-point basis. Event analysis attempts to answer the question, "Did the field progress or did it not?" Event-based progression analysis was used in the EMGT, the AGIS and the Collaborative Initial Glaucoma Treatment Study (CIGTS).[74-76] GPA1 uses event-based criteria established by the EMGT, in which a depression of three of the same points on two consecutive fields will be marked as "possible progression" and "likely progression" if those same points are on three consecutive fields. The Octopus CTA also uses event analysis. Trend analysis converts the field to a linear regression graphical representation, which may better allow clinicians to view the rate of change over time. If the patient has fields in a two-year period, the software will also predict future progression. The rate of field change over time may be expressed in dB/year. GPA2 uses center weighted points and is less influenced by media opacities such as cataracts.[77] In addition to GPA2, the Octopus CCTA uses trend analysis.

Short-wavelength perimetry (SWAP) uses a blue stimulus on a yellow background. Some have reported the detection of visual field defects prior to detection with standard automated perimetry (SAP).[78,79] Numerous subsequent studies have disproved this idea, and SWAP is now considered less valuable in the detection and monitoring of glaucomatous visual field progression.[43]

Remember to discard and repeat unreliable visual fields, as they may potentially obfuscate true glaucomatous progression and delay more aggressive management. It may also be a good idea to reset field baselines whenever treatment is changed.

At this time, no consensus exists on the best or most appropriate method of monitoring glaucomatous field progression.[43]

Greater sensitivity may be possible using a visual evoked potential (VEP) test, which measures the objective functional response of the visual pathway and may help identify early glaucomatous defects.[80] Also, research emphasizes the importance of measuring CCT.[2] Today, 69% of ODs employ this technology.[67]

Our Patient

From 2012 to 2013, his peak IOP was 21mm Hg OU, and his mean IOP was 15.9mm Hg OD and 16mm Hg OS. His treated mean IOP exhibited a 20% reduction OD and 26% OS. His visual fields continued to progress in his right eye, as seen with GPA2 or visual field index (VFI), but remained stable in the left eye. The fact that he progressed within a year in the right eye, even with a 20% IOP reduction, shows he is at significant risk for future progression. One of the highest risks for future progression is past progression.[81]

In 2013, we added Combigan (Allergan) BID and between then and now, his peak IOP has been 13mm Hg OD and his mean IOP 11.4mm Hg OD. His left eye's peak IOP is 13mm Hg and mean is 12.5mm Hg. His mean IOP from baseline experienced a 42% reduction in both eyes.

His visual fields and all structural testing are stable.

Fortunately, his visual acuity remains 20/20 and he remains asymptomatic. We are sending him for SLT in the near future.

We will continue to watch him closely and will consider a trabeculectomy in the right eye with evidence of further progression.

Never before have we had such an array of glaucoma diagnostic tools at our fingertips. It is incumbent upon us to stay abreast of advances in technology to provide the best possible care for our patients. ■

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1. Glaucoma is the second leading cause of blindness in:
a. The US. c. Tanzania.
b. The World. d. a and b.

2. The most prominent and consistent risk factor for glaucomatous progression is:
a. Age. c. IOP.
b. Cell count. d. Sleep apnea.

3. The following studies show lowering IOP delays or prevents glaucomatous progression:
a. CNTG. c. OHTS.
b. EMGT. d. All of the above.

4. What percentage of peak IOPs occur outside of normal office hours?
a. 25%. c. 75%.
b. 67%. d. 99%.

5. Which type of tonometry is able to measure ocular pulse amplitude?
a. Pascal. c. Tono-Pen.
b. Goldmann. d. iCare.

6. Which method of taking IOP requires anesthetic?
a. ORA. c. NCT.
b. Tono-Pen. d. iCare.

7. A contact lens sensor can give us:
a. Corneal thickness.
b. Aqueous flow.
c. 24-hour IOP measurements.
d. Visual evoked potential.

8. Glaucomatous optic nerve progression includes everything except:
a. Nerve fiber layer defects.
b. Reverse laminar dots.
c. Neuroretinal rim thinning.
d. Neuroretinal rim excavation.

9. With flicker technology, change appears as:
a. Polarization.
b. Movement.
c. The Troxler effect.
d. Reduced standard deviation.

10. Which has the most well developed and tested glaucomatous progression analysis?
a. HRT. c. Fourier-domain OCT.
b. Time-domain OCT. d. GDx.

11. The fastest neuroretinal rim change in the confocal laser scanning portion of the OHTS occurred in which sector?
a. Supratemporal. c. Nasal.
b. Infratemporal. d. Temporal.

12. Problems with time-domain OCT include

OSC QUIZ

everything except:
a. Unnecessarily fast scanning speed.
b. Artifacts.
c. Reproducibilty.
d. Limited axial resolution.

13. Which instrument shows good clinical-histological correlations?
a. Avanti. c. Spectralis.
b. Cirrus. d. All of the above.

14. The most common pattern of RNFL progression as determined by OCT is:
a. Deepening of RNFL defect.
b. Widening of RNFL defect.
c. Eruption of defect.
d. None of the above.

15. Scanning tissue within the macular area as it relates to glaucomatous progression is:
a. Complementary to RNFL scans.
b. A complete waste of time.
c. Controversial.
d. Bizarre.

16. Which technology is readily available in the offices of most eye care providers?
a. ORA.
b. Visual evoked potential instrument.
c. Pascal.
d. Visual fields.

17. Between 2001 and 2009:
a. Visual field usage increased.
b. Visual field usage decreased.
c. Visual field usage stayed the same.
d. None of the above.

18. The 2011 Consensus statement on glaucomatous progression recommends at least:
a. Two visual fields in two years.
b. Three visual fields in two years.
c. Four visual fields in two years.
d. Eight visual fields in two years.

19. How many central points are tested in the 24-2 and 30-2 strategy?
a. Two. c. Four.
b. Three. d. Five.

20. Visual field progression analysis software comes in:
a. Event. c. Circadian.
b. Trend. d. a and b.

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

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VISUAL AURA AND SCOTOMAS: What Do They Indicate?

Tracing these anomalous phenomena to their physiological origins can help you determine the appropriate level of care. **By George T. Banyas, OD**

Visual aura represent a type of neurologic deficit familiar to any eye care practitioner. Although classically preceding migraine or seizure, an aura, simply defined, is a symptom, not a medical condition unto itself. Likewise, terms such as *visual scotomas*, *amaurosis fugax* or *transient visual obscurations* also represent a disturbance of vision; however, they do not classically precede migrainous headache or cortical seizure activity and are associated with other types of pathology. Nonetheless, each term, when used in the right circumstance, may define remarkably similar visual deficits in one or both eyes.

A substantial list of differentials must be considered when a patient describes such visual disturbances, some associated with significant morbidity. Because of this, the complaint of visual aura or scotoma requires a comprehensive evaluation and should not simply be assumed to be migrainous (a diagnosis of exclusion). An understanding of the different types of aura and scotomas and how they present allows eye care practitioners to differentiate causes and order testing appropriately for potentially very different pathologies.

Presentation and Pathogenesis

Visual auras or scotomas are not blur. A visual aura is a transient or longstanding visual perceptual disturbance experienced with migraine or seizure that may originate from the retina or the occipital cortex. Visual changes described by patients are often referred to as blur, a word abused by patients as frequently as the word "dizzy." Blur has different connotative meanings to patients. Aura can

With scintillating, or fortification, scotomas, the central scotoma is bordered by a crescent of shimmering zigzags.

be defined as either positive (seeing something that is *not* there) or negative (not seeing something that *is* there). Furthermore, an actual image may be adulterated (appears larger, perseverates, etc.). Visual auras may be transient (e.g., a few seconds) or longstanding (perhaps for months) and, importantly, they may be accompanied by headaches or other types of aura such as vertigo, numbness, tingling or aphasia.

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The definition of visual scotoma is similar to that of visual aura. The differentials for scotoma likewise include migraine and seizure, but the term is more appropriately linked to ischemia, retinal degenerations and inflammations, paraneoplastic syndromes and other neurologic disorders. For visual scotomas, the primary pathogenesis may occur at the level of the receptors, retinal arterial tree, short posterior ciliary arteries, ophthalmic artery, optic nerve, carotid artery, vertebrobasilar artery or cerebral hemisphere.

Migrainous types of aura actually involve no detectable tissue pathology as well as little or no expectation of permanent deficit; however, migraine with aura has demonstrated increased risk of stroke.[1] Ischemic causes, provided the ischemic threshold is not significantly surpassed, enjoy total recovery. Other causes of visual scotoma may be self-limited or require significant intervention to prevent further morbidity or mortality.

Retinal Causes

Aura that originates in the retina will present solely as an unformed scotoma or visual defect that is either positive or negative. At the retinal level, formed images are not possible.[2] Although technically aura may include macropsia or micropsia, at the level of the retina this would occur with specific and typically demonstrable changes such as macular edema or cellophane maculopathy, and it would not be transient in nature.[3] Transient causes of micropsia or macropsia otherwise occur cortically.

Aura at the retinal level is likely to be unilateral in presentation, but this can be difficult to elucidate from the patient's history. Unilaterality is practically essential to attribute aura to the retina. On average, retinal aura lasts up to one hour and is most commonly embolic and rarely migrainous.

A negative visual aura deemed secondary to embolization is commonly referred to as amaurosis fugax. The Amaurosis Fugax Study Group has defined five distinct classes of transient monocular blindness based on their supposed cause: embolic, hemodynamic, ocular, neurologic and idiopathic.[4] The absence of vision may or may not progress across the visual field. Retinal migraine may result in the same type of visual deficit (negative aura); however, positive scotoma or blindness is also possible. Note that retinal migraines are often, but not always, associated with headache on the same side as the visual deficit within an hour. Embolic events also may or may not be associated with headache.

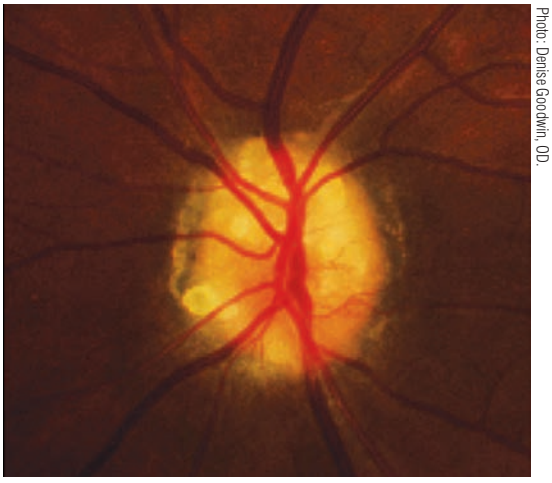


Photo: Denise Goodwin, OD.

Optic nerve drusen visible on the surface of the nerve in a 32-year-old female.

Retinal ischemic events are more likely in older persons with a history of cardiovascular disease/hypertension.[5] To some extent, coagulopathies or hyperviscosity syndromes may also be complicit and require consideration if a source of emboli is not identified.[6] Younger patients with no history of cardiovascular disease are more likely suffering from migraine. Because monocular transient vision loss localized to the retina (classically referred to as amaurosis fugax) may have many causes, meticulous case history is important. It is critical that patients and their internist be counseled by the eye care provider regarding appropriate testing to assist in diagnosis from a large potential differential. Again, note that migraine is a diagnosis of exclusion.

Non-migrainous scotomas associated with retina may also occur at the level of the photoreceptors/RPE. The retina in a state of rest is depolarized. The state of depolarization requires energy. RPE disease in particular may interfere with energy production, which may result in localized areas of constant hyperpolarization of the affected rods and cones. As a result, a constant twinkling or sparkling scotoma may occur and can last for months or years.[7] Positive continuous sparkling scotomas have been reported with conditions such as cancer-associated retinopathy, retinitis pigmentosa, or other retinal degenerations/inflammations such as multiple evanescent white dot syndrome or idiopathic blind spot enlargement.[8-10]

Often, retinal disorders will reveal themselves ophthalmoscopically; however, definitive dysfunction at the level of the RPE/photoreceptors may require multifocal ERG testing because fundus appearance as well as fluorescein angiography may be normal.

Cancer-associated retinopathy (CAR) may result in the perception of swirling clouds of smoke and occasional dim flashes of light.[11] Ophthalmoscopically, the fundus appearance early on is normal, but ERG testing may reveal significant dysfunction of rods and cones.

CAR is a paraneoplastic syndrome in which self-antibodies are directed toward the neoplasm but also attack specific sites in the retina, eventually resulting in arteriole attenuation, RPE mottling and disc pallor. It is most commonly associated with small-cell lung carcinoma, but has been described with other malignancies such as breast, gynecologic and prostate cancers. Chest or full-body CT scanning may be suggested in otherwise healthy patients to reveal tumor formation.

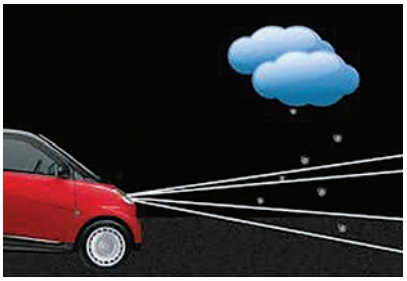
Patients with such disorders affecting cone function tend to see worse in bright light (hemeralopia), which often is clinically counterintuitive. Patient complaints of glare and (acquired) photophobia are common with cone disease. Rod disease, however, is associated with night vision difficulties (nyctalopia). Patients complaining about difficulties with both day and nighttime vision require multifocal ERG testing, which may reveal dysfunction even with the absence of visual aura. Photopsias are simply related to vitreoretinal interaction.

Optic Nerve Disease

At the level of the optic nerve, nonmigrainous vision loss may occur with disorders associated with central retinal artery or short posterior artery disease. In addition, optic nerve drusen or papilledema may also be associated with aura.

As a rule, optic nerve disease produces unformed scotomas that are negative and rarely positive.[2] Embolic disease blocking the central retinal artery or immediately at the bifurcation produces a negative scotoma that is either diffuse or altitudinal, respectively. The deficit may occur for seconds or up to 20 to 30 minutes. Rarely, the aura is positive and unlikely to march. Although tempting to categorize the attacks of amaurosis fugax as embolic, eye care providers must always keep in mind arteritis, where the pathogenesis may involve the ophthalmic artery or any of its branches.

Cardiac, carotid and other studies should be accompanied by a sed rate and C-reactive protein, particularly in patients over 50.[12] The intention is to rule out arteritis and the potential for arteritic ischemic optic neuropathy. Short



Positive scotomas are often described as snow falling through a beam of light.

posterior ciliary disease is typically inflammatory because these vessels are not anatomically susceptible to embolic disease.[13] Non-arteritic disease resulting in anterior ischemic optic neuropathy is believed to be secondary to arteriolarsclerotic disease or even possibly vascular dysregulation; however, the exact pathophysiology is unknown.[14,15]

Patients strongly suspected of temporal arteritis should immediately be placed on 60mg to 80mg of prednisone orally and sent to a lab for sed rate and C-reactive protein testing. Laboratory testing will not be affected for several days after initiation of prednisone therapy and will not reveal a false negative sed rate or C-reactive protein.[16] Prednisone therapy is provided with the intention of preventing arteritic ischemic optic neuropathy and immediate blindness, which is a real consequence of arteritis and may occur at any time. Arteritis is one of the more common causes of preventable vision loss if detected and should always be suspected in patients with transient central, altitudinal vision loss or both.

Congestion of the optic nerve head may also result in transient dimming of vision, particularly with postural changes. Optic nerve drusen are space-occupying lesions believed to result in some degree of disc congestion that may produce defined persistent field defects, negative scotomas or both that may vary considerably (10 to 15 seconds up to a few hours) and are often precipitated with postural changes.[17]

Disc drusen are often misdiagnosed as papilledema because their chairside evaluation often resembles papilledema. Clues to diagnosis of disc drusen include the presence of a spontaneous venous pulsation, anomalous branching of arterials (trifurcations), as well as peripapillary pigmentary changes. Hemorrhages may be present and are deeper and concentric. Autofluorescence of disc drusen may be demonstrated with blue filter photography. Buried drusen are best visualized with ultrasound to reveal hyaline bodies within the nerve head; non-contrast CT is also useful, as is OCT, to differentiate optic disc drusen from papilledema.[18]

Although possessing an entirely different pathogenesis, papilledema may also result in negative visual scotomas (seconds to hours) that may also occur with postural changes in one or both eyes, similar to disc drusen.[19]

Patients with genuine papilledema will classically present with bilateral swollen or elevated optic discs with indistinct disc margins, although unilateral papilledema with opening pressures as high as 350mm have been recorded, confounding the diagnosis.[20] Papilledema patients may be entirely asymptomatic, much like disc drusen patients, revealing no visual or significant field changes. Both papilledema and disc drusen patients may possess enlargement of the blind spot in both eyes, so this is not a differentiating characteristic.[21,22] Typically, patients with papilledema will demonstrate no spontaneous venous pulsation because of increased cerebrospinal pressure (typically > 250mm H_2O). When questioned, patients may be inclined to describe a recent history of headaches, particularly upon awakening.[23] Hemorrhages, if present, tend to be superficial in the nerve fiber layer, and venules may reveal passive congestion.

Papilledema is considered one of the true ocular emergencies and patients should be transported directly to the hospital if there is any suspicion. Other compartmental or "hydraulic" pathologies resulting in compression at the apex of the orbit may also result in postural visual obscurations; therefore, orbital disease should also be kept in mind.[24]

Vertebrobasilar Dysfunction

Transient ischemia affecting the vertebrobasilar circulation tends to produce bilateral negative scotomas, but occasionally a patient may present with a positive scotoma. The positive visual phenomenon is often described as "snow falling through the beam of a headlight." Like other transient ischemic attacks, the visual phenomenon may last for several minutes or longer and may be associated with other vertebrobasilar signs such as reduced awareness, diplopia, tinnitus, vertigo or dysarthria.[7]

Although migrainous episodes may be associated with the same deficits, like most migrainous syndromes, headache will follow in less than 60 minutes and any deficits will entirely resolve within 60 minutes. The visual deficit is still likely some type of scintillating or fortification scotoma, even if migrainous. Because of the similarity of symptoms and the potential for recovery with either mechanism, neurologic and cardiovascular evaluation is warranted with initial onset.

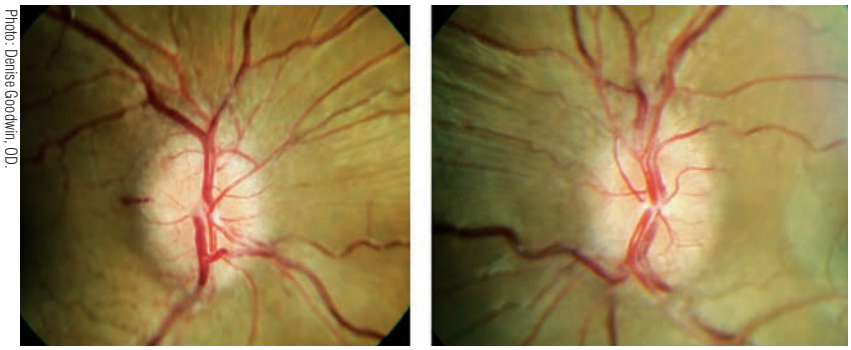


Photo: Denise Goodwin, OD.

Note the bilateral, symmetric appearance of papilledema.

Cerebral Hemisphere Involvement

Most typically, visual aura that occur as a result of cerebral disease are embolic, migrainous or seizure-related. Cortical aura will be bilateral and may last anywhere from seconds up to an hour. Depending on the location (parietal, temporal or occipital), the aura will occupy that portion of the visual field commensurate with the affected lobe.

Once again, ischemic events are likely to produce negative scotoma or aura and not march. Migrainous aura are more likely to be positive (often described as scintillating) and as a rule will march. These visual aura may or may not be accompanied by headache. In the case of ischemia, headache typically occurs during onset of the aura and the visual deficit is typically negative. With migraine, the aura is accompanied or followed within 60 minutes by headache.[24] If visual association areas (i.e., higher vision centers) are affected, potential phenomena that may occur include pallinopsia, macropsia, micropsia and formed visual hallucinations. Seizures affecting the occipital or sometimes the temporal lobe may also cause unformed visual hallucinations (colored circles), but may also result in formed visual hallucinations.[7] Seizure activity is likely to be associated with other seizure phenomena, such as eye deviation or rapid blinking.

Besides headache, other neurologic deficits may accompany visual aura. With migraine, waves of depression that spread out of the visual cortex will typically proceed anteriorly, affecting sensation or motor strength. Each new deficit in succession may last up to 60 minutes. Therefore, the initial visual aura may be followed by parietal involvement (numbness and tingling) then frontal involvement (motor weakness). This process would be expected to take up to 180 minutes (3x60). As is typical with migraine, the deficits are expected to march and headache may occur anytime within 60 minutes after the onset of the first aura. Classically, ischemic events result in no marching and the headache is most likely to occur at the onset of vision loss. One exception to conventional visual aura is the complication of migraine "persistent aura without infarction." In this instance, persistent visual aura—typically bilateral—will remain for at least one week and possibly months or years without evidence of ischemic injury.[25] Confirmation is determined with imaging studies, which remain negative. ■

Dr. Banyas practices in a corporate optometry setting, and provides eye care to over 15 nursing facilities in the greater Pittsburgh area.

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Case Report

The Swollen Optic Disc: *Is this an* EMERGENCY?

Idiopathic intracranial hypertension is a diagnosis of exclusion.

By David Lynne, OD, Erica Walker, OD, and Joseph Pizzimenti, OD

Obesity is on the rise in the United States and worldwide. In turn, the incidence of idiopathic intracranial hypertension (IIH), also known as pseudotumor cerebri (PTC), is also rising.[1] IIH initially presents as bilateral optic disc edema of unknown cause, and there are several etiologies, some of which are potentially deadly. Therefore, IIH is always a diagnosis of exclusion. This article reviews the presentation, diagnosis, testing and treatment of IIH.

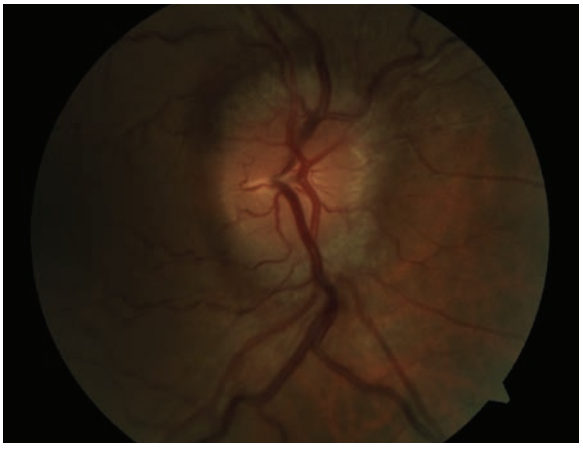

Fig. 1. At left, fundus photograph of a 52-year-old Hispanic male patient's right eye. Note 360-degree disc edema with swelling of the adjacent retinal nerve fiber layer. Details of the major retinal arterial branches leaving the disc superonasally and inferiorly are obscured by the edema.

Case Study

A 52-year-old Hispanic male presented with a chief complaint of episodic partial darkening of vision in his right eye that lasted five to 10 seconds, occurred up to 20 times a day and was associated with sudden lateral eye movement. He had first noticed this symptom eight months prior, but it had recently become more frequent. His medical history was remarkable for diabetes, hypertension and hyperlipidemia. His best-corrected visual acuity was 20/20-1 OD and 20/20-2 OS. Intraocular pressure measured 17mm Hg OD and 18mm Hg OS.

The right optic nerve exhibited 360-degree edema, which also involved the surrounding retinal nerve fiber layer (RNFL) (*Figure 1*). Subtle Paton's lines (circumferential retinochoroidal folds) were also visible temporal to the disc. Retinal arterial branches leaving the disc were somewhat obscured by the edema in the right eye. The left optic nerve showed indistinct margins superonasally and inferiorly with apparent elevation of the superior and inferior neuroretinal rim (*Figure 2*). No major vessel obscuration was seen in the left eye. No hemorrhages, exudates or cotton wool spots were present. The retinal venules were not dilated or tortuous. He denied symptoms of diplopia, scalp tenderness, jaw claudication, new neck pain or headache and did not have pulse-

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less, tender, enlarged or nodular temporal arteries upon palpation. Blood pressure measured 120/70mm Hg. His body mass index (BMI) was calculated to be 33.

This patient manifested a truly emergent ophthalmic condition: bilateral optic disc edema. Causes range from IIH to potentially fatal etiologies such as intracranial mass, dural venous sinus thrombosis and meningitis. We further investigated his bilateral disc edema with prompt additional testing, including baseline optical coherence tomography (OCT) (*Figures 3, 4a and 4b*), as well as same-day Humphrey visual field 30-2 SITA Fast testing.

The patient's visual fields showed a superior defect in each eye with more prominent defect in the right eye and an enlarged blind spot in the right eye. We noted the patient had significant brow ptosis with dermatochalasis greater in the right eye than the left, and despite taping the lids the visual fields (*Figures 5 and 6*) still showed a residual superior defect in each eye, which we attributed to the patient's very prominent overhanging brow. The enlarged blind spot present in the right eye could be correlated to the prominent disc edema noted in the right eye. Color vision with Ishihara pseudo-isochromatic plates was normal and cranial nerve testing did not reveal any other abnormalities.

History and Imaging

We thoroughly reviewed his medications and medical history and did not discover any potentially causative medication or systemic disease associated with intracranial hypertension. Even if you discover associated diseases or potentially causative medications, other life-threatening causes of bilateral disc edema must be ruled out.

We educated our patient about the emergent nature of bilateral disc edema and directly contacted the nearest emergency room with instructions to perform same-day MRI of the brain and optic nerves with contrast, plus magnetic resonance venography (MRV) of the brain.

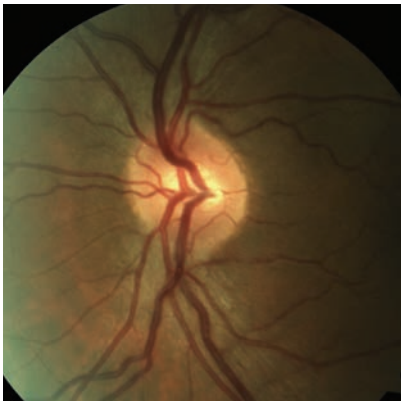

Fig 2. Fundus photo of the patient's left eye. Note the more subtle findings than in the right eye, with slightly indistinct superior and inferior disc margins.

We requested that, in the event the results of neuroimaging were unremarkable, lumbar puncture (LP) be performed with opening pressure recorded and cerebrospinal fluid (CSF) sent for biochemistry, microbiology and cytology. MRI and MRV were unremarkable. Opening pressure with LP was elevated and recorded at 26cm H^20. CSF biochemistry, microbiology and cytology were normal.

After exclusion of potentially life-threatening conditions and following normal examination by a general neurologist, a diagnosis of IIH was established.

IIH Presentation

The overall annual incidence of IIH is between one and two in 100,000 in the general population.[1,2] Among young, obese women, the estimated incidence is between 12 and 20 in 100,000.[1,2]

Differential Diagnosis of Bilateral Disc Edema

- Tumor
- Intracranial bleed
- Hydrocephalus
- Aquaductal stenosis
- Venous sinus thrombosis
- IIH

Symptoms of IIH are varied. The most common presenting symptom is headache, occurring in more than 90% of patients.[1,2] Also common are transient visual obscurations, described as monocular or binocular blurring lasting a few seconds, thought to represent momentary ischemia of the already compressed optic nerve microvasculature.[1] Many patients will complain of hearing something resembling a heartbeat in their ear, called pulsatile tinnitus. Other common complaints are photopsia and retrobulbar pain. Some patients may have diplopia related to an accompanying cranial nerve (CN) VI palsy (or rarely an associated CN III or IV palsy).[1-3] In some instances there may be no reported symptoms, with the only sign being the presence of disc swelling.[4] Our patient had some atypical characteristics for IIH being older and male, but studies have shown that both older patients and male patients with IIH had fewer complaints of headaches and were more likely to complain of visual disturbances.[5]

The hallmark sign of IIH is optic disc swelling (papilledema). Papilledema is caused by increased intracranial pressure.[1,4] Disc swelling of unclear cause should be labeled as disc edema and should not be categorized as papilledema until it is confirmed with an elevated intracranial pressure reading.[4] The disc swelling in papilledema is almost always bilateral, although it may be very asymmetric, making it appear

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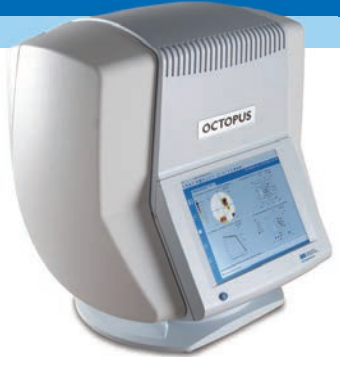

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unilateral.[1,4] In papilledema, the elevated CSF pressure disturbs the normal gradient between intraocular pressure and retrolaminar pressure, resulting in increased tissue pressure within the optic nerve. This interferes with axoplasmic flow and produces neuronal flow stasis.[6] Under this resistance the constipated axons swell, producing congestion at the level of the optic disc.[7] Along with the general congestion, resultant optic nerve ischemia, orbital venous stasis and collateral impact on optic pathways resulting from adjacent dilated ventricles may play a role in producing variable visual sequelae.[6,8]

Obesity has long been reported to be the most commonly associated risk factor in IIH.[9] Studies indicate between 74% and 94% of patients diagnosed with IIH are obese.[5] Generally, obesity is considered to be a body mass index (BMI) greater than 30. Many patients who are not necessarily obese may be categorized as overweight. Some patients with IIH actually have normal BMI. However, in a recent study of 407 adult patients with IIH, only 4% had normal BMI, and of those, more than a quarter were reportedly exposed to medications known to be associated with IIH.[5] Thus, care should be taken to uncover potential alternative causes of IIH in patients with normal BMI.[5]

Also, both obese and non-obese patients with a recent weight gain of 5% to 15% are at greater risk for developing IIH.[9]

The pathophysiology to explain the exact role of obesity in IIH remains unknown. Hypotheses have included metabolic and hormonal dysregulation. Also, obesity has been suggested to raise intra-abdominal pressure, in turn increasing pleural and cardiac filling pressure leading to increased intracranial venous pressure and thus elevated intracranial pressure.[9] Our patient was atypical in that he was older and male but typical for IIH in that his BMI was >30, which categorizes him as obese; however obesity alone does not make the diagnosis.

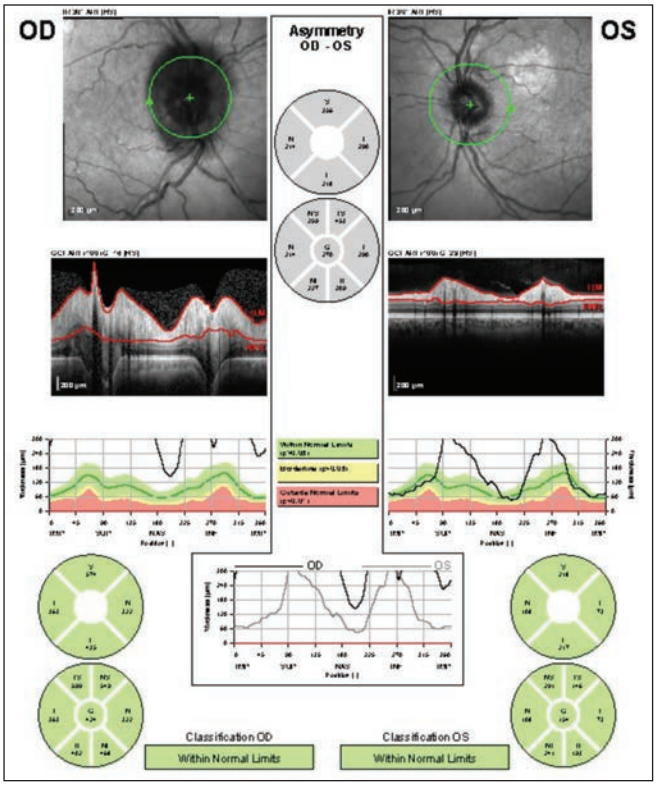


Fig. 3. Our patient's OCT RNFL results. Note the off-the-chart RNFL edema in the right eye and the pronounced edema superior and inferior in the left eye (compare to the subtle superior and inferior edema in the photograph of the same eye in Figure 2).

Diagnostic Criteria

The basis for the diagnosis of IIH is the modified Dandy criteria (*Table 1*).[1,10,11] The first step in diagnosing IIH is to obtain same-day MRI of the brain and optic nerves with contrast, plus MRV of the brain in order to rule out life-threatening causes of bilateral disc edema. Compressive tumors or hemorrhages can be visualized with MRI. Large space-occupying lesions can directly cause bilateral disc edema, while smaller brain tumors may

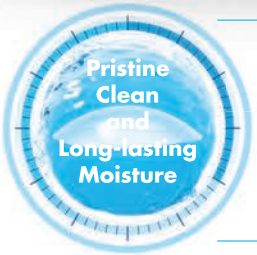
Table 1. Modified Dandy Criteria[1,10,11]

1. Presence of signs and symptoms of increased intracranial pressure.
2. Absence of localized findings on neurologic examination except those known to occur from increased intracranial pressure.
3. Absence of deformity, displacement or obstruction of the ventricular system and otherwise normal neurodiagnostic studies, except for evidence of increased cerebrospinal fluid pressure (>20cm H^20).
4. Abnormal neuroimaging except for empty sella turcica, optic nerve sheath with filled out CSF spaces and smooth-walled non flow-related venous sinus stenosis or collapse should lead to another diagnosis.
5. Awake and alert patient.
6. No other cause of increased intracranial pressure present.

Is It Time to RETHINK LENS CARE?

When multi-purpose solutions (MPS) first appeared on the market more than 20 years ago, they offered the promise of outstanding disinfection and cleaning efficacy, as well as greater lens comfort and compliance with the lens care regimen. Over the ensuing decades, however, it has become apparent that some MPS have fallen short of these lofty goals. Indeed, a study has shown that good compliance with MPS lens care regimens can be strikingly low—as low as 2%*—with 42% of patients "topping off" their used contact lens solution occasionally or every night.[1]

The Centers for Disease Control and Prevention have identified poor compliance with MPS regimens, including poor contact lens hygiene and infrequent storage case replacement, as a major risk factor for keratitis. Poor compliance puts a strain on the healthcare system, accounting for nearly 1 million doctor's office, clinic, and emergency room visits for keratitis each year, and $175 million in healthcare expenditures.[2] Additionally, in recent years the Food and Drug Administration (FDA) has shown that preservative uptake into some lens materials may compromise MPS disinfection.[3–5] Preservative uptake may result in additional concerns such as reduced biocompatibility as measured by increased corneal staining. Corneal staining is linked to discomfort—the number 1 cause of dropouts.[6–8] Contact lens dropouts result in a median future loss of $21,695 of lifetime revenue for each patient.[8]



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To manage these issues related to MPS use, eye care professionals have long turned to CLEAR CARE® peroxide solution, with its proprietary Triple Action Cleaning formula that gives patients unsurpassed disinfection efficacy and fresh, pristine clean lenses. Now, Alcon has found a way to take CLEAR CARE® to the next level with the introduction of CLEAR CARE® PLUS. This new peroxide solution combines the best of both worlds: the trusted benefits of CLEAR CARE® with the exclusive HydraGlyde® Moisture Matrix technology. HydraGlyde® technology, which eye care professionals have been recommending to their patients for years as part of Opti-Free® PureMoist® Multi-Purpose Disinfecting Solution, reconditions and surrounds soft lenses in long-lasting moisture that provides extra hydration and superior wettability so soft lenses feel clean, fresh, and comfortable.**[9] CLEAR CARE® PLUS has the same cleaning and powerful disinfection abilities as CLEAR CARE®, exceeding the FDA and ISO criteria.[10,11] CLEAR CARE® PLUS is effective in killing bacteria, yeast, and fungi, and is also effective against *Acanthamoeba* trophozoites and cysts.†[10] CLEAR CARE® PLUS is preservative free and has the same neutralization performance as CLEAR CARE®, providing more complete neutralization of H_2O_2‡ to become a gentle saline, more like natural tears than PeroxiClear^ and Biotrue^.[12,13]

Which patients are the right candidates for CLEAR CARE® PLUS? Daily disposables are usually the natural first choice for many lens wearers, but for patients who are not candidates for daily disposables, CLEAR CARE® PLUS is an excellent choice that reduces the risk of infections and contact lens-related inflammation, improves compliance, and helps reduce dropouts.[8,9,14,15] Patients and their eye care professionals can be confident that CLEAR CARE® PLUS is as easy to use as MPS, using a simple, 3-step (Rinse, Fill, Soak) regimen. For your weekly and monthly contact lens patients, CLEAR CARE® PLUS is the clear choice!

*"Good" represents a score of 90% or higher based on a compliance questionnaire.
**Superior surface wettability was demonstrated with silicone hydrogel and soft contact lenses.
†There is no current standard to test the efficacy of lens care solutions against *Acanthamoeba* species.
‡As compared to PeroxiClear^. After neutralization for the labeled disinfection time, all levels were below the ocular awareness threshold of 100 ppm.[16]

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cause obstructive hydrocephalus.[4] This occurs when CSF is blocked along the narrow passages connecting the ventricles of the brain, which is detectable on MRI showing dilation of one or more of the ventricles in the brain.[4,12] A specific form of obstructive hydrocephalus called aqueductal stenosis can also be detected with MRI.

MRV is needed to visualize a dural venous sinus thrombosis that may be missed with MRI alone. Because MRI and MRV can both miss a communicating hydrocephalus, which occurs when CSF is blocked after exiting the ventricles of the brain, LP is always indicated if MRI and MRV are normal.[4,12] LP with CSF sent for biochemistry, microbiology and cytology is necessary to reveal infectious or carcinomatous meningitis, which are potential causes of a communicating hydrocephalus.[4]

If CSF is normal and opening pressure on LP is elevated (>20cm H^20), the modified Dandy criteria are still not met. The next step is a neurology referral to rule out localized findings with the exception of those known to occur with increased intracranial pressure such as CN VI palsy. The final diagnostic criteria mandate identifying all other potential causes of increased intracranial pressure. One cause of IIH is excess vitamin A consumption via use of some acne medications or diet supplementation.[1,4,13] Other medications, such as tetracycline, corticosteroids and systemic conditions—e.g., obstructive sleep apnea, renal failure and severe anemia—are associated with IIH.[4]

OCT and Ophthalmoscopy

Although ophthalmoscopy remains the most valuable and critical first-level investigation, OCT can be a useful tool for differentiation between disc edema and pseudo disc edema. Pseudoedema may be present in patients with optic disc drusen, which has been found in approximately 2% of adult autopsy eyes.[14] Optic disc drusen can alter the presentation of the optic disc, making it appear swollen. B-scan ultrasonography and fundus autofluorescence can be helpful to identify optic nerve drusen as well. With OCT, disc drusen are revealed with a "lumpy, bumpy internal contour," whereas true optic disc edema is recorded with a "smooth internal contour."[14,15] Our patient's OCT revealed a smooth internal contour consistent with true disc edema (*Figures 4a and 4b*). Other causes of pseudopapilledema include tilted optic disc, myelinated nerve fibers, congenitally crowded discs and optic nerve hypoplasia. Investigators found with OCT that the mean RNFL thickness is significantly greater in patients with true disc edema compared with those with pseudoedema of any type.[15] In fact, in cases of pseudopapilledema, RNFL thickness tends to be normal in all four quadrants.[16]

Ophthalmoscopy can also be used to distinguish true edema from pseudoedema. One study found that the presence of disc edema with associated chorioretinal folds (Paton's lines) had 100% sensitivity, and this finding is pathognomonic for true optic disc edema.[14] Unfortunately, these folds were only present in 23% of patients in the study. Of the signs evaluated, swelling of the peripapillary RNFL had the highest accuracy as a single sign.[14] Our patient had Paton's lines associated with the edema of the right optic disc, indicating true disc edema (*Figure 1*).

The standard method of grading disc edema by ophthalmoscopy, the

Table 2. Modified Frisén Scale [17]

Edema Grade	Degree of Edema	Description of Findings
0	Normal	Prominence of the retinal nerve fiber layer at the nasal, superior, and inferior poles in inverse proportion to disc diameter. Radial nerve fiber layer striations, without tortuosity.
1	Minimal	C-shaped halo that is subtle and grayish with a temporal gap and obscures underlying retinal details.* Disruption of normal radial nerve fiber layer arrangement striations. Temporal disc margin normal.
2	Low	Circumferential halo.* Elevation (nasal border). No major vessel obscuration.
3	Moderate	Obscuration of > or = one segment of major blood vessels leaving disc.* Circumferential halo. Elevation (all borders). Halo (irregular outer fringe with finger-like extensions).
4	Marked	Total obscuration of a segment of a major blood vessel on the disc*. Elevation (whole nerve head, including the cup). Border obscuration (complete). Halo (complete).
5	Severe	Obscuration of all vessels on the disc and leaving the disc*

**Key features (major findings) for each grade*

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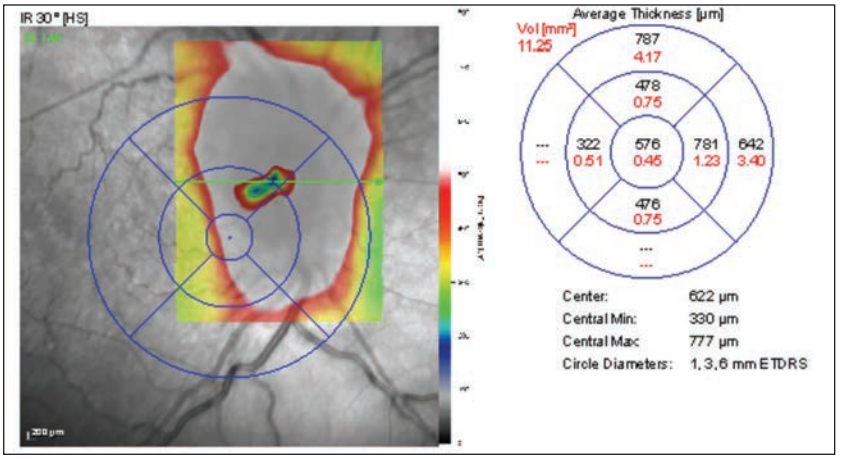
Modified Frisén Scale, is an ordinal scale with subjective evaluation and grades ranging from zero (no edema) to five (severe degree of edema) with characteristic findings for each increased grade (*Table 2*).[17]

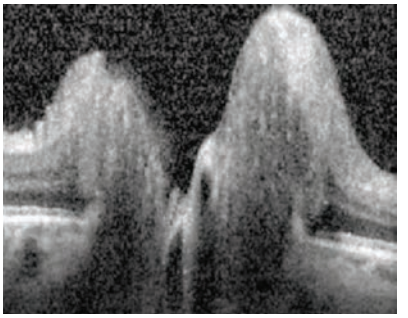
As opposed to Frisén grading, OCT offers an objective and continuous scale for measurement of edema and has been shown to be helpful to confirm the presence of edema, especially in mild cases.[18] The recently concluded Idiopathic Intracranial Hypertension Treatment Trial (IIHTT) found a strong correlation between OCT of papilledema at baseline and measures of swelling at the optic nerve head (ONH) with Frisén grading of papilledema at baseline.[18] The data in this OCT sub-study of the IIHTT is being evaluated to see whether OCT parameters are superior to Frisén grading in monitoring papilledema resolution. The investigators are also evaluating whether OCT findings can be correlated to visual outcome.[18]

In addition to the measurement of peripapillary RNFL thickness with OCT, it has been suggested that total ONH volume and ONH height may also be useful data points for diagnosing and monitoring disc edema.[15,19] OCT offers a non-invasive method to confirm disc edema in initial diagnosis and serves as an adjunct to ophthalmoscopy in following the course of the disease throughout treatment.

Don't Forget Perimetry

Both OCT and automated perimetry should be used in combination to follow patients with papilledema.[3] Visual field defects can range from absent to severe. During baseline visual field testing in the IIHTT researchers discovered that nerve fiber bundle-like defects, similar to those in glaucoma, are





Figs. 4a and 4b. Spectralis OCT optic nerve head thickness map of our patient's right eye. Although the thickness map is not centered directly over the nerve on this scan, the raster line is directed through the optic nerve head, revealing temporal and nasal edema with smooth internal contour.

characteristic of optic nerve damage from increased intracranial pressure. This type of defect was present in 60% of patients at baseline testing in the IIHTT. The most common specific visual field defect found in this study was a partial arcuate scotoma combined with an enlarged blind spot. Diffuse visual field loss or neurologic-like defects were not commonly found.[6]

Medications and Weight Loss

The standard first-line medical treatment in IIH is oral acetazolamide. Until the IIHTT, no properly designed clinical trials had been performed to provide an evidence base for acetazolamide treatment. In the study, patients diagnosed with IIH were treated with either acetazolamide combined with a low sodium diet for weight reduction or placebo combined with the same low sodium diet.[18]

The acetazolamide group was reported to have decreased papilledema, increased vision-related quality of life and showed a modest improvement in visual field function compared with the placebo group.[18] Acetazolamide is thought to work by inhibiting carbonic anhydrase causing a reduction in transport of sodium ions across the choroid plexus epithelium where CSF is produced in the brain. Research shows it can decrease CSF production in humans by anywhere from 6% to 50%.[10]

Neurologists and neuro-ophthalmologists treating IIH may pursue alternatives to acetazolamide if it is not tolerated. The most frequent early side effects with acetazolamide use are tingling in the fingers, toes and perioral region, called parasthesias. Other common symptoms include gastrointestinal upset, diuresis and malaise.[20] Second-line medical therapy is usually with an oral diuretic like furosemide. Sometimes topiramate is used.

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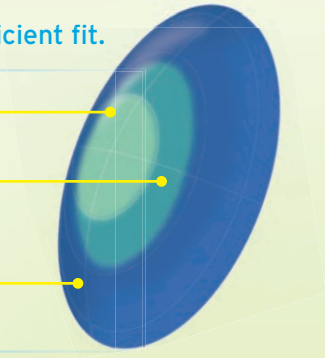

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REFERENCE: 1. Analysis based on use of a Hartmann-Shack wavefront sensing instrument to map lens power across contact lenses. More than 6000 unique measurements over the central 6mm of a contact lens were plotted to determine local power measurement as a function of radial distance from the center of the lens.

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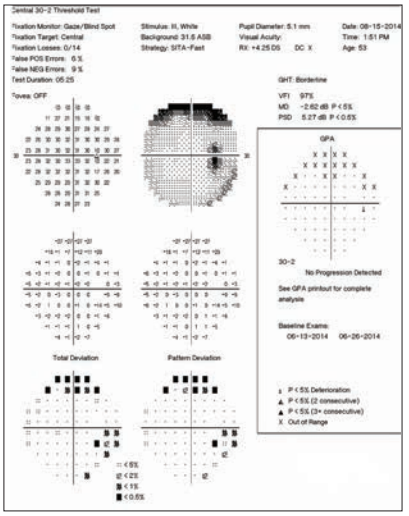

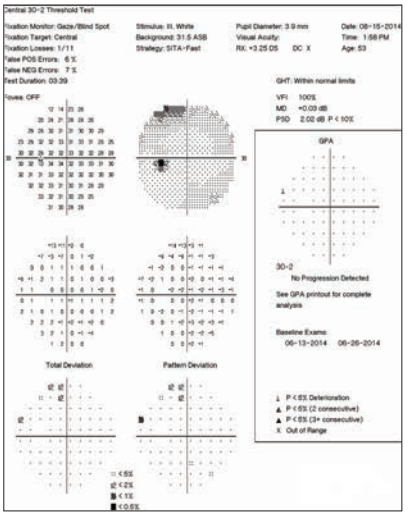

Figs. 5 and 6. At right, our patient's right eye HVF 30-2 SITA Fast results. The superior defect likely represents a brow artifact, but the clustered inferior temporal points reveal an enlarged blind spot. At left, his left eye HVF 30-2 SITA Fast results. The mildly depressed points superiorly likely represent interference of the patient's brow.

All medical therapies should be combined with a weight loss plan in obese and overweight patients to ultimately resolve the papilledema and preserve visual function. Normal BMI in IIH is rare, and in a recent study, none of the patients with IIH and normal BMI had severe visual loss in either eye. They were only treated medically, since weight loss was not needed for these patients.[5] Weight loss is the key to long-term success in the vast majority of patients treated for IIH. Clinical improvement in IIH has been associated with as little as 6% weight loss.[21] However, one study found a relapse rate of 28% in those treated for IIH and the relapse was associated with recent significant weight gain compared with non-relapsed patients.[2] Ultimately, some patients may be offered gastric bypass surgery for significant weight loss and long-term resolution of papilledema in IIH.[1]

Surgical Intervention

In patients with sudden and severe papilledema who may be at risk for irreversible vision loss, surgical intervention may be urgently needed. This form of IIH (called fulminant IIH) is rare, but potentially devastating. Blindness is reported in up to 10% of these patients, and visual field defects of some type occur in up to 90%.[21] Optic nerve sheath fenestration (ONSF) and cerebrospinal fluid shunting (CSFS) are the most commonly used surgical procedures, but it remains unclear whether one procedure is superior to the other.[1]

ONSF improves, or at least stabilizes, vision in 70% to 90% of cases and it has a lower complication rate compared with CSFS.[1,22] However, headache symptoms significantly improved in only 31% of cases with ONSF.[1] CSFS (by either ventriculoperitoneal or lumboperitoneal shunt) fail and require revision in up to 50% of cases.[1] However, one series showed 95% of patients reported significant improvement in their headache symptoms after shunting was completed.[1]

Investigators have proposed that in patients where vision is threatened and headaches are not severe, ONSF is the procedure of choice, and in cases with severe headaches, regardless of visual status, CSFS may be the preferred procedure after failure of medical therapy combined with weight loss.[4]

Our patient is currently medically managed by a neurologist. He is also followed at regular intervals in our eye clinic with dilated fundus evaluation, optic disc photos, OCT of the optic nerve and nerve fiber layer and automated threshold perimetry. Our patient's BMI classifies him as obese. We educated him on the value of weight loss in the management of IIH and referred him to a dietician. He was initially medicated with acetazolamide, and the enlarged blind spot on the visual field of his right eye has since resolved.

Although visual outcomes may be favorable with weight loss and medication, relapses with weight gain and the fact that optic nerve atrophy has been detected in well-treated patients suggest that long-term follow-up of IIH is necessary.[2] Regular fundus examination with repeated OCT and automated threshold perimetry over many years should be performed along with comanagement with a neurologist or neuro-ophthalmologist.

Eye care professionals may be the first to detect IIH, as we perform ophthalmoscopy on a routine basis. Bilateral optic disc edema is a true ocular and medical emergency, even in patients without symptoms who are 20/20 and have a normal visual field. Life-threatening conditions must be urgently ruled out, as IIH is a diagnosis of exclusion. ■

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Academy CE in The Big Easy

Optometrists head to New Orleans for education, exhibits and so much more at the 2015 Academy meeting. **By Jane Cole, Contributing Editor**

Home to Mardi Gras parades, Jackson Square and fresh beignets from Café Du Monde, New Orleans will be the backdrop for the 2015 American Academy of Optometry (AAO) meeting from Oct. 7-10.

Academy 2015 promises to surpass even last year's Denver meeting, which was the largest meeting in Academy history, says Joseph P. Shovlin, OD, Academy president-elect and annual meeting chair.

Photo: © istock.com/jobsonhealthcare

"Keep Your Eye on New Orleans" is the American Academy of Optometry's catch phrase for this year's conference.

The meeting includes more than 300 hours of continuing education and standouts such as the first joint Academy of Ophthalmology and Academy of Optometry symposium, highlighting both disciplines' collaborative work in pediatric eye care.

Joint Symposium

This landmark joint symposium, titled "Amblyopia and Beyond: Current Evidence-Based Pediatric Eye Care," will be on Friday, Oct. 9 from 2pm to 4pm. The program will be presented at both the New Orleans meeting and the American Academy of Ophthalmology's annual meeting in Las Vegas in November. This inaugural program is a united effort to support joint educational initiatives. Evidence-based pediatric eye care will be discussed by Donald Mutti, OD, PhD, Bruce Moore, OD, Susan Cotter, OD, MS, and American Academy of Ophthalmology representatives Jonathan Holmes, MD, Mary Louise Collins, MD, and Jean Ramsey, MD. This is the first time the two Academies have worked together to better prepare and support their members in delivering the highest quality eye care.

Meeting Highlights

"In addition, the plenary features perhaps the most talented team in medicine today," Dr. Shovlin says. "Drs. Carol and Jerry Shields, co-directors of Ocular Oncology at Wills Eye Hospital, will present a lengthy discussion on ocular melanoma."

Their luncheon session, "Today's Research, Tomorrow's Practice: Recognizing and Treating Ocular Melanomas," will be held on Wednesday, Oct. 7 from 12pm to 2pm and will highlight how to differentiate between suspicious and non-suspicious pigmented and amelanotic lesions, as well as new treatment and management options.

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Section Standouts at the AAO

The AAO meeting in New Orleans offers myriad learning opportunities. Here are the Section and SIG Symposium courses scheduled:

WEDNESDAY, OCT. 7

• *10am – 12pm.* **Academic Medical Center Optometrists SIG Symposium: The Future of Optometrists in Academic Medicine**
Moderator: Patti Fries, OD. Speakers: Michael F. Chiang, MD, and Susan M. Pollart, MD.

• *2pm – 4pm.* **Anterior Segment Section Symposium: Controversies and Evolutions in the Management of Corneal Infectious Disease**
Moderator: Paul Karpecki, OD. Speakers: Joseph P. Shovlin, OD, Christine Sindt, OD, and Michael DePaolis, OD.

• *2pm – 4pm.* **Binocular Vision, Perception and Pediatric Optometry Section: ARVO/AAO Joint Symposium: Visual Neural Plasticity: Cells to Systems**
Moderator: Tawna Roberts, OD, MS. Speakers: R. Douglas Fields, PhD, Benjamin Thompson, PhD, Tara Alvarez, PhD, and Michael Earley, OD, PhD.

• *2pm – 4pm.* **Public Health and Environmental Vision Section Symposium: Global Health and Emerging Diseases 2015**
Moderator: Jeffrey L. Weaver, OD. Speakers: Pierre Buekens, MD, John Mason, PhD, and Susan McLellan, MD.

• *3pm – 5pm.* **Nutrition, Disease Prevention and Wellness SIG Symposium: The Role of Carotenoids in Visual Performance and Cognition**
Moderators: Kimberly Reed, OD, and A. Paul Chous, MA, OD. Speakers: Lisa Renzi, PhD, Dennis Ruskin, OD, and Stuart Richer, OD, PhD.

• *7pm – 9pm.* **Glaucoma Section Symposium: Evidence Based Decision Making in the Management of the Open Angle Suspect**
Moderator: Richard Madonna, MA, OD. Speakers: Denise Pensyl, OD, MS, Anthony Litwak, OD, and John McSoley, OD.

THURSDAY, OCT. 8

• *10am – 12pm.* **Neuro-Ophthalmic Disorders in Optometry SIG: 2015 Lawrence Gray Symposium on Neuro-Ophthalmic Disorders**
Moderator: Leonard Messner, OD. Speakers: Tina Porzukowiak, OD, Nancy Newman, MD, and Valerie Biousse, MD.

• *10am – 12pm.* **Optometric Education Section Symposium: Effective Clinical Teaching: What's the Best Evidence?**
Moderators: Michael Giese, OD, and Meredith Whiteside, OD. Speakers: John Littlefield, PhD, and Brenda Talley, MD.

• *3:30pm – 5:30pm.* **Primary Care Section Symposium: Innovative Optometry: Light, Technology, Telemedicine and the Future**
Moderator: Tina Porzukowiak, OD. Speakers: Nicole Putnam, PhD, Kirk Smick, OD, Charles Kinnaird, OD, Anastas Pass, OD, JD, and Scot Morris, OD.

• *7pm – 9pm.* **Low Vision Section Symposium: Update on the Preferred Retinal Locus: What Have We Learned About Visual Function and the Development of the Pseudofovea in the Context of Macular Disease?**
Speakers: Nicole C. Ross, OD, MSc, Russell Woods, PhD, MCOptom, Susana Chung, OD, PhD, Kristina Visscher, PhD, and Gary Rubin, PhD.

FRIDAY, OCTOBER 9

• *10am – 12pm.* **Section on Cornea, Contact Lenses and Refractive Technologies Symposium: Layers, Lenses and Lasers**
Moderator: Jeffrey Krohn, OD. Speakers: Ronald Krueger, MD, and Jayne Weiss, MD.

• *10am – 12pm.* **Vision in Aging SIG and Public Health and Environmental Vision Section Symposium: Aging in the International Year of Light**
Moderator: Sue Leat, PhD. Speakers: Alan Lewis, OD, PhD, Jack Werner, PhD, Mariana Figueiro, PhD, and Michael Flanagan, PhD.

• *4pm – 6pm.* **Vision Science Section and Low Vision Section Symposium: New Approaches to Rehabilitation of Visual Field Loss after Brain Damage**
Moderators: Alex Bowers, MCOptom, PhD, and Nicole C. Ross, OD, MSc. Speakers: Denise Goodwin, OD, Russell Woods, PhD, MCOptom, Alex Bowers, MCOptom, PhD, Alison Lane, PhD, and Krystel Huxlin, PhD.

• *4pm – 6pm.* **Optometric Education Section Hands-On Session: Teaching in the Clinic: Methods of Efficient Education**
Speakers: Michael Giese, OD, Meredith Whiteside, OD, John Littlefield, PhD, and Brenda Talley, MD.

SATURDAY, OCTOBER 10

• *10am – 12pm.* **Retina SIG Symposium: Diabetes: A Comprehensive Team Approach**
Moderator: Steven Ferrucci, OD. Speakers: A. Paul Chous, MA, OD, Mandeep Brar, MD, and Michael Tolentino, MD.

Another highlight will be the Monroe J. Hirsch Research Symposium, "Research Matters: How Research Changed Practice," on Thursday, Oct. 8 from 8am to 10am. The symposium will give a bird's eye view of the steps required to transition from bench to bedside, including the key target areas that drive laboratory-based research and the establishment of diagnostic methodologies to test for and monitor therapeutic and postoperative responses. Speakers will include: Lucia Sobrin, MD, MPH, who will lead a discussion about the impact of the growing field of molecular genetics on patient care and clinical outcomes; Matthew Petroll, PhD, who will present his work on the development and clinical implementation of ophthalmic imaging devices, with an emphasis on confocal microscopy, including data

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he acquired from the first confocal microscope ever used to image the human cornea; and Andrew Lee, MD, who will speak about the impact of clinical trials, focusing on the Optic Neuritis Treatment Trial's influence on practice patterns.

The Prentice and Fry Lectures and Awards Program will be held on Friday, Oct. 9 from 8am to 10am. The most deserving optometrists and vision scientists will be honored for their research and dedication to the profession. The Glenn A. Fry and Charles F. Prentice Award recipients will give presentations on their research findings. Clinicians can earn one hour of CE by attending both hours of the program, and a continental breakfast will be provided.

Two investigators, who were supported early on by AOF Ezell Fellowships, will present "Tackling Traumatic Brain Injury" on Friday, Oct. 9 from 10am to 12pm. They will focus on how retinal and visual function is affected by TBI. New measures to quantify traumatic brain injury recovery will also be discussed so that clinicians can make more educated decisions as to whether or not an athlete is ready to return to play. Speakers include Kristine Dalton, OD, PhD, and Andrew Hartwick, OD, PhD.

The meeting will also offer specialty topics (*see, Section Standouts at the AAO*), including the Anterior Segment Section Symposium: Controversies and Evolutions in the Management of Corneal Infectious Disease on Wednesday, Oct. 7 from 2pm to 4pm. The panel features Paul Karpecki, OD, Dr. Shovlin, Christine Sindt, OD, and Michael DePaolis, OD. The team will review the changing landscape of corneal infectious disease. Old controversies, such as the use of empiric vs. culture-driven treatment strategies and the use of corticosteroids, will be reviewed in light of antimicrobial resistance. They will also consider new practice trends such as the increasing clinical appearance of herpes zoster in the healthy adult population and the use of corneal crosslinking as an antimicrobial strategy.

Optometrists can earn a maximum of 35 continuing education credit hours during the meeting. Be sure to mark your calendars for some unsurpassed educational opportunities and a hearty dose of New Orleans Cajun flavor.

For more information, go to www.aaopt.org. ■

Living it Up in NOLA

If you are looking for a break between CE sessions, check out some of these top attractions:

- **Algiers/Canal Ferry.** Used by locals on their daily commutes, the ferry also serves as a fast, scenic way to navigate from one end of the city to the other.
- **Audubon Butterfly Garden and Insectarium.** Dedicated to over 900,000 species of insects and their relatives, this insectarium gives you a glimpse into the beautiful, exciting world of these curious creatures.
- **Blaine Kern's Mardi Gras World.** This museum also serves as the largest float-building facility in the world. Guided tours give a history of the festival, from the mule-drawn carriages of the past to the sophisticated mega-floats of today.
- **Carousel Gardens Amusement Park.** Since 1906, everyone has enjoyed the "flying horses" of City Park's antique carousel, one of only approximately 100 antique wooden carousels left in the country and the last one in Louisiana.
- **Congo Square.** Slaves and free blacks gathered here throughout the 19th century for meetings, open markets and the African dance and drumming celebrations that played a substantial role in the development of jazz. Local voodoo practitioners still consider Congo Square a spiritual base and gather there.
- **French Market.** Founded in 1791, New Orleans' French Market is the oldest public market in the country. This open-air mall features shopping, dining and music in a tradition that is uniquely New Orleans.
- **French Quarter.** This is home to some of New Orleans' best restaurants, art galleries and shops, in addition to the famed Jackson Square and the debauchery of Bourbon Street.
- **Jackson Square.** This New Orleans treasure earned its name for one of three bronze statues of Andrew Jackson located in the center of the square. Jackson Square is also the location of an open-air artist colony, where artists display their work on the outside of the iron fence.
- **The National World War II Museum.** The country's official World War II Museum lets you experience America's role in the war. Don't miss "Beyond All Boundaries," a multi-sensory cinematic feature from producer Tom Hanks, playing at the 4D Solomon Victory Theater.
- **Steamboat Natchez.** This classic Mississippi riverboat sails three times daily, at 11:30am, 2:30pm and 7pm. Traditional New Orleans jazz is featured and the evening cruise includes dinner (reservations required).
- **Voodoo Museum.** Voodoo has always been a significant part of New Orleans culture. This museum strives to preserve this aspect of NOLA history through education and entertainment. The museum explores the mysteries, legends and traditions of voodoo and educates visitors on the influence voodoo has had throughout the city's history.

Find more at www.neworleansonline.com.

Disinfection Efficacy Delivered by a Peroxide Solution in 4 Hours

Bausch + Lomb PeroxiClear® Solution offers convenience for busy patients

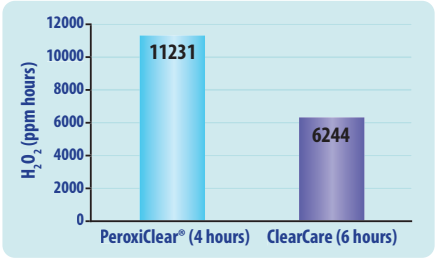
By
Jim Hoffman, OD, FAAO
Orange Park Eye Center
Orange Park, FL

Now is a great time for eye care professionals to engage in conversations about Hydrogen Peroxide solutions with their patients to enhance the contact lens wearing experience. Typically these solutions have been perceived to be cumbersome and inconvenient compared to multipurpose solutions. Hydrogen peroxide disinfecting systems can be a great option for patients and now our practice recommends a peroxide solution that is convenient to use.

The profession agrees that hydrogen peroxide solutions provide exceptional cleaning and comfort for patients who may experience deposits or build-up on their contact lenses, especially toric and multifocal lenses.[1] In addition, these solutions are preservative-free, making them an ideal option for contact lens wearers who are sensitive to multipurpose solutions. Now there is a peroxide solution that cuts down on the disinfection cycle time, enabling patients to wear their lenses sooner.

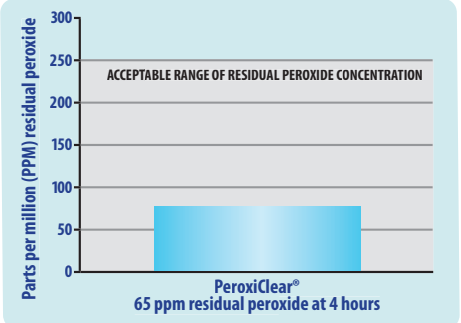
Bausch + Lomb PeroxiClear® Solution is the only system designed to control peroxide neutralization during the first hour of disinfection, increasing the total peroxide exposure and shortening the minimum soaking time to just four hours. The difference is that PeroxiClear® contains *carbamide*, a platinum modulating compound (PMC) that slows the neutralization rate in the initial phase which speeds up the disinfection cycle in two hours less time.[1] By comparison, the regimen time for Clear Care to achieve adequate disinfection is six hours.

PeroxiClear®: Higher peroxide exposure delivers 99.9% disinfection in 2 hours less time[2]



I have been impressed that within four hours, PeroxiClear® is highly effective at killing 99.9% of microbes. The resulting residual peroxide levels are well within the acceptable range of residual peroxide concentration, so our patients can safely and comfortably reinsert their contact lenses sooner. Patients comment how comfortable their lenses are when they first put them on.

Peroxide concentration after neutralization[3]



For some patients who use a peroxide solution, a shorter disinfection time can be a huge advantage. PeroxiClear® lets patients disinfect and reinsert their contact lenses in only four hours. One of my patients is a businessman with an unpredictable travel schedule. Thanks to the shorter disinfection time with the PeroxiClear® system, he can comfortably reinsert his lenses after a brief night of sleep. Another patient who uses PeroxiClear® is a nurse at a busy hospital. She can conveniently get her lenses ready for use before starting an overnight shift.

Successful contact lens wearers eagerly embrace innovative technology that improves their lens wearing experience. Many patients will benefit from the flexibility of a shorter disinfection time with PeroxiClear®. In addition, many of my patients who use PeroxiClear® say their lenses feel more comfortable immediately on insertion and stay moister and cleaner throughout the day.

I have found that PeroxiClear® simplifies the ever-expanding choices in contact lens care systems. PeroxiClear® meets the established standards in disinfection efficacy within four hours, maintains safe residual peroxide levels at the end of the neutralization and disinfection cycle, and provides long-lasting comfort. I recommend PeroxiClear® because it provides the convenience, safety, and comfort my patients want and need.

References: 1. Millard KA, Groemminger S, Xia E, Kilbury J. Evaluation of platinum modulating compounds to delay the neutralization of hydrogen peroxide in one-step disinfecting systems. Abstract presented at: Global Specialty Lens Symposium; 2014 Jan 23-26; Las Vegas, NV. **2.** Mean results of total area under curve (AUC) measurements of PeroxiClear® and Clear Care 3% hydrogen peroxide systems show the total peroxide exposure to a contact lens during the disinfecting regimen, which includes a minimum of 4 hours neutralization time for PeroxiClear® and 6 hours for Clear Care. Both product systems were tested four times. System lens cases were conditioned with one complete neutralization cycle prior to second cycle measurments of peroxide concentrations at 0, 5, 15, 30, 60, 120, 240, and 360 (Clear Care only) minutes. Method of peroxide concentration measurement is adapted from USP monograph for hydrogen peroxide 3% topical solutions (USP35-NF30) for the assay of hydrogen peroxide. Peroxide concentrations at each point in time were plotted to generate a neutralization curve. Total AUC is calculated using graph prism software. **3.** Data on file. Bausch & Lomb Incorporated. Rochester, NY; 2013

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Testing: More Than Just a Code

New technology is great to have, but be sure to know the rules on coding these tests.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Incorporating new technology into your practice is always an exciting event. Being able to harness this newfound clinical assistant allows you to continue to provide care at the highest level. However, incorporating new technology isn't always a slam-dunk; each CPT code used to describe the testing performed has specific rules, regulations and guidelines that you have to follow before the test can be ordered, performed and billed. You can't simply do the test because you have a covered diagnosis or the procedure is covered by a plan.

Medical Necessity vs. Covered Procedure

Somewhere between "this is how you turn it on" and "this is a list of covered diagnoses you can bill for" lies the professional responsibility of establishing *medical necessity* for ordering tests or performing procedures. You must demonstrate in the medical record that the procedure or test is needed to diagnose, follow a diagnosis, treat or monitor treatment. You must tell the story of the patient encounter, including your reasoning for what you are doing. If you feel that a procedure is necessary to aid you in a patient's diagnosis or treatment, then tell the record why you feel that way. It's your only defense in a post-payment review process.

With ICD-10, you are going to have to know whether the procedure is bilateral or unilateral, whether it requires a modifier to change it and whether your patient's specific presentation requires the test in one or both eyes. This, of course, will have to support the specific laterality of the ICD-10 codes.

A whole host of patient symptoms appear in lists of *covered diagnoses* for a specific procedure. However, the ICD is quite specific in what you can—and can't—use as a billable diagnosis. Current ICD rules indicate that codes describing symptoms and signs, as opposed to diagnoses, are acceptable for reporting purposes only when a provider has not established a related definitive diagnosis. You cannot simply perform any test you want just because the symptom is covered; you must base your decision on the actual diagnosis and medical necessity of that individual patient. Remember, HIPAA requires you to follow the rules of the ICD, so you are legally bound to do so.

Interpretation and Report

An interpretation and report (I&R) needs to be clearly identified within your medical record for the specific test with which it is associated. Each test you perform requires its own I&R because each must have proper medical necessity established in the medical record if a third-party carrier is going to pay for it.

If you have a specific reason why you believe a test may be denied, use an advance beneficiary notice and the appropriate modifier accordingly.

Here are the typical items you should include in an I&R:

- Clinical findings, which are the pertinent data of the test results.
- Your interpretation of those findings.
- Comparative data to previous test results (if applicable).
- Clinical management, which explains how the test results will affect the management of the condition/disease. Examples include: change/increase/stop medication; recommend surgery or further diagnostic testing; refer to a specialist/subspecialist for additional treatment; or return for additional office visits for treatment or monitoring.

Simply performing the technical component of the test is not enough, nor is initialing the test to show that you've looked at it. When a carrier finds that an I&R hasn't been completed, the entire test is deemed invalid and financial recoupment is for the test in total. If you perform only the technical component without completing the I&R, the test isn't complete in the eyes of the insurer and the entire payment amount, not just the professional component, is recouped by the carrier.

Diagnostic testing is more common in today's optometry practice than ever, and will continue to grow. Fulfilling the diagnostic test requirements by appropriately completing an I&R for each test performed will not only benefit your patient, but will also reduce your risk. ■

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Dendriform Dilemmas

Pay attention to the presentation when choosing the appropriate treatment.

Edited by Joseph P. Shovlin, OD

Q I recently attended a lecture on viral eye disease in which the speaker differentiated between mucous plaques and dendriform lesions in herpes zoster that might harbor live virus. Do they respond to topical antivirals? How can I make the distinction between those lesions that are possibly treatable with antivirals vs. those that would likely not respond to antiviral treatment?

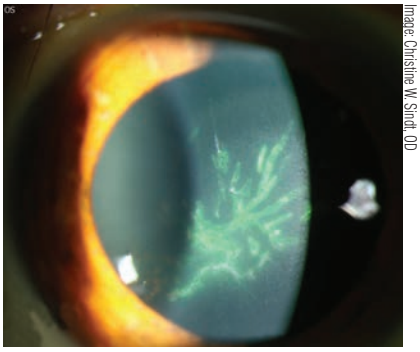

Image: Christine W. Sindt, OD

Pseudodendrites present as an acute keratitis within a month after skin lesions.

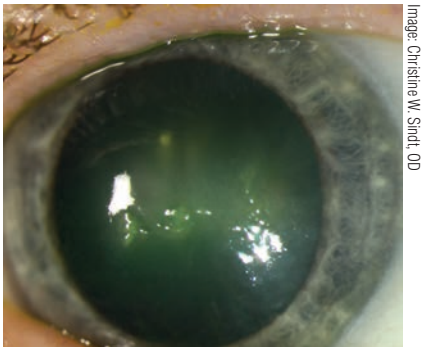

Image: Christine W. Sindt, OD

A mucous plaque stained with rose bengal dye.

A "It is often difficult to distinguish on clinical exam (i.e., slit lamp examination) between true herpes zoster mucous plaques, which are virtually always noninfectious, [and] various herpes zoster dendriform lesions, which, depending on the timing of the culture, may or may not harbor live virus," says Vincent de Luise, MD, assistant clinical professor or ophthalmology at Yale University School of Medicine. "Both types of lesions can appear as elevated, plaque-like, medusoid epithelial lesions with variable staining patterns to rose bengal, lissamine green and fluorescein, although mucous plaques usually are much larger and pleomorphic."

Dr. de Luise says that the FDA has not yet approved any topical antivirals for use against the herpes zoster virus (VZV) when it manifests as keratitis. "Off-label topical trifluridine 1% solution is rarely effective," he notes, while "the off-label use of topical ganciclovir 0.15% gel may have some efficacy. Oral antivirals have not demonstrated efficacy in any consistent manner." He adds that short courses of topical corticosteroids, with careful follow-up, can be used to treat herpes zoster nummular keratitis and herpes zoster disciform keratitis, as well as other manifestations that are inflammatory in nature. But, that is not the case with herpes zoster mucous plaques or infectious zoster dendriform keratitis.

Jason Duncan, OD, associate professor at the Southern College of Optometry, recommends use of ganciclovir gel should you decide you are dealing with dendrites associated with zoster ophthalmicus. Ganciclovir gel may be less toxic to the cornea when compared to other topical antivirals that are successful in treating dendritic keratitis associated with simplex disease. This becomes important to consider because the length of topical antiviral treatment may be longer and the taper more extended when dealing with zoster ophthalmicus. Also, "you must be sure that you are dealing with epithelial lesions, as zoster ophthalmicus often produces corneal stromal lesions, or nummular keratitis. The appropriate treatment for such lesions is the judicious use of topical steroids in conjunction with some form of antiviral therapy," Dr. Duncan says. Gancyclovir gel is typically prescribed five times daily until the epithelial lesion has healed and then used for an additional two to four weeks BID.

Mark Abelson, MD, founder of Andover Eye Associates in Massachusetts, points out that regardless of the type of lesion, the virus strain should respond similarly to antivirals unless the patient harbors an antiviral resistant strain. "The distinction should be based upon empirical assessment with an appropriate course of antiviral therapy," he says, adding that patients should be informed that the treatment provides symptomatic relief, but that the virus will remain in a latent state even when no symptoms are obvious. He recommends encouraging all patients over 50 years of age to receive immunization against herpes zoster to prevent possible recurrences. ■

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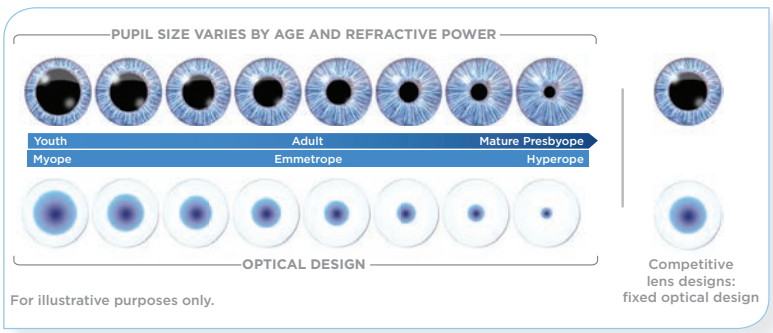

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The OD's Role in Stroke Care

With noninvasive imaging, we can trace damage back to the retinal ganglion cell complex. **By Surbhi Bansal, OD, Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

Cerebrovascular accident (CVA), or stroke, is a medical emergency that occurs as a result of ischemia or hemorrhage leading to abnormal perfusion of brain tissue. The initial symptoms of stroke may include sudden loss of speech, dizziness, confusion, weakness or paralysis of one side of the body, headache and seizure. Because ocular manifestations may be the presenting signs of stroke, the optometrist's role in the stroke patient's care is rapidly expanding.

Symptoms and Signs

Common visual symptoms of stroke include blur, visual field loss and diplopia. A patient experiencing a transient ischemic attack (TIA) may report amaurosis fugax, or a fleeting (usually monocular) blindness that occurs rapidly and resolves in a few minutes. Symptoms of bilateral transient vision loss may result from vertebrobasilar insufficiency. Patients who suffer a TIA are at risk for a major CVA shortly thereafter.[1]

Retinal emboli may precede cerebral stroke and are associated with an increased incidence of vascular disease and stroke-related death.[2] Other common ocular manifestations include cranial nerve involvement and homonymous field loss.

Prompt Action

Prompt diagnosis and treatment of stroke and TIA are crucial, as early action can minimize brain damage and potential complications. A patient may come to you with some of the symptoms and signs listed above, thinking it is only an eye issue. It is important for the clinician to think beyond the eye in such cases. If you observe signs that indicate a patient is having, or about to have, a stroke, immediately call 9-1-1 so that medical personnel can begin life-saving treatment on the way to the emergency room.

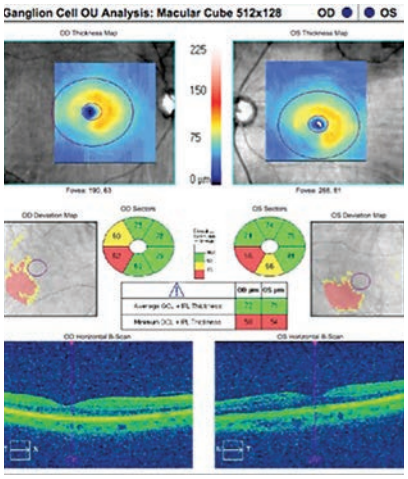

Fig. 1. SD-OCT ganglion cell analysis in a patient with corresponding homonymous field defects following a stroke. Note GCC thinning inferotemporal to the foveal center OD and inferonasal OS, shown as areas within the blue squares on the deviation maps.

Ideally, a multidisciplinary health care team will evaluate the stroke patient. Neuroimaging plays a vital role in the workup of acute stroke by providing information essential to accurately triage patients, expedite clinical decision-making with regards to treatment and improve outcomes (*Table 1*). If the patient suffered an ischemic stroke, the team may administer a thrombolytic drug, such as tissue plasminogen activator (tPA).[3] In hemorrhagic stroke, medications, surgery or endovascular procedures may be necessary to stop the bleeding and save brain tissue.[4]

Assessing the Damage

Axonal degeneration is a phenomenon that occurs when a nerve fiber loses function after an injury or other traumatic event, such as CVA. This process begins at the anatomic site of the damage and can happen in an anterograde fashion (toward the post-synaptic end) or in a retrograde fashion (toward the presynaptic cell body). When the degeneration crosses a synapse, it is referred to as trans-synaptic.[5]

In stroke patients, recent research shows that thinning of the retinal ganglion cell complex (GCC), as seen on optical coherence tomography (OCT), can help practitioners detect retrograde degeneration.[5] Evidence of retrograde trans-synaptic degeneration exists in patients with homonymous hemianopsias from occipital ischemia. This relationship is reflected in thinning of the GCC and maintains the topographic relationship of the visual field defect (*Figure 1*).

Rehab and Neuroplasticity

Stroke affects the brain's information processing, and the effect on a patient's visual function is variable, often depending on the location and

extent of the insult. Visual changes associated with stroke can be categorized as sensory (visual acuity and visual field), motor (extraocular muscle motility) and perceptual.[6]

The term neuroplasticity refers to the brain's ability to structurally alter itself and create new axonal connections in response to changing internal and external conditions. Neuroplasticity occurs in the brain under two main conditions:

(1) During normal brain development when the immature brain first begins to process sensory information through adulthood.

(2) As an adaptive mechanism to compensate for lost function or to maximize remaining function in the event of brain injury or stroke.[7]

The goal of vision rehabilitation in post-CVA patients is to maximize visual function for various activities of daily living. The cognitive and psychological status of the patient are assessed and integrated into the rehabilitation program. Optometric physicians skilled in vision rehabilitation are an important part of the multidisciplinary stroke rehabilitation team.

Stroke is the fifth leading cause of death in the United States and results in a drastic reduction in quality of life.[8] Optometrists are integral to the care team, helping stroke patients manage the ocular effects and maintain quality of life. ■

1. Purvin V. Cerebrovascular disease and the visual system. Ophthalmol Clin N Am. 2004;17:329-55.

Table 1. Neuroimaging for Stroke[1]

The neuroradiology team will select from among various studies:

- **Computed Tomography (CT):** Advantages include widespread access and speed of acquisition. In hyperacute phases, a non-contrast CT scan is usually ordered to exclude or confirm hemorrhage.
- **Magnetic Resonance Imaging (MRI):** Provides excellent soft tissue contrast. Fluid attenuated inversion recovery (FLAIR) images provide detection of acute subarachnoid hemorrhage. Magnetic resonance angiography (MRA) can be performed in combination with MRI to help guide therapeutic decision-making. MRI can detect atherosclerotic lesions in the neck and head.
- **Nuclear Imaging:** Functional imaging with positron emission tomography (PET) and single photon emission computed tomography (SPECT) might reveal carotid plaque vulnerability for rupture and help in evaluating brain viability.
- **Ultrasonography:** Carotid Doppler for the diagnosis of carotid stenosis.
- **Cerebral Angiography:** Imaging of vascular diseases of the brain and great vessels of the neck.

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8. Centers for Disease Control and Prevention. Stroke. www.cdc.gov/stroke/.

Stroke Q & A

Surbhi Bansal, OD, answers frequently asked questions regarding the optometrist's role in a patient's post-stroke care and vision rehabilitation:

Q: *How can optometrists help patients after the diagnosis of stroke has been established and treatment is administered?*
A: A neuro-optometrist will evaluate the following areas in addition to a routine eye exam: integrity of the visual field, unilateral spatial inattention (e.g., clock test, line bisection test), eye movements (fixation, pursuits, saccades), binocular and accommodative systems and photosensitivity (if present).

Q: *What are some specific neuro-optometric rehabilitative techniques and optical products that are useful for stroke patients?*
A: Techniques that involve visual scanning and peripheral awareness are beneficial for patients with a homonymous hemianopsia/quadranopsia resulting from stroke. Prism adaptation training can be useful for patients demonstrating unilateral spatial inattention. Optical tints, yoked prisms, spotting/sector prisms and sector occlusion can be helpful as well.

Q: *Is it beneficial to initiate vision rehab at an inpatient facility?*
A: Depending on the severity of the stroke, a visual evaluation should be performed three to six months after the event. While the vision rehab process can begin at an inpatient facility, the patient will experience their visual limitations once they re-enter the community (e.g., difficulty reading, using the computer). Therefore, the vision rehab process can focus on those areas of difficulty.

Dr. Bansal is an assistant professor at Nova Southeastern University, and specializes in neuro-optometric rehabilitation.

Suggested Resources:

- Schofield TM, Leff AP. Rehabilitation of hemianopa. Curr Opin in Neuro. 2009;22:36-40.
- Suter PS, Margolis NW. Managing visual field defects following acquired brain injury. Brain Injury/Professional. 2005;2:26-9.
- Massucci ME. Prism adaptation in the rehabilitation of patients with unilateral spatial inattention. J Behavior Optom. 2009;4:101-5.
- Suchoff IB, Ciuffreda KJ. A primer for the optometric management of unilateral spatial inattention. Optometry. 2004;75:305-18.
- Ciuffreda KJ, Han Y, Kapoor N, et al. Oculomotor rehabilitation for reading in acquired brain injury. NeuroRehabilitation. 2006;21:9-21.

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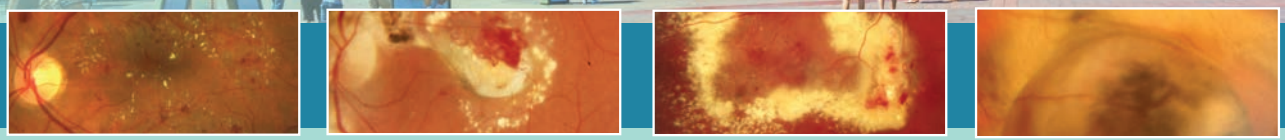

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Should Have Stayed on Vacation

While on a trip to Europe, a patient experiences blurred vision. Paired with his comorbidities, can you identify the cause? **By Mark T. Dunbar, OD**

A 64-year-old Hispanic male presented with complaints of dryness and blurred vision in both eyes. He was vacationing in Europe a month earlier and noted difficulties focusing and seeing clearly.

His ocular history is significant for having had cataracts extracted from both eyes 10 years earlier with excellent visual outcomes.

He also reported being told that he has keratoconus, but that had not caused any issues beyond needing glasses. His last eye exam was approximately 18 months earlier.

His past medical history is significant for lung cancer, which was diagnosed approximately two years earlier, and hypertension, which was well controlled. He had been on a chemotherapy medication, but that had been discontinued approximately three months prior due to being put on a study medication for the lung cancer.

On examination, his entering acuities measured 20/30 in each eye; however, he was easily refracted to 20/20. Confrontation visual fields were full to careful finger counting in both eyes. His pupils were equally round and 3+ reactive with no afferent pupillary defect. The anterior segment was significant for 1+ punctate epithelial erosions in both eyes and well-positioned posterior chamber intraocular lenses (IOLs). A dilated fundus exam showed small cups with good rim coloration and perfusion in both eyes. In the right eye, there were changes temporal to the macula (*Figure 1*). An SD-OCT was also performed (*Figure 2*). The left eye was completely normal.

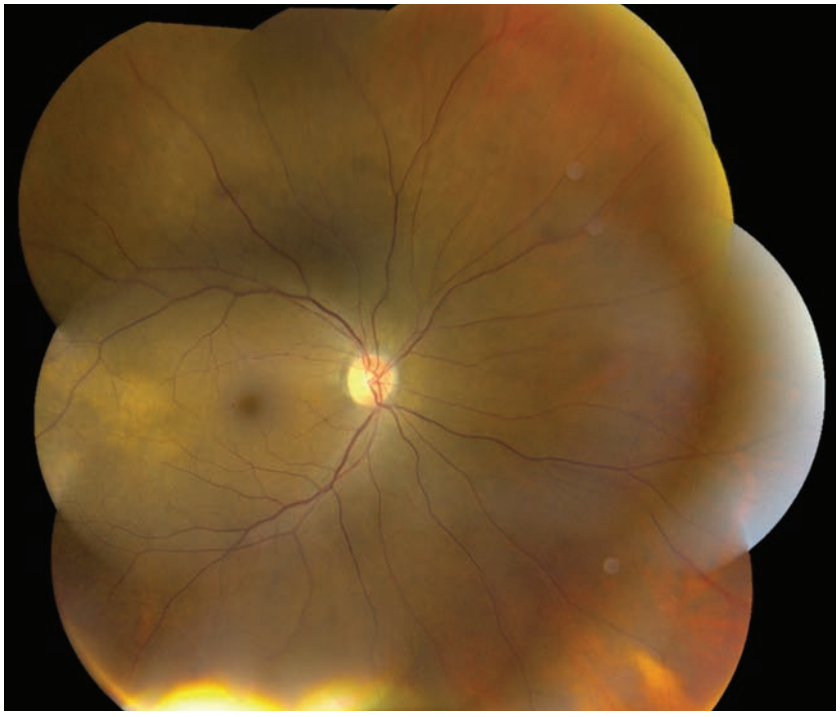

Fig. 1. Montage image of the right eye of our patient—what do you see?

Take the Retina Quiz

1. What does the SD-OCT show?
   a. It's normal.
   b. Irregularity of the RPE.
   c. A PED temporal to the macula.
   d. A choroidal mass.

2. What additional testing would be helpful in establishing a diagnosis?
   a. Visual field.
   b. Fluorescein angiography.
   c. Standardized ultrasound.
   d. Fine-needle biopsy.

3. What is the correct diagnosis?
   a. Amelanotic choroidal melanoma.
   b. Amelanotic choroidal nevus.
   c. Idiopathic central serous chorioretinopathy.
   d. Metastatic carcinoma to the choroid.

4. How should this patient best be managed?
   a. Observation only.
   b. Refer the patient to an oncologist.
   c. Anti-VEGF injection.
   d. Plaque radiotherapy.

For answers, see page 138.

Diagnosis

Upon clinical exam of the right eye, we observed a poorly circumscribed, creamy yellow lesion that appeared slightly elevated. In addition, near the ora serrata inferior nasally at 5 o'clock, we observed another creamy yellow lesion that also appeared depigmented. The latter lesion appeared flat.

The SD-OCT through the lesion near the macula confirmed the subtle elevation that was seen on clinical exam. What is particularly interesting is the contour of the RPE in that area, which is being bowed, or pushed, anteriorly. This is due to a mass within the choroid that was pushing forward and causing a dome-shaped elevation. Given our patient's history of having lung cancer, and the multiple cream-colored lesions, we felt certain this represented choroidal metastasis.

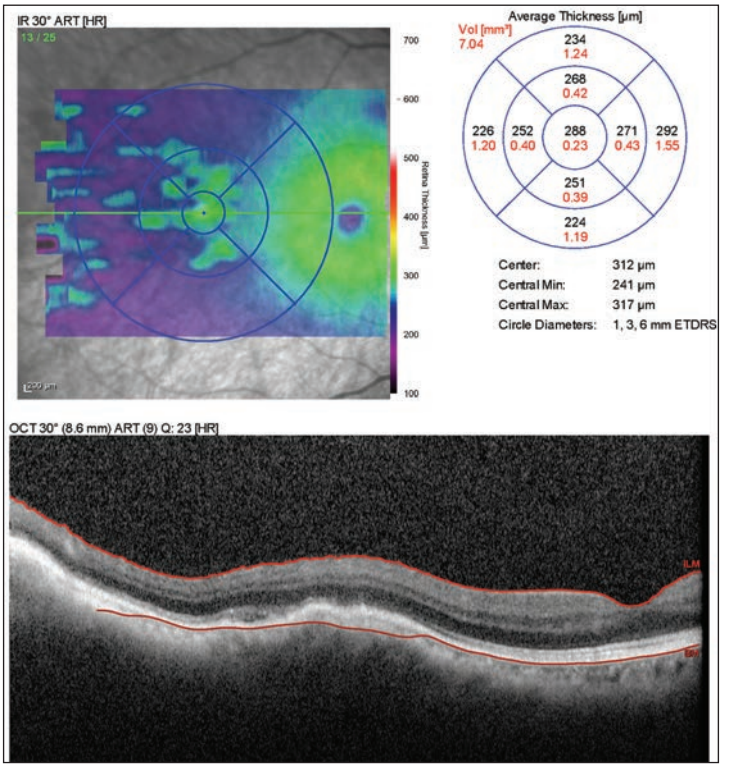

Fig. 2. This is the patient's SD-OCT image temporal to the macula.

Standardized echography was performed, which showed a 1mm thick choroidal mass measuring 10mm x 7.5mm. The internal reflectivity showed moderate reflectivity, which was consistent with choroidal metastasis.

Choroidal metastasis is the most common malignant tumor of the eye with a reported incidence of 2% to 9%, with the majority of the cases being due to breast cancer in women, followed by lung cancer in males.[1] The choroid is one of the principal sites for development of metastasis in the eye due to its abundant arterial supply and favorable microenvironment for "seeding" of the cancer cells.[2]

The vast majority of patients who develop metastasis of the eye will have a known site of the primary tumor at the time of diagnosis. But that is not always the case. In some instances, the discovery of metastasis in the eye may be the presenting sign of cancer. In one report of 420 patients with choroidal metastasis, 34% had no history of cancer at the time of diagnosis. The tumors were unilateral in 76% of patients, and multiple lesions were seen in 20% of eyes, with a mean number of two lesions.[3]

Our patient already had a confirmed diagnosis of lung cancer, so it was not difficult to make the connection between the findings in his eye and his lung cancer.

The clinical features of choroidal metastasis are that of an ill-defined creamy-yellow lesion at the level of the choroid. There may be multiple lesions and, as indicated, they may be bilateral.

Depending on size and extent, non-rhegmatogenous retinal detachment may be present. A metastatic lesion arising from skin melanoma may be darker in color, and a lesion developing from renal cell carcinoma may appear reddish-orange in color.

Once a diagnosis of metastatic carcinoma is made, coordinated care with the oncologist and eye care provider is important. Treatment options generally center around systemic platinum-based chemotherapy or localized ocular therapy, such as external beam radiation (EBRT) or plaque therapy. For bilateral tumors, EBRT is the treatment of choice.[4]

The patient's oncologist was notified of our findings and referred him to an ocular oncologist. The patient was put back on his chemotherapy regimen and by the time he saw the ocular oncologist, it was felt the metastatic tumor was beginning to regress, so no localized treatment was initiated. He continues to be followed. ■

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4. Kanthan GL, Jayamohan J, Yip D, Conway RM. Management of metastatic carcinoma of the uveal tract: an evidence-based analysis. Clin Experiment Ophthalmol. 2007;35:553–65.

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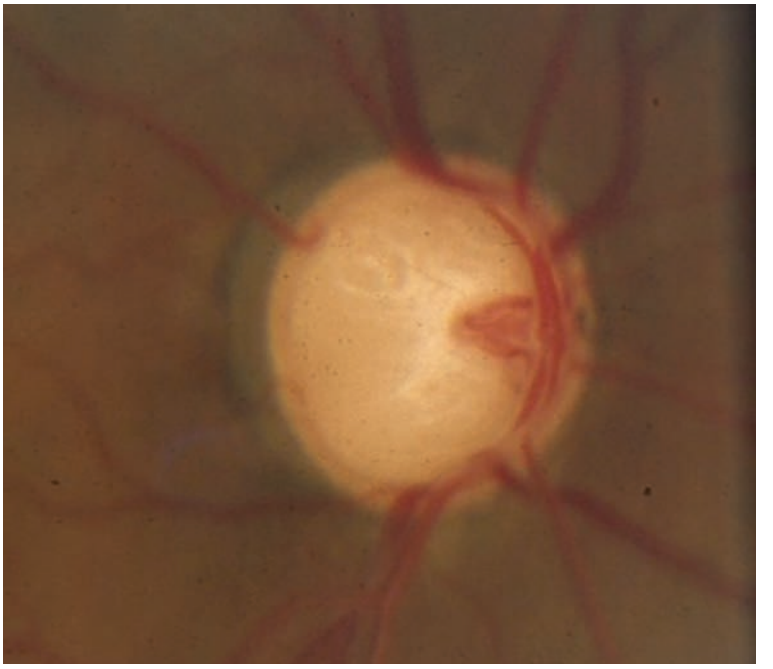
Coming Attractions

Are exciting new glaucoma therapies coming soon to a pharmacy near you?

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

We have all gone to a movie theater and sat through the coming attraction prior to the feature presentation. Some of these clips are quite enticing and leave us anticipating the release of promising entertainment. Similarly, the promise of new therapies fills us with excitement for our profession and patients.

This month, we present a few "coming attractions" in glaucoma pharmacology to pique your professional enthusiasm.



Advanced glaucomatous damage. New therapies currently being investigated may offer additional IOP-lowering options.

Rhopressa

Rhopressa (Aerie Pharmaceuticals) represents a new class of drug; namely, a Rho-kinase inhibitor. The Rho-kinase family consists of three guanosine triphosphate (GTP)-binding proteins (RhoA, RhoB, RhoC) that regulate aspects of cell shape, motility, proliferation and cellular apoptosis. Research shows inhibition of these kinases relaxes the trabecular meshwork tissues, leading to an enhancement of trabecular aqueous outflow and subsequent IOP reduction.[1]

Rhopressa, if approved, would be a once-daily product that specifically targets the trabecular meshwork to increase aqueous outflow through this conventional pathway. Currently, only the poorly tolerated miotics exploit this IOP reduction mechanism. Rhopressa is considered "triple action," as preclinical results demonstrate not only an enhancement of the trabecular outflow, but also a reduction in episcleral venous pressure (which directly contributes to overall IOP levels) and some degree of aqueous production inhibition.[2]

Despite the seeming potential of this new compound, there have been bumps along the approval road. In a study comparing the compound in Rhopressa (AR-13324 0.02%) with latanoprost, investigators found Rhopressa less effective, by approximately 1mm Hg in patients with elevated IOP.[3]

Additionally, as explained in a press release, Rhopressa did not meet the primary efficacy endpoint of demonstrating non-inferiority to twice-daily dosing of timolol. Rhopressa also showed a slight loss of efficacy over time. However, studies of Rhopressa are ongoing and include a 12-month safety trial with a 90-day interim efficacy assessment and a safety-only study being conducted in Canada.

Roclatan

Another promising agent, Roclatan (Aerie Pharmaceuticals) is a fixed combination agent composed of Rhopressa and latanoprost 0.005%. With the addition of latanoprost to the mix, Roclatan claims to possess "quadruple action," adding increased uveoscleral

outflow to the mechanisms reported by Rhopressa alone. A double-masked, randomized, parallel comparison study compared the IOP-reducing efficacy of the fixed combination Roclatan with its individual components in patients with open-angle glaucoma or ocular hypertension.

The results indicate Roclatan achieved statistical superiority to both latanoprost and Rhopressa, lowering IOP by an additional 1.9mm Hg and 2.6mm Hg, respectively.[4]

Vesneo

Vesneo (latanoprost bunod, Bausch + Lomb) is a novel nitric oxide-donating prostaglandin F2-alpha analog licensed by Nicox and currently in Phase III clinical development.

In Phase III studies, Vesneo reached its desired primary endpoint of non-inferiority to timolol maleate 0.5% BID, actually showing superiority to the beta-blocker. Vesneo showed a reduction in mean IOP of 7.5mm Hg to 9.1mm Hg from a baseline between two and 12 weeks through Phase III studies.

The VOYAGER study shows latanoprost bunod 0.024% dosed once daily lowered IOP to a significantly greater degree—and had comparable side effects—relative to latanoprost 0.005%. The most common side effect was hyperemia, which was well tolerated.[5]

Trabodenoson

The compound trabodenoson (Inotek Pharmaceutical) is also in Phase III clinical trials. This compound is a selective adenosine mimetic whose action appears to increase trabecular aqueous outflow.

Trabodenoson stimulates adenosine receptors, increasing metabolic activity in the trabecular meshwork. The increased metabolic activity upregulates proteases that digest and remove accumulated proteins which hinder trabecular aqueous outflow.[6] Phase II studies show trabodenoson has a duration of action that may make QD dosing possible. Alone, it approximates the IOP lowering efficacy of prostaglandin analogs.

It also appears to have an additive effect to other, second-line glaucoma medications such as beta-blockers and carbonic anhydrase inhibitors.

Taptiqom

One company may attempt to bring a fixed combination prostaglandin analog/beta-blocker to the

United States. The agent, Taptiqom (Santen Pharmaceuticals), combines tafluprost 0.0015% with timolol 0.5% in a preservative-free, unit-dose vial. Early studies are promising. One FDA-approval challenge for fixed combination agents is its inability to demonstrate a significant and sustained efficacy compared with the individual components. A six-month, prospective, randomized, double-masked, parallel group, multicenter Phase III study was performed in patients with ocular hypertension and open-angle glaucoma comparing the fixed combination agent with concomitant use of both components.

The preservative-free tafluprost/timolol fixed combination showed IOP reductions that were both statistically and clinically significant, and non-inferior to those of the concomitant usage of the individual components.

In fact, the fixed combination agent outperformed the concomitant use of both products.[7]

Similarly, in a study involving exfoliative glaucoma patients, the fixed combination agent performed similarly to the two agents used concomitantly.[8]

In the past several years, no major developments in topical glaucoma therapy, save the creation of fixed combination agents involving long-used components, have hit the market. These "teasers" promise exciting new innovations in fixed combination agents as well as entirely new medication classes. It remains to be seen if they portend any "blockbusters." ■

Drs. Sowka and Kabat have no financial interest in any company or product mentioned.

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Feel the Burn

Can intense pulsed light therapy help your patients with dry eye due to MGD?

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

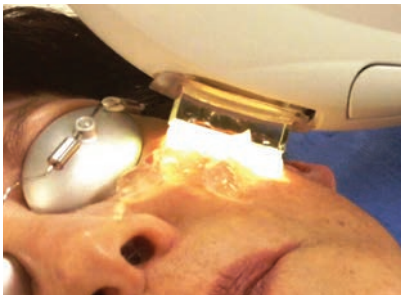
Surgical eye care is not often undertaken by choice. LASIK is the marquee item in elective eye surgery, and cataract patients can incorporate refractive correction into the surgical protocol. Most others would consider surgery only as a last resort. But now, some dry eye patients can choose to augment their traditional management approach of lid hygiene and topical therapy with a procedure that isn't quite surgery, done with a device that isn't quite a laser.

Intense pulsed light (IPL) therapy is used in dermatology to treat skin conditions such as rosacea and as a cosmetic enhancement for the removal of hair, vascular lesions and pigmented lesions. IPL is also popular for creating smoother, blemish-free skin.

Recently, we have heard many doctors performing IPL report that patients claim to have improved dry eye symptoms after treatments. IPL has been described as a potentially beneficial treatment for ocular rosacea and dry eye related to meibomian gland dysfunction (MGD). A recently published study showed an increase in lipid layer grade and noninvasive tear break-up time, in addition to a decrease in patient symptoms compared to placebo.[1]

How it Works

IPL is a non-laser, broad wavelength, high intensity flash of light that is applied using a handpiece that contacts the skin through a sapphire or quartz block. The intensity, duration and wavelengths can all vary based on the patient's skin type and treatment goals.



A contact gel is used with IPL to increase transmission and decrease burn risk.

The specific mechanism of action is not well understood, but is believed to be partially due to the thermal heating of the meibum coupled with the therapeutic effects of treating superficial telangiectasia. Energy is absorbed by skin chromophores, leading to lysis, causing only minimal collateral damage to neighboring cells.

The block heats up significantly during treatment, so many systems use a refrigerant to cool the block between pulses, lessening the risk of burn and improving comfort.

The only significant risk with IPL is the use of excessive amounts of energy, which can cause permanent skin depigmentation. While not a serious medical problem, it can be a permanent aesthetic concern.

IPL for MGD

Although there is no well-accepted method of treatment, most clinics will apply treatment to both cheek areas encroaching onto the lower eyelid, but not touching the eyelid margin. The treatment usually causes mild discomfort, and redness can persist for days before full resolution. Anywhere between three to seven treatments per year are usually recommended.

Many clinics are choosing to manually express the meibomian glands (MGs) immediately after IPL treatments to evacuate the glands and stimulate new meibum production. Because of IPL's immediate heating effects, the glands are usually easier to express. After MG expression, patients may notice mild lid tenderness and swelling for several days, but it is rarely severe and always self-limiting.

Good candidates for IPL therapy have any combination of the following: dry eye symptoms related to MGD, eyelid or dermal telangiectasia, non-expressive MGs or poor tear lipid layer. It is important to counsel patients that this is not a cure, but an advanced management therapy. Although increased MG expression is often seen after a single treatment, multiple treatments are usually recommended over several months. However, long-term stability of MG improvement has not yet been established, and IPL may evolve into a Botox-like procedure that requires consistent reapplication to maintain effect. ■

1. Craig JP, Chen YH, Turnbull PR. Prospective trial of intense pulsed light for the treatment of meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2015 Feb;56(3):1965-70.

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■ **24-27.** *2015 WOA Convention & Annual Meeting*. Kalahari Resort & Conference Center, Wisconsin Dells, WI. Hosted by: Wisconsin Optometric Association. CE hours: 26 total; 22 per OD. To register, email Joleen Breunig at joleen@woa-eyes.org or go to www.woa-eyes.org.

■ **25-27.** *NOA Fall Convention*. Younes Conference Center, Kearney, NE. Hosted by: Nebraska Optometric Association. CE hours: 12. To register, email Alissa Johnson at noa@assocoffice.net or go to www.nebraska.aoa.org.

■ **25-27.** *2015 KOA Fall Conference*. Embassy Suites Hotel, Lexington, KY. Hosted by: Kentucky Optometric Association. CE hours: 20. To register, email Sarah Unger at sarah@kyeyes.org, call (502) 875-3516 or go to www.kyeyes.org.

■ **26-27.** *CE in Austin*. Omni Austin Hotel Downtown, Austin, TX. Hosted by: University of Houston College of Optometry. Key faculty: Pat Segu. CE hours: 16. To register, email optce@uh.edu or go to ce.opt.uh.edu/.

■ **26-27.** *Forum on Optometry*. Marriott Hotel, Mystic, CT. Hosted by: PSS EyeCare. Key faculty: Deepak Gupta, Leonard Messner, Elliott Kirstein, William Jones, Kristen Brown. CE hours: 18. To register, go to www.psseyecare.com.

■ **26-28.** *CE in Italy*. Castiglion Fiorentino, Tuscany, Italy. Hosted by: James Fanelli. Key faculty: James Fanelli, Joseph Pizzimenti. CE hours: 12. To register, email James Fanelli at jamesfanelli@CEinItaly.com or go to www.CEinItaly.com/.

■ **30-Oct. 2.** *CE in Italy*. San Domenico Palace on the Sea, Taormina, Sicily. Hosted by: James Fanelli. Key faculty: James Fanelli, Joe Pizzimenti. CE hours: 12. To register, email James Fanelli at jamesfanelli@CEinItaly.com or go to www.CEinItaly.com/.

October 2015

■ **1-4.** Kansas Optometric Association Fall Eyecare Conference. DoubleTree by Hilton Wichita Airport, Wichita, KS. Hosted by: Kansas Optometric Association. CE hours: 13. To register, email Todd Fleischer at todd@kansasoptometric.org, or go to www.kansasoptometric.org.

■ **1-4.** *EastWest Eye Conference*. Cleveland Convention Center, Cleveland. Hosted by: Ohio Optometric Association. CE hours: 250+; 27 per OD. To register, email Linda Fette at linda@ooa.org, call (800) 999-4939 or go to www.eastwesteye.org.

■ **5-6.** *AFOS at Academy 2015*. New Orleans Marriott, New Orleans. Hosted by: Armed Forces Optometric Society. CE hours: 6. To register, email Gina Borgognoniv at execdir@afos2020.org or go to www.afos2020.org.

■ **6.** *Optometric Glaucoma Society Annual Scientific Meeting*. New Orleans Morial Convention Center, New Orleans. Hosted by: OGS & AAO. CE hours: 8. To register, email Michael Chaglasian at MChaglas@ico.edu or go to www.optometric-glaucomasociety.org.

■ **6.** *ONS Fall 2015 Educational Symposium*. New Orleans Morial Convention Center, New Orleans. Hosted by: Ocular Nutrition Society. CE hours: 6. To register, email info@ocular-nutrtionsociety.org, call (800) 383-1202 or go to www.ocular-nutritionsociety.org.

■ **6.** *OCRT 12th Annual Education Symposium*. New Orleans Morial Convention Center, New Orleans. Hosted by: Optometric Council on Refractive Technology. Key faculty: Paul Karpecki, David Geffen, Tracy Swartz, Chris Freeman. CE hours: 8. To register, email jcfreeopt@yahoo.com or go to www.ocrt.org.

■ **7-8.** *IOA Fall Seminar*. Indiana Memorial Union, Bloomington, IN. Hosted by: Indiana Optometric Association. CE hours: 14. To register, email Bridget at blsims@ioa.org or go to www.ioa.org.

■ **7-10.** *Academy 2015*. New Orleans Morial Convention Center, New Orleans. Hosted by: American Academy of Optometry. CE hours: 300+; 35 per OD. To register, email Helenv@aaoptom.org or go to www.aaopt.org.

■ **10-11.** *Forum on Ocular Disease*. Swan and Dolphin hotel, Orlando, FL. Hosted by: PSS EyeCare. Key faculty: Ron Melton, Randall Thomas, Deepak Gupta, Jerome Sherman. CE hours: 18. To register, email education@psseyecare.com or go to www.psseyecare.com.

■ **11.** *Pediatrics & Low Vision Course*. Marshall B. Ketchum University, Fullerton, CA. Hosted by: SCCO at Marshall B. Ketchum University. Key faculty: Carmen Barnhardt, Sue Cotter, Lynn Lowell, John Tassinari. CE hours: 8. To register, email Antoinette Smith and Bonnie Dellatorre at ce@ketchum.edu, call (714) 449-7495 or go to www.ketchum.edu/ce.

■ **13-14.** *Michigan Optometric Association 47th Annual Fall Seminar*. Lansing Center, Lansing, MI. Hosted by: Michigan Optometric Association. CE hours: 12 to 14. To register, email Amy Root at amy@themoa.org, call (517) 482-0616 or go to www.themoa.org.

■ **15-18.** *MOA Annual Conference, Trade Show & Golf*

Tournament. Downtown Marriott, Kansas City, MO. Hosted by: Missouri Optometric Association. CE hours: 16. To register, email Sue Brown at sue@moeyecare.org, call (573) 635-6151 or go to www.moeyecareconference.org.

■ **16-25.** *Classic China 2015*. Beijing, Xi'an, Shanghai, China. Hosted by: iTravel CE. Key faculty: John McGreal. CE hours: 16. To register, email info@iTravelCE.com or go to www.iTravelCE.com.

■ **17.** *San Francisco Optometric Glaucoma Symposium*. Marriott Union Square, San Francisco, CA. Hosted by: Review of Optometry. Key faculty: John Flanagan, Andrew Iwach. CE hours: 6. To register, go to www.reviewofoptometry.com/conferences/.

■ **23-25.** *GOA Fall Education Conference*. UGA Hotel and Conference Center, Athens, GA. Hosted by: Georgia Optometric Association. CE hours: 18. To register, email Vanessa Grosso at VanessaGOA@aol.com, call (770) 961-9866 ext. 1 or go to www.GOAeyes.com.

■ **24-25.** *VOA 2015 Fall Conference*. Kingsmill Resort, Williamsburg, VA. Hosted by: Virginia Optometric Association. CE hours: 8. To register, email office@thevoa.org, call (804) 643-0309 or go to www.thevoa.org.

■ **24-25.** *CE in Fort Worth*. Marriott Dallas/Fort Worth Hotel & Golf Club at Champions Circle, Fort Worth, TX. Hosted by: University of Houston College of Optometry. Key faculty: Suzanne Wickum. CE hours: 16. To register, email optce@uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu/.

■ **24-25.** *Primary EyeCare Conference*. Renaissance hotel, Westchester, NY. Hosted by: PSS EyeCare. Key faculty: Deepak Gupta, Mile Brujic, Kimberly Reed. CE Hours: 16. To register, email education@psseyecare.com or go to www.psseyecare.com.

■ **24-26.** *Annual Education Conference*. Mystic Marriott Hotel and Spa, Groton, CT. Hosted by: Connecticut Association of Optometrists. CE hours: 18. To register, email Stephanie Bartos at sbartos@cteyes.org, Lynn Sedlak at lsedlak@cteyes.org, call (860) 529-1900 or go to www.cteyes.org.

■ **30-Nov. 1.** *26th Annual Education Conference*. Abe Martin Lodge, Nashville, IN. Hosted by: Fellowship of Christian Optometrists, International. CE hours: 12. To register, email Kelly Frantz at kfrantz@ico.edu, call (312) 949-7281 or go to www.fcoint.net. ■

To list your meeting, please send the details to:

Rebecca Hepp, Senior Associate Editor
Email: rhepp@jobson.com
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12th Annual Education Symposium

Cornea, Cataract and Refractive Surgery Symposium

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The Meeting of the year for OD's involved in cornea, cataract and refractive eye care and doctors interested in innovative technologies

OCTOBER 6, 2015

New Orleans Convention Center • Room 220-221 • New Orleans, LA

SCHEDULE

8:00am - 11:00am	**CE Program**
11:00am - 11:15am	Break
11:15am - 12:15pm	**CE Program**
12:15pm - 1:15pm	Lunch
1:15pm - 3:15pm	**CE Program**
3:15pm - 3:30pm	Break
3:30pm - 5:30pm	**CE Program**

- The Optometric Council on Refractive Technology will sponsor it's 12th annual education symposium, bringing together the most notable experts in the field of cornea, cataract, and refractive technology to discuss evolving clinical innovations and management of ocular surface disease and other anterior segment complications
- The meeting is held the day before the American Academy of Optometry meeting in New Orleans, LA. This interactive meeting encourages questions, comments, and audience participation with panel discussions.
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Could it Be the Glaucoma?

By Andrew S. Gurwood, OD

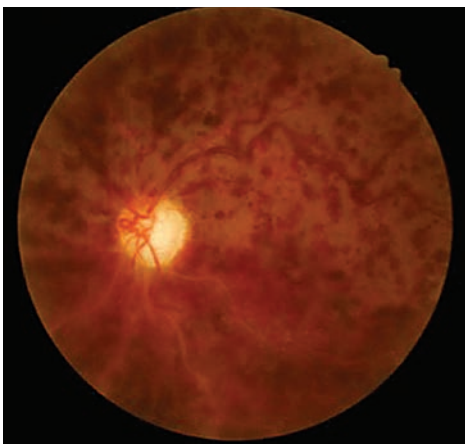
History

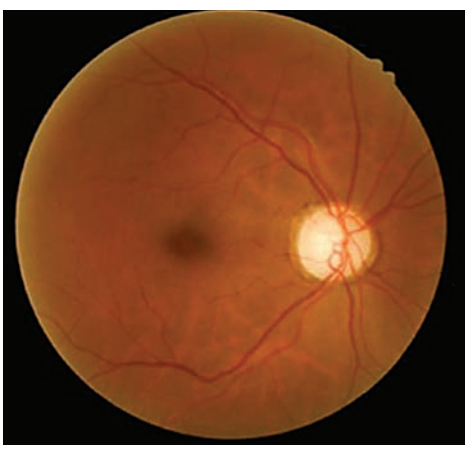
A 63-year-old black female presented with history of headache and decreased vision in her left eye for two weeks. Pertinent medical history included borderline diabetes managed by diet, and hyperlipidemia and hypertension controlled with medications. Her ocular history included a diagnosis of primary open-angle glaucoma suspect secondary to increased cup-to-disc ratios.

Diagnostic Data

Her best-corrected acuities measured 20/20 OD and 20/60 OS. External examination was normal with no afferent defect. Peripheral confrontational fields were normal in both eyes. Central facial Amsler grid was distorted in her left eye. Biomicroscopy demonstrated normal anterior segment tissues with no evidence of pigmentary dispersion (PDS), neovascularization of the iris (NVI) or keratic precipitates (KP), in either eye. Intraocular pressures (IOP) measured 29mm Hg OD and 18mm Hg OS with Goldmann applanation tonometry. The pertinent posterior segment findings are demonstrated in the photographs.

Additional testing included gonioscopy to inspect the angle for neovascularization or additional pathology related to increased cupping and glaucoma. The test revealed open angles with no synechiae, neovascularization, exfoliation or angle recession. The angle was open to the trabecular meshwork 360 degrees around with 2+ pigment in both eyes. Additionally, photodocumentation of both nerves and fundi was completed.





Using our 63-year-old patient's fundus images as well as her medical history and test results, can you determine her diagnosis?

A laboratory work up was suggested to the general practitioner to rule out undiscovered systemic causes for the fundus pathology and a full glaucoma workup in both eyes (perimetry, central corneal thickness and structural testing) was scheduled. Blood pressure (BP) was also measured in each arm and found to be 158/90. Additional history uncovered that the patient had self-discontinued her systemic hypertension medication for one month's duration.

Your Diagnosis

Does this case require any additional tests? Are you able to determine a diagnosis? How would you manage this patient? What is the likely prognosis?

To find out, please visit *Review of Optometry* online at www.reviewofoptometry.com.

Retina Quiz Answers *(from page 119):* 1) d; 2) c; 3) d; 4) b.

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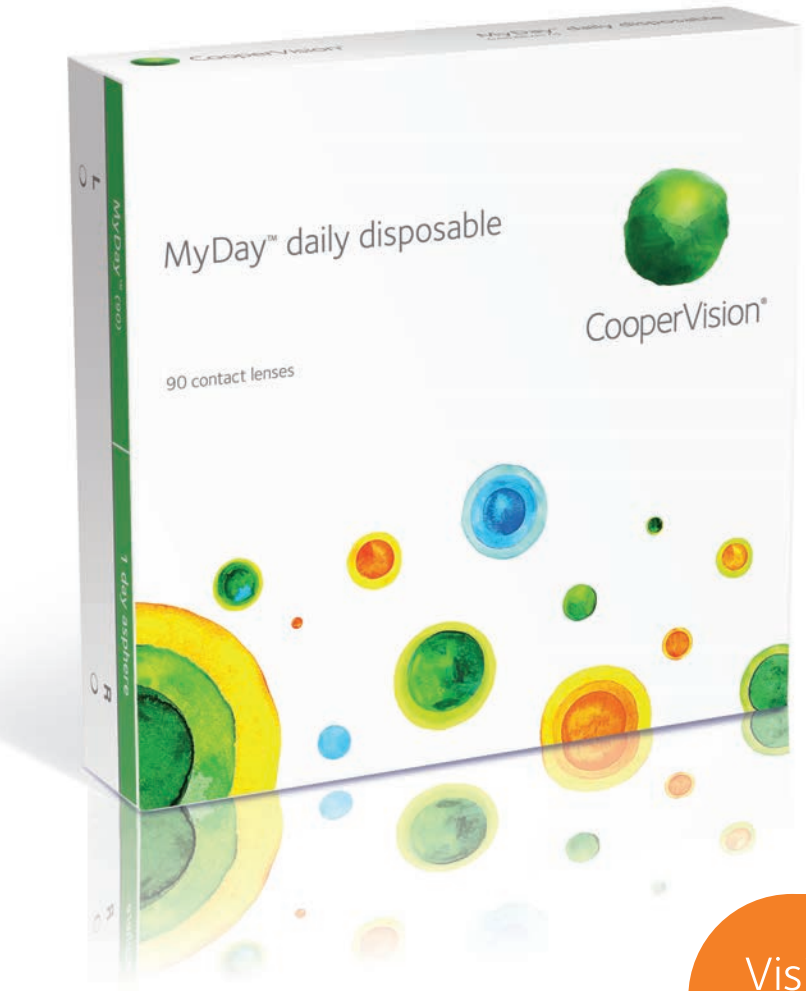

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[1]Gabriel M, Bartell J, Walters R, et al. Biocidal efficacy of a new hydrogen peroxide contact lens care system against bacteria, fungi, and Acanthamoeba species. Optom Vis Sci. 2014; 91: E-abstract 145192.