Pain Medication Protocols, p. 92 • The Battle for Corneal Clarity, p. 102

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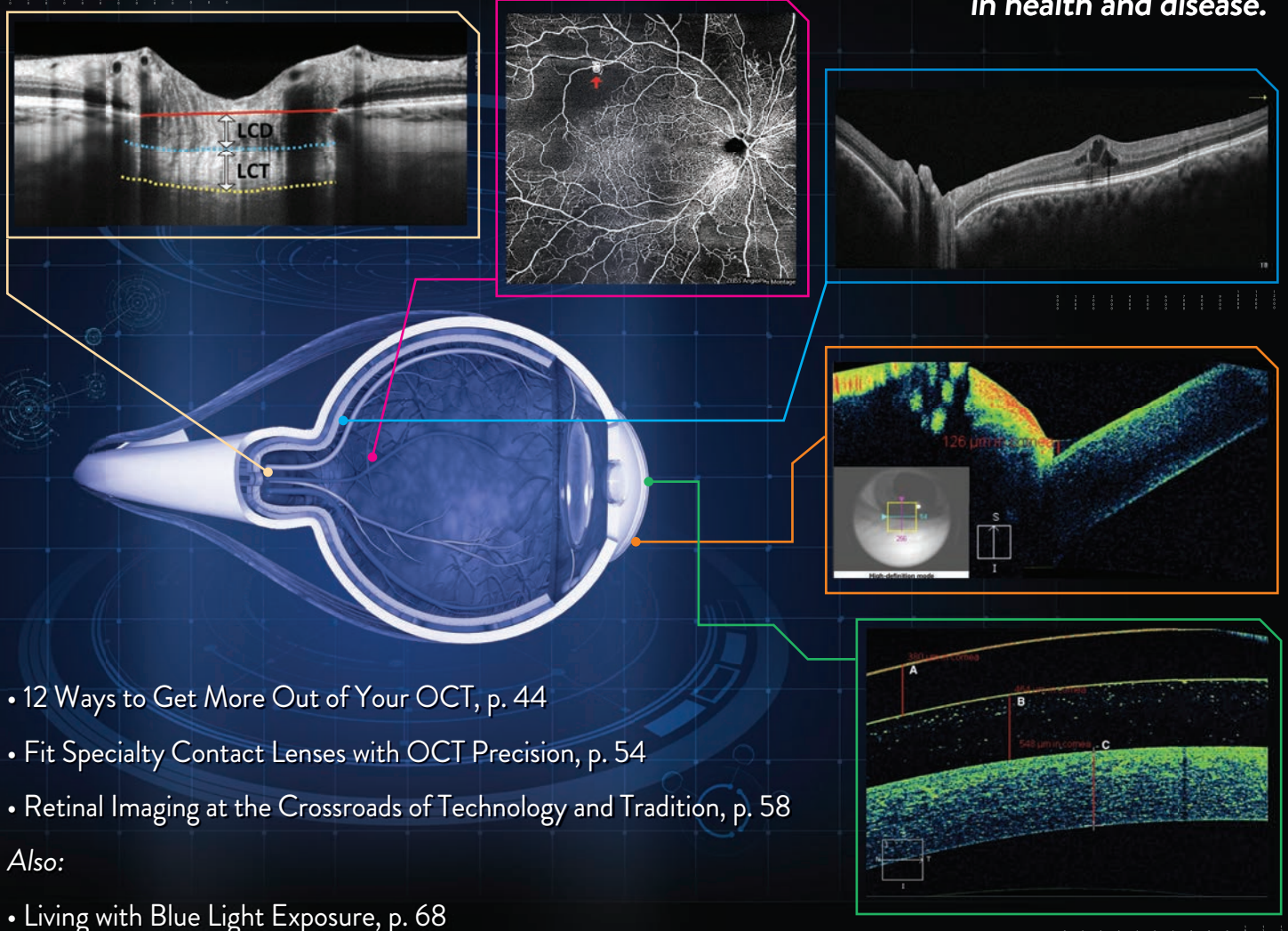

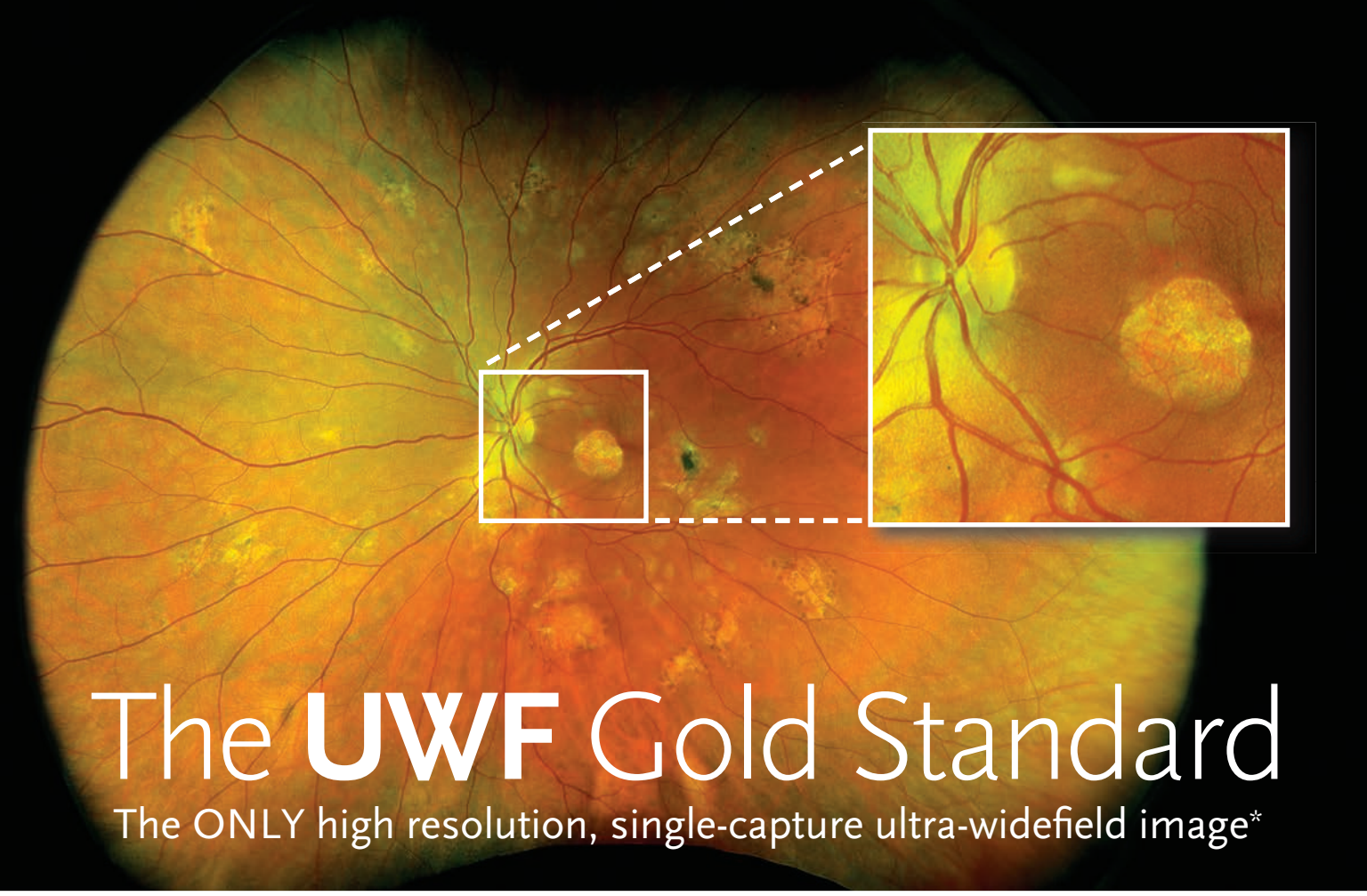

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*Classification & Guidelines for Widefield Imaging Recommendations from the International Widefield Imaging Study Group. Ophthalmology Retina. 2019.

VOL. 156 NO. 9 ■ SEPTEMBER 15, 2019

IN THE NEWS

Chinese investigators recently found that fluctuating IOP is a significant and independent predictor for subsequent visual field (VF) deterioration in patients with primary angle-closure disease. The researchers discovered that greater IOP fluctuation was significantly associated with VF deterioration, independent of mean IOP. High-threshold mean IOP and high-threshold IOP fluctuation had the most rapid VF deterioration.

Cheung CY, Li SL, Chan PPM, et al. Intraocular pressure control and visual field changes in primary angle closure disease: the CUHK PACG Longitudinal (CUPAL) study. Br J Ophthalmol. August 7, 2019. [Epub ahead of print].

From in-person interviews, a study in Maryland found **glaucoma patients were concerned with limitations in performing specific vision-dependent activities, visual function problems, treatment burden and intraocular pressure when considering new treatments**. Researchers think this decision checklist may be useful in future evaluations of new treatments.

Le JT, Mohanty K, Bicket AK, et al. Identifying outcomes that are important to patients with ocular hypertension or primary open-angle glaucoma: a qualitative interview study. Ophthalmology Glaucoma. July 31, 2019. [Epub ahead of print].

One hour of nocturnal artificial blue light induces an increase of sucrose intake and a decrease in plasma insulin concentration in rats. In chow-fed rats, the glucose tolerance test results were higher for those exposed to blue light. Researchers believe that these findings could possibly indicate the harmful effects regarding human artificial blue light exposure from devices and screens.

Masis-Vargas A, Hicks D, Kalsbeek A, et al. Acute exposure to blue light at night impairs glucose tolerance, alters insulin secretion and increases sugar intake in a diurnal rodent. Oral presentation at: Annual Meeting of the Society for the Study of Ingestive Behavior; July 2019; Ultrecht, Netherlands.

Nomogram for Uveal Melanoma Predicts VA

Using point system can better determine risk of poor outcomes.

By Catherine Manthorp, Associate Editor

Researchers recently developed a nomogram that may help clinicians predict visual acuity outcomes for patients with uveal melanoma undergoing plaque radiotherapy and prophylactic intravitreal anti-VEGF.

This retrospective review, by ocular oncology experts at Wills Eye Hospital in Philadelphia and the Mayo Clinic of Rochester, MN, included 1,131 cases that were treated at four-month intervals for two years. The team used two point systems for visual acuity outcome—one with clinical risk factors and the other with both clinical and treatment risk factors.

They found that the most important clinical risk factors—ranked with a point system—for poor visual acuity included:

- subretinal fluid involving four quadrants (100pts)
- tumor thickness >4mm (69pts)
- presenting visual acuity ≤20/30 (65pts)
- non-Caucasian race (58pts)
- mushroom-shaped and bilobed or multilobulated tumor (57pts)
- insulin-dependent diabetes (54 pts)

The researchers note that the risk of poor visual acuity at two and four years increased from 11% and 24%, respectively, with 40 points to 97% and >99%, respectively, with 304 points.

The second nomogram included both clinical and treatment risk factors, with differing points:

- presenting visual acuity ≤20/30 (100pts)
- tumor largest basal diameter >11mm (80pts)
- radiation dose rate to tumor base ≥164cGy/hour (78pts)
- tumor thickness >4mm (76pts)
- insulin-dependent diabetes (75pts)
- abnormal foveolar status by optical coherence tomography at presentation (72pts)

When using this system, the risk of poor visual acuity at two and four years increased from 6% and 14%, respectively, with 56 points, to 88% and 99%, respectively, with 496 points.

The nomogram is available online at fighteyecancer.com/nomograms/.

Dalvin LA, Zhang Q, Hamershock RA. Nomogram for visual acuity outcome after iodine-125 plaque radiotherapy and prophylactic intravitreal bevacizumab for uveal melanoma in 1131 patients. Br J Ophthalmol. August 13, 2019. [Epub ahead of print].

NEWS STORIES POST EVERY WEEKDAY MORNING AT www.reviewofoptometry.com/news

Tinted Glasses No Help in Night Driving

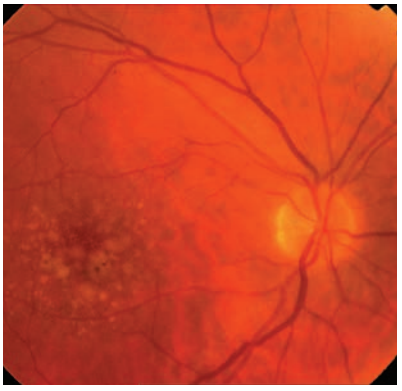
Researchers determined the same effect as wearing sunglasses.

A study in *JAMA Ophthalmology* reports tinted lenses don't improve road visibility or diminish glare and halos and may actually worsen visibility in some cases.

Yellow, red and blue lenses all cut out a portion of light, which basically equates to wearing sunglasses when driving at night, says lead study investigator Alex Hwang, PhD, professor at the Harvard Medical School Department of Ophthalmology.

While people who wear the yellow-lens night driving glasses may feel as though they are able to see more "brightly," their vision is not really improved, he adds. In fact, this perception may actually make overall night driving riskier because the wearer may be overconfident about their night vision, Dr. Hwang says.

The study, conducted at the Schepens Eye Research Institute in Massachusetts, included 22 patients who operated a driving simulator that included the option of bright oncoming headlights. Each participant drove scripted night-driving scenarios three times with three commercially available yellow-lens glasses and once with clear-lens glasses. The study included eight different night-driving conditions with the bright headlight option turned on and off.



Glare recovery time from headlights could be worse in drivers with AMD.

The first group consisted of 12 younger participants—between the ages of 27 and 28—who responded when they saw a pedestrian in a navy blue shirt, and the second group was comprised of six younger and four older subjects who responded when they saw a pedestrian wearing an orange shirt. All participants had normal visual acuity.

The study found yellow-lens night-driving glasses did not appear to improve pedestrian detection at night or reduce the negative association between headlight glare and pedestrian detection. Researchers also found no difference in pedestrian detection with the yellow lenses.

Investigators observed younger participants were impacted most by headlight glare with the navy blue shirt pedestrian and older patients with the orange shirt pedestrian. Additionally, the study found older participants had a greater difference in response times with or without the headlight glare (1.5 seconds) compared with younger participants (0.3 seconds).

Dr. Hwang suggests eye care practitioners advise their patients not to wear night driving glasses. These glasses should be used during the daytime only, just like any other sunglasses, he says.

He adds a warning for patients: no magic glasses exist that make night driving safer or reduce oncoming headlight glare. If patients feel like headlight glare is increasing or are bothered by it while driving at night, they should see their eye doctor, who should check for cataracts and age-related macular degeneration (AMD). Dr. Hwang stresses the importance of patients seeing an eye care practitioner in these instances since cataracts increase light scatter, which, in turn, increase the negative glare effect. AMD increases glare recovery time, so patients can be impacted by headlight glare even after the oncoming light has passed.

Hwang AD, Tuccar-Burak M, Peli E. Comparison of pedestrian detection with and without yellow-lens glasses during simulated night driving with and without headlight glare. JAMA Ophthalmol. August 1, 2019. [Epub ahead of print].

Corrections

On page 44 of the July 15, 2019 print edition, Table 3 should include "Recommend vitamin supplementation with AREDS 2 formulation" for a patient with intermediate AMD.

On page 78 of the August 15, 2019 print edition, bleach should be identified as sodium hypochlorite, not sodium hydroxide. On page 79, Sterilid Antimicrobial (Akorn) should list a 24-month, not 24-hour, shelf life.

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Ocular Infection Causes Revealed in Lab Cultures

Pathogen trends could shed light on future antimicrobial treatments.

Researchers from the University of Pittsburgh may have unearthed the predominant underlying causes of three ocular infections and, in turn, created a roadmap for the development of new antimicrobials to treat these diseases. Their study combed through more than 3,000 cases of keratitis, endophthalmitis and conjunctivitis gathered over a 26-year period and looked for the prevalence of bacteria, fungi, viruses and *Acanthamoeba* in the collective samples.

Since 1993, investigators at the school's Charles T. Campbell Eye Microbiology Laboratory have stocked pathogens and patient samples isolated for keratitis, conjunctivitis and endophthalmitis to explore new tests and treatments.

The study found the following ocular pathogen trends:

- ***Keratitis:*** Researchers analyzed 1,387 samples that were gathered from 2004 to 2018. They reported bacteria was the main infectious agent at 72.1% (*Staphylococcus aureus* 20.3%, *Pseudomonas aeruginosa* 18%, *Streptococcus spp*. 8.5%, other gram-positives 12.4% and other gram-negatives 12.9%), followed by herpes simplex virus at 16%, fungi at 6.7% and *Acanthamoeba* at 5.2%.
- ***Endophthalmitis:*** Out of the 770 endophthalmitis cases gathered from 1993 to 2018, the study found coagulase-negative *Staphylococcus* at 54%, *Streptococcus spp*. at 21%, *S. aureus* at 10%, other gram-positives at 8% and other gram-negatives at 7%.
- ***Conjunctivitis:*** This collection included 847 conjunctivitis cases gathered from 2004 to 2018. Investigators noted the following distributions: adenovirus at 34%, *S. aureus* at 25.5%, *Streptococcus pneumoniae* at 9%, *Haemophilus* at 9%, other gram-negatives 8.8%, other gram-positives 6%, coagulase-negative *Staphylococcus* at 4.5% and *Chlamydia* at 3.2%.

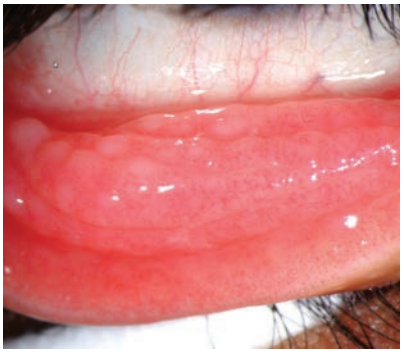

Photo: Marc Bloomenstein, OD

The majority of samples taken from conjunctivitis cases was from *S. aureus*.

The prevalence of ocular pathogens as infections of keratitis, endophthalmitis and conjunctivitis/blepharitis have never been reported in relation to each other (bacteria, viruses, fungi and parasites), says investigator Regis P. Kowalski, MS, M(ASCP), who is executive director of the lab. "Our data creates an awareness of the different infectious etiologies, the importance of laboratory studies and future treatment requirements for infectious ocular disease," Dr. Kowalski says.

Kowalski RP, Nayyar SV, Romanowski EG, et al. The prevalence of bacteria, fungi, viruses and *Acanthamoeba* from 3,004 cases of keratitis, endophthalmitis, and conjunctivitis. Eye & Contact Lens. August 1, 2019. [Epub ahead of print].

VF Progression in Well-controlled IOP

Researchers at the Duke Eye Center in Durham, NC, have found that about a quarter of eyes with well-controlled IOP can still show glaucomatous visual field (VF) progression over time. They cited thin central corneal thickness (CCT) and corneal hysteresis (CH) as major risk factors.

The study analyzed 179 eyes that had a mean follow-up of 4.3±0.8 years and IOP less than 18mm Hg. Of these eyes, 42 demonstrated VF progression. Researchers found no significant difference between progressing and stable patients in baseline mean deviation, mean IOP, IOP fluctuation or peak IOP. However, they found that progressing eyes had significantly lower CH and thinner CCT compared with stable eyes. Having these factors resulted in a 68% higher risk of progression.

Studying corneal biomechanics can reveal a better picture of VF progression, researchers said.

Susanna BN, Ogata NG, Jammal AA, et al. Corneal biomechanics and visual field progression in eyes with seemingly well-controlled intraocular pressure. Ophthalmology. August 9, 2019. [Epub ahead of print].

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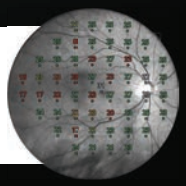

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2019 Income in *Review*

Like any other year, 2018 was full of highs and lows for optometry, income-wise. Has this year been any better? ***We want to hear how you've been doing financially in 2019.***

While earnings decreased among those with the most experience and the gap widened between self-employed and employed ODs last year, average income increased, the mid-career plateau disappeared and the gender gap narrowed.

Will 2019 continue the positive trend? Last year, 56% of our survey respondents thought so, reporting that they expected an increase in income, while 36% didn't expect a change and only 8% were speculating a decrease. Here's your chance to find out if you were right!

If you're a practicing OD, please take a few minutes to respond to our annual income survey and share your financial experience over the last year with us. The results will be published anonymously in the December issue. All personal and financial information is confidential and used for no other purpose than this survey.

Just in case you need a little extra push, here's an incentive: upon completing all of the questions in the survey, you'll be entered to win a ***$100 American Express Gift Card***. It just takes a few minutes, as there are only a handful of questions. Thank you for your participation—we wouldn't know where the field stood financially without you!

Take the Survey

To participate in the survey, visit www.surveymonkey.com/r/2019incomesurvey or scan the QR code.

Repeat Vitrectomies Raise IOP in RD

When a patient undergoes pars plana vitrectomy (PPV), it is often to address a condition such as macular hole, macular pucker or vitreous hemorrhaging. In fact, it can address a slew of issues as benign as floaters and as severe as a retinal detachment.[1] But when the original procedure doesn't do the trick, patients are sometimes asked to return for a repeat surgery. In most cases, an additional procedure doesn't have a significant impact on intraocular pressure (IOP). But a new study is suggesting that repeat PPV can significantly raise IOP in patients who initially presented with retinal detachment (RD).[2]

Dutch researchers performed a retrospective study of 447 eyes that underwent PPV. They found that IOP increased by 3mm Hg after a PPV in patients who had an indication of retinal detachment but remained stable after PPV for other indications, including epiretinal membrane, macular hole and vitreous hemorrhage. At the end of the follow-up period, the number of IOP-lowering medications was significantly higher in all groups except those with macular holes. Also, the number of eyes that underwent glaucoma surgery was significantly higher compared with fellow eyes.

The researchers noted a significant association between the number of PPVs and the final IOP for those with retinal detachment and between the number of PPVs and glaucoma surgeries.

Since elevated IOP plays a crucial role in the development of glaucoma, clinicians should know the effect of PPV on IOP. Patients with or suspect for glaucoma are more prone to develop a higher IOP in the first month postoperative compared with patients not suspect for glaucoma.

Surgical Indications[1]

Pars plana vitrectomy is commonly recommended to patients with:

- Macular hole
- Macular pucker
- Vitreomacular traction
- Refractory macular edema
- Vitreous hemorrhage
- Tractional retinal detachment
- Rhegmatogenous retinal detachment
- Dislocated intraocular lens
- Refractory uveitis
- Retained lens material
- Intraocular foreign bodies
- Floaters
- Aqueous misdirection syndrome

1. Spirn M. Pars plana vitrectomy. Eyewiki – American Academy of Ophthalmology. eyewiki.aao.org/Pars_Plana_Vitrectomy. April 22, 2019. Accessed August 20, 2019.
2. Kovacic H, Wolfs R, Kılıç E, Ramdas W. The effect of multiple vitrectomies and its indications on intraocular pressure. BMC Ophthalmol. 2019;19(1):175.

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Contact Lens Care Advice Easily Missed

One third of wearers said they never heard any lens wear and care recommendations.

Even though daily lens disposal is becoming more commonplace, proper lens care remains an important part of safe and successful contact lens wear. The trouble is, at least one in three patients isn't getting the message, or so they say.

Two recent surveys looked at the state of contact lens care education; the first assessed patients' experiences with the recommendations they received from eye care providers while the second asked doctors to describe their methods for communicating wear and care recommendations to their patients. In all, 4,088 patients and 1,100 providers participated in the studies.

As reported in the CDC's Morbidity and Mortality Weekly Report, one third (32.9%) of contact lens wearers said they never heard any lens wear and care recommendations. Fewer than half (47.9%) recalled hearing their doctor recommend not sleeping in lenses, and only 19.8% reported being told to avoid topping off their contact lens solution.

Among doctors, a majority reported sharing recommendations "always or most of the time" at initial visits, regular checkups and complication-related visits. The most common recommendations doctors encouraged among patients were: complying with the recommended lens replacement schedules (mentioned at 85.0% of regular visits), not sleeping in lenses (79.0%) and not topping off solutions (64.4%).

"These findings can assist in the creation of health communication messages to help encourage eye care providers to communicate more effectively with their patients," the study authors wrote.

To improve patient retention of lens care advice, the American Optometric Association advocates using the "teach-back" method, wherein doctors ask their patients to reiterate care recommendations back to them. This confirm that the practitioners have communicated information in a way that patients understand. You can access the educational resource about the technique online.

The CDC also has resources available online to help bridge the education gap, such as the "Contact Lens Health Week Campaign Promotion Toolkit."

Konne NM, Collier SA, Spangler J, Cope JR. Healthy contact lens behaviors communicated by eye care providers and recalled by patients—United States, 2018. MMR Weekly 2019;68(32):693–7.

AI Matches ODs, MDs in Glaucoma Detection

A recent study found that the diagnostic performance of an artificial intelligence (AI) software platform was comparable with European optometrists and ophthalmologists when diagnosing glaucoma. The researchers believe that deep learning-based AI systems could provide specialist-level accuracy for screening and detecting glaucomatous optic neuropathy, potentially reduce the number of false positives and provide such analysis at very low costs.

The retrospective study compared the performance of AI software with 208 British optometrists and 243 European ophthalmologists (glaucoma specialists) in detecting glaucomatous optic neuropathy from 94 scanned stereoscopic photographic slides.

They found no statistically significant difference between the performance of the deep learning system and the practitioners. The software detected glaucomatous optic neuropathy with an accuracy of 83.4%, while optometrists and ophthalmologists had 80% and 80.5% accuracy, respectively. The AI system also had a higher intra-observer agreement than optometrists and ophthalmologists.

Researchers found that the AI software was fast at grading images, only taking seven seconds to grade each image (including the upload time). They think it is likely that the AI could be faster than humans at analyzing stereoscopic optic disc images, which could help reduce time and expenses for screening programs. The researchers note that further clinical and economic analysis is necessary before considering the integration of AI systems into real-world clinical practices. ■

Amador-Patarroyo MJ, Lin T, Meshi A, et al. Identifying the factors for improving quality of oral fluorescein angiography. Br J Ophthalmol. July 4, 2019. [Epub ahead of print].

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- 74% of LOTEMAX® SM patients were completely pain-free vs vehicle (49%) at Day 8 (N=371, *P*<0.0001)[1,3‡]

†Pooled analysis of Phase 3 clinical studies. **Study 1:** 29% LOTEMAX® SM (N=171) vs 9% vehicle (N=172). **Study 2:** 31% LOTEMAX® SM (N=200) vs 20% vehicle (N=199); *P*<0.05 for all.

‡Pooled analysis of Phase 3 clinical studies. **Study 1:** 73% LOTEMAX® SM (N=171) vs 48% vehicle (N=172). **Study 2:** 76% LOTEMAX® SM (N=200) vs 50% vehicle (N=199); *P*<0.05 for all.

Indication

LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38% is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

Important Safety Information

- LOTEMAX® SM, 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.
- 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 LOTEMAX® SM is used for 10 days or longer, IOP should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.

Important Safety Information (cont.)

- The use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation. In those with diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification such as slit lamp biomicroscopy and, where appropriate, fluorescein staining.
- 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.
- Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and may exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use. Fungal cultures should be taken when appropriate.
- Contact lenses should not be worn when the eyes are inflamed.
- There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

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

Please see brief summary of Prescribing Information on adjacent page.

References: 1. LOTEMAX SM Prescribing Information. Bausch & Lomb, Incorporated. **2.** Cavet ME, Glogowski S, DiSalvo C, Richardson ME. Ocular pharmacokinetics of submicron loteprednol etabonate ophthalmic gel, 0.38% following topical administration in rabbits. Poster presented at 2015 ARVO Annual Meeting; May 4, 2015; Denver, Colorado. **3.** Data on file. Bausch & Lomb, Incorporated.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38%

For topical ophthalmic use
Initial U.S. Approval: 1998

INDICATIONS AND USAGE

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

DOSAGE AND ADMINISTRATION

Invert closed bottle and shake once to fill tip before instilling drops. Apply one drop of LOTEMAX® SM into the conjunctival sac of the affected eye three times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

CONTRAINDICATIONS

LOTEMAX® SM, 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, in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

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

Cataracts: Use of corticosteroids may result in posterior subcapsular cataract formation.

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

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

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

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

Contact Lens Wear: Contact lenses should not be worn when the eyes are inflamed.

ADVERSE REACTIONS

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. Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera. There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

USE IN SPECIAL POPULATIONS

Pregnancy: Risk Summary: There are no adequate and well controlled studies with loteprednol etabonate in pregnant women. Loteprednol etabonate produced teratogenicity at clinically relevant doses in the rabbit and rat when administered orally during pregnancy. Loteprednol etabonate produced malformations when administered orally to pregnant rabbits at doses 4.2 times the recommended human ophthalmic dose (RHOD) and to pregnant rats at doses 106 times the RHOD. In pregnant rats receiving oral doses of loteprednol etabonate during the period equivalent to the last trimester of pregnancy through lactation in humans, survival of offspring was reduced at doses 10.6 times the RHOD. Maternal toxicity was observed in rats at doses 1066 times the RHOD, and a maternal no observed adverse effect level (NOAEL) was established at 106 times the RHOD. The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2 to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies. Data: *Animal Data.* Embryofetal studies were conducted in pregnant rabbits administered loteprednol etabonate by oral gavage on gestation days 6 to 18, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations at 0.1 mg/kg (4.2 times the recommended human ophthalmic dose (RHOD) based on body surface area, assuming 100% absorption). Spina bifida (including meningocele) was observed at 0.1 mg/kg, and exencephaly and craniofacial malformations were observed at 0.4 mg/kg (17 times the RHOD). At 3 mg/kg (128 times the RHOD), loteprednol etabonate was associated with increased incidences of abnormal left common carotid artery, limb flexures, umbilical hernia, scoliosis, and delayed ossification. Abortion and embryofetal lethality (resorption) occurred at 6 mg/kg (256 times the RHOD). A NOAEL for developmental toxicity was not established in this study. The NOAEL for maternal toxicity in rabbits was 3 mg/kg/day. Embryofetal studies were conducted in pregnant rats administered loteprednol etabonate by oral gavage on gestation days 6 to 15, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations, including absent innominate artery at 5 mg/kg (106 times the RHOD); and cleft palate, agnathia, cardiovascular defects, umbilical hernia, decreased fetal body weight and decreased skeletal ossification at 50 mg/kg (1066 times the RHOD). Embryofetal lethality (resorption) was observed at 100 mg/kg (2133 times the RHOD). The NOAEL for developmental toxicity in rats was 0.5 mg/kg (10.6 times the RHOD). Loteprednol etabonate was maternally toxic (reduced body weight gain) at 50 mg/kg/day. The NOAEL for maternal toxicity was 5 mg/kg. A peri-/postnatal study was conducted in rats administered loteprednol etabonate by oral gavage from gestation day 15 (start of fetal period) to postnatal day 21 (the end of lactation period). At 0.5 mg/kg (10.6 times the clinical dose), reduced survival was observed in live-born offspring. Doses ≥ 5 mg/kg (106 times the RHOD) caused umbilical hernia/incomplete gastrointestinal tract. Doses ≥ 50 mg/kg (1066 times the RHOD) produced maternal toxicity (reduced body weight gain, death), decreased number of live-born offspring, decreased birth weight, and delays in postnatal development. A developmental NOAEL was not established in this study. The NOAEL for maternal toxicity was 5 mg/kg.

Lactation: There are no data on the presence of loteprednol etabonate in human milk, the effects on the breastfed infant, or the effects on milk production. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for LOTEMAX® SM and any potential adverse effects on the breastfed infant from LOTEMAX® SM.

Pediatric Use: Safety and effectiveness of LOTEMAX® SM in pediatric patients have not been established.

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in the chromosomal aberration test in human lymphocytes, or *in vivo* in the mouse micronucleus assay. Treatment of male and female rats with 25 mg/kg/day of loteprednol etabonate (533 times the RHOD based on body surface area, assuming 100% absorption) prior to and during mating caused preimplantation loss and decreased the number of live fetuses/live births. The NOAEL for fertility in rats was 5 mg/kg/day (106 times the RHOD).

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

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Based on 9669600-9669700 Revised: 02/2019

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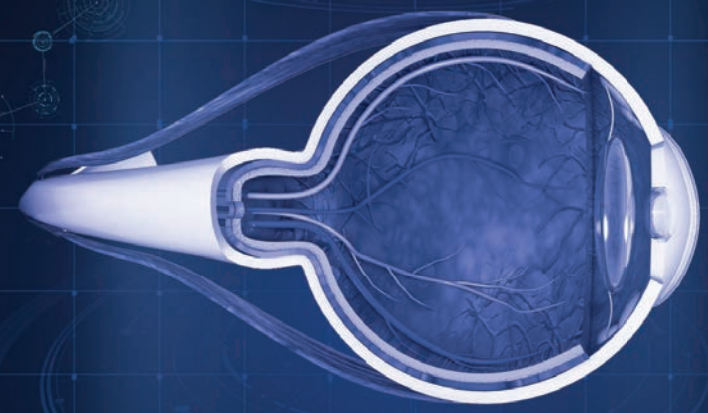

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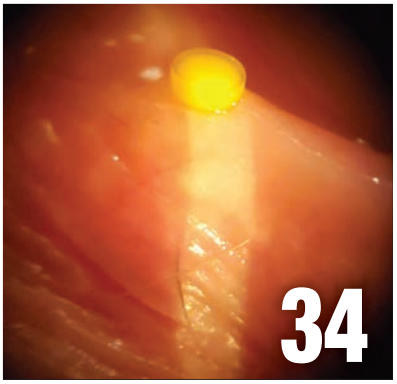

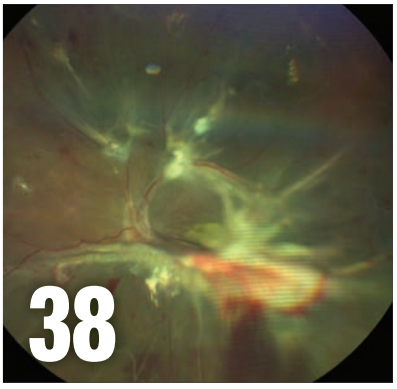

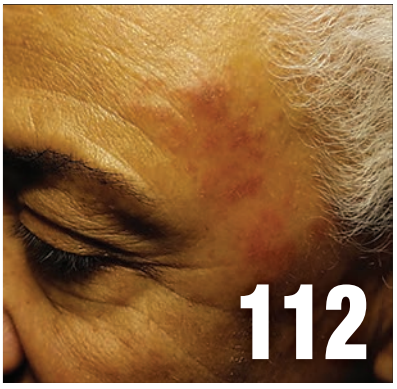

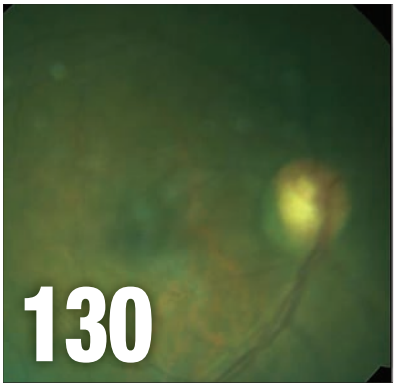

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There's no substitute.[2,4] Check out patient resources, insurance coverage, and more at **Xiidra-ECP.com**

References:

1. Xiidra [Prescribing Information]. Lexington, MA: Shire US. **2.** TFOS DEWS II Research Subcommittee. Report of the Research Subcommittee of the Tear Film & Ocular Surface Society Dry Eye WorkShop II (2017). *Ocul Surf.* 2017;15(3):269-649. **3.** FDA approves new medication for dry eye disease. FDA News Release. July 2016. http://www.fda.gov/newsevents/newsroom/pressannouncements/ucm510720.htm. Accessed July 12, 2016. **4.** Food and Drug Administration. Electronic Orange Book. http://www.fda.gov/downloads/Drugs/DevelopmentApprovalProcess/UCM071436.pdf. Accessed June 26, 2018.

Indication

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

Important Safety Information

Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients.

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

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

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

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

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

Shire

xiidra® (lifitegrast ophthalmic solution) 5%

Rx Only

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

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

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

CONTRAINDICATIONS
Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients in the formulation.

ADVERSE REACTIONS
Clinical Trials Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in clinical studies of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice. In five clinical studies of dry eye disease conducted with lifitegrast ophthalmic solution, 1401 patients received at least 1 dose of lifitegrast (1287 of which received lifitegrast 5%). The majority of patients (84%) had ≤3 months of treatment exposure. 170 patients were exposed to lifitegrast for approximately 12 months. The majority of the treated patients were female (77%). The most common adverse reactions reported in 5-25 % of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.

Postmarketing Experience
The following adverse reactions have been identified during postapproval use of Xiidra. 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.

Rare cases of hypersensitivity, including anaphylactic reaction, bronchospasm, respiratory distress, pharyngeal edema, swollen tongue, and urticaria have been reported. Eye swelling and rash have been reported.

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

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

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

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

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

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

Shire

Manufactured for: Shire US Inc., 300 Shire Way, Lexington, MA 02421.
For more information, go to www.Xiidra.com or call 1-800-828-2088.

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Outlook

By Jack Persico, Editor-in-Chief

A Recipe for Behavior Change

A cautionary tale about diet and the eye might convince patients to accept personal responsibility.

Those of us old enough to have lived through the 1970s can probably remember a novelty song called "Junk Food Junkie" from 1976. It was a sly commentary on the health food craze of the time, as Baby Boomers channeled their idealism into health-conscious, planet-friendly lifestyles. The song's narrator talks up the good dieting habits he parades around in front of his peers, but then owns up to indulging his cravings when no one's looking: *"In the daytime I'm Mr. Natural, just as healthy as I can be. But at night I'm a junk food junkie, good Lord have pity on me."* It's a silly song full of light-hearted jabs at the performative self-care of the crunchy-granola era.

This bit of pop culture ephemera wafted through my mind earlier this month when the news broke of a 17-year-old boy whose junk food diet left him blind from optic neuropathy. It was a story tailor-made for viral success on social media: a human tragedy, a cautionary tale and a medical oddity all in one. The boy subsisted for years on not much more than Pringles, French fries, white bread and a little pork. With only negligible amounts of vitamin B12 in his diet, lasting—and now permanent—neuropathic damage ensued. (In this, "Junk Food Junkie" was somewhat prophetic: *"I'm afraid someday they'll find me, just stretched out on my bed. With a handful of Pringles potato chips, and a Ding Dong by my head."*)

The public outrage directed at the boy and his parents for such behavior—this wasn't a momentary lapse of reason, but prolonged inattention to nutrition—was at odds with the actual eating habits commonly practiced. One need only look at the obesity statistics to see the disconnect between what we preach and what we practice. Americans are pretty schizophrenic about food. Seeing, on social media, nasty jabs at this poor kid intermingled with lavish praise for the new Popeye's chicken sandwich was jarring, to say the least.

Those early adopters of organic eating habits in the '70s knew the importance of individual responsibility in shaping the course of their health. Nowadays we call this concept wellness. Though it's gaining in mainstream acceptance, too many people still associate it, derisively, with the era of macramé and platform shoes.

Truth is, we need to take healthy eating more seriously. And optometrists are increasingly well-situated to spark such a change. Diligence toward creating a sustainable health-promoting diet can dramatically alter the course of diabetes, and to a lesser extent AMD, in this country.

At the risk of being exploitative of the unfortunate individual who brought 'nutritional neuropathy' into the public consciousness, I think the case could be a real wake-up call to those patients who need to start making some changes. Conventional public health awareness campaigns, though well-intentioned, lack urgency and drama. But showing your patients a direct, if extreme, example linking diet to vision loss just might (excuse the pun) open a few eyes. ■

Technology in balance

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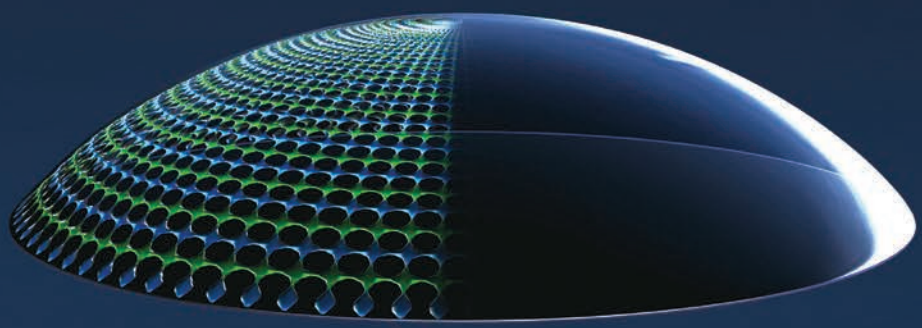

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- Super smooth surface
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*Menicon data on file April 2016

ACUVUE™ RevitaLens MPDS: a solution for your patients

James Cook, Meredith Jansen-Bishop, Chantal Coles-Brennan and David Ruston*

Overview

Functions of a multipurpose contact lens solution

- Effective disinfection against wide range of pathogens
- Non-toxic to the eye
- Compatible with wide range CL materials
- Enhance CL comfort & wettability

→

Challenges faced by multipurpose solutions

- Exposed to greater range of pathogens in real-world compared to standard testing
- Poor Px compliance challenges effectivity: poor CL and case cleaning practices, poor hygiene and exposure to water

→

Introducing ACUVUE™ RevitaLens MPDS

- Delivers peroxide-quality disinfection with the simplicity and all-day comfort of an advanced multi-purpose solution
- Compatible with a wide range of CL materials including ACUVUE® Brand Contact Lenses

Functions

A multipurpose contact lens solution (MPS) has to perform a delicate balancing act: to deliver effective disinfection against pathogens whilst being gentle enough to be introduced directly to the eye. Further functions include enhancing on-eye contact lens comfort and wettability, ease of use, and being compatible with a wide range of contact lens materials. Formulating a MPS to meet all of these functions is truly a complex challenge.

Efficacy

Formulations of MPS vary, and clearly so does their performance in standard testing. Two categories exist. Multipurpose disinfecting solutions (MPDS) pass the more rigorous ‘stand-alone’ test, where numbers of test bacteria and fungi are significantly reduced

without any cleaning intervention.[1] In contrast, a MPS solution cannot achieve this level of disinfection under stand-alone conditions, and has to pass a second 'regimen' test that involves a contact len undergoing the full recommended rub and rinse routine.

Real-world challenges

Beyond standard testing, contact lens solutions are further challenged by real-world conditions. These include exposure to a wide range of pathogens, including Acanthamoeba, and to wide-spread patient non-compliance to correct cleaning and safe wearing practices such as poor hygiene, incorrect case care and exposure to water.[2] It is also important for a contact lens solution to be able to demonstrate efficacy in these situations.

Introducing ACUVUE™ RevitaLens MPDS

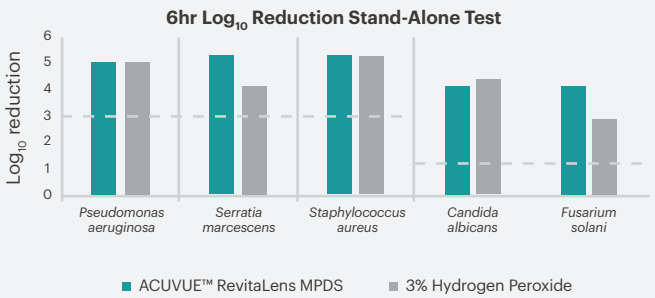
ACUVUE™ RevitaLens MPDS is a care solution which performs across these key functions. It has been shown to deliver peroxide-quality disinfection with the simplicity and all-day comfort of an advanced multi-purpose solution.[3-7] It is also compatible with a wide range of contact lens materials including ACUVUE® Brand Contact Lenses.

Performance

DISINFECTION COMPARABLE TO PEROXIDE SYSTEMS

ACUVUE™ RevitaLens MPDS produces exceptional levels of disinfection in the rigorous stand-alone test. These results demonstrate a similar level of performance to hydrogen peroxide systems,[3-5] and enable the solution to be categorized as a multipurpose **disinfecting** solution (MPDS).

ACUVUE™ RevitaLens MPDS also provides greater than 99.9% kill-rate against both active and cyst forms of *Acanthamoeba*.[3,8]



DELIVERS ALL-DAY COMFORT

After one month of use, ninety percent of wearers agreed that ACUVUE™ RevitaLens MPDS was effective in keeping their contact lenses feeling comfortable. More than 9 in 10 (94%) agreed that it also was effective in keeping their contact lenses feeling clean.[6]

COMPATIBLE WITH A WIDE-RANGE OF LEADING CONTACT LENSES

Use of ACUVUE™ RevitaLens MPDS resulted in positive comfort scores and all-day comfort across two studies with ACUVUE® Brand Contact Lenses.[7,9] All-day comfort has also been demonstrated with other leading reusable brand contact lenses.[7]

ACUVUE™
RevitaLens
Multi-Purpose Disinfecting Solution

NEW
TO ACUVUE®

*Mr. James Cook is Senior Manager Product Development, Dr Meredith Jansen-Bishop and Dr Chantal Coles-Brennan are Principal Research Optometrists and Mr. David Ruston is Director of Global Professional Education and Development at Johnson and Johnson Vision Care, Inc.

Summary

It is challenging to be a multipurpose contact lens care system. ACUVUE™ RevitaLens MPDS has been shown to deliver peroxide-quality disinfection with the simplicity and all-day comfort of an advanced multi-purpose solution. It is also compatible with a wide range of contact lens materials.

For a typical patient, wearing reusable contact lenses and leading a busy life, ACUVUE™ RevitaLens MPDS delivers a safe, simple and comfortable option. Truly a solution for your patients.

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Letters to the Editor

Keep Visual Function in Sight

The 10th Annual Retina Report published in the June 15, 2019, issue includes another excellent medical article on age-related macular degeneration, "How to Stay One Step Ahead of AMD." However, it is another example of how far optometry has strayed from its original foundation in vision care.

As optometrists, we must remember that all of the new technology for diagnosis and management we use, the nutritional and lifestyle changes the patient must make, the office visits, tests and injections into the eye they must endure are all done and suffered through for one reason: they want to see!

In a discussion with Henry Greene, OD, President of Ocutech, we reminded each other of the benefits of medical optometry. In the early days of our careers (1970s and 1980s), virtually all of our low vision patients had less than 20/400 vision. In fact, I hardly saw any wet AMD patients due to such poor acuity. Nowadays, nearly all of our patients are between 20/70 and 20/200, with much better results achieved with low vision care.

The point is this: it is still vision loss. We are still in the business of making people see to function.

In my opinion, every article on AMD must remind optometrists that referral to low vision rehabilitation is now the standard of care for those facing vision loss. Thank you for your consideration.

—Richard J. Shuldiner, OD, FAAO, FIALVS
President, International Academy of Low Vision Specialists
Clinical Director, Low Vision Optometry of Southern California

Dr. Legge replies: Dr. Shuldiner's point is well taken. We have a low vision specialist within our own office and I have been tending to send patients with AMD or other eye diseases for a consult earlier and earlier. Even patients with best-corrected vision of 20/30 or 20/40 can benefit from certain devices or specialty spectacles, depending on their visual demands.

That said, the intention of my article was more geared toward early diagnosis, intervention and prevention of vision loss rather than managing patients throughout a progressive AMD diagnosis and vision deterioration.

Editor's note: Low vision is indeed a vital part of optometrists' efforts to restore as much functional vision as possible. Recognizing its importance, we have included in this month's issue a feature article devoted to some of the latest advances in the field.

The complexity of AMD also warrants the inclusion of articles limited in scope to a particular aspect, such as diagnosis, so that the topic can be discussed in depth. Early disease recognition and intervention holds the potential to forestall the vision loss itself, perhaps preventing or delaying the need for low vision interventions or improving the uncorrected acuities of patients so that they may have an easier time incorporating visual aids into their lives.

Rethinking an Old Therapy

I think we sometimes overlook possible treatments that are right in front of us. I was in practice for 30 years and only had five cases of epidemic keratoconjunctivitis. All of these were treated with apparent success. I used 5% NaCl salve every two hours, positioned a half-inch in the lower cul-de-sac. I demonstrated proper instillation to the patient and instructed them to close the eye for five minutes to allow the salve to melt. In each case, it worked in about three days with no recurrence.

I think, if I am correct, routine adoption of this technique would be a better treatment for the patient than pharmaceutical use, as it would spare the patient both cost and potential toxicity to the tissue.

— John Conrad, OD, Salem, OH

Letters to the Editor Welcome

To share feedback on articles or general commentary about the state of the optometric profession, write to jpersico@jobson.com.

Hypochlorous Acid Re-Imagined for Eye Care

How This Naturally Occurring Substance Can Elevate Eyelid Hygiene and Help Manage Dry Eye Symptoms

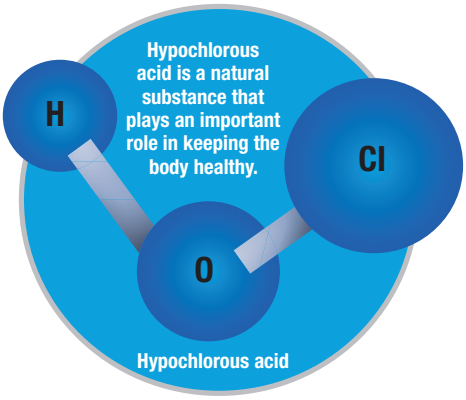
By Marguerite McDonald, MD, FACS

Eyelid hygiene is a crucial component of ocular health, especially for patients with conditions such as dry eye, blepharitis, or meibomian gland dysfunction. However, some older lid hygiene practices are not optimally effective, and many patients aren't compliant with their eyelid cleansing regimens. In fact, studies have shown that though 93% of eye care professionals recommend eyelid cleansing for this group,[1] only 19% of dry eye patients regularly cleanse their eyelids.[2] The groundbreaking Dry Eye Workshop II (DEWS II) report strongly advised that clinicians utilize newer, more efficacious hygiene solutions available on the market today to improve patient outcomes and compliance when it comes to lid hygiene, as opposed to more traditional strategies.[3]

One new way to elevate eyelid hygiene and help increase patient adherence involves the use of hypochlorous acid—a substance generated naturally in the body. Hypochlorous acid is produced in neutrophils and functions as an antimicrobial agent that destroys bacteria, serving as an important part of the immune system. Studies are finding that solutions containing hypochlorous acid not only possess powerful antimicrobial properties, but are well-tolerated for continuous use and yield minimal cytotoxic effects.[4-6]

The Need for Improved Lid Cleansing Approaches

The importance of appropriate lid hygiene was emphasized in the DEWS II report, offering foundational guidance from 150 worldwide experts in the areas of ocular surface care and disease management. The authors stressed the need to appropriately manage a variety of lid conditions that result in dry eye, particularly blepharitis. If used correctly, they determined, lid hygiene could reduce



lipid byproducts and lipolytic bacteria associated with these conditions.[3] The report also noted certain outdated lid hygiene practices that should be updated by eye care professionals, as well as a lack of patient compliance with best practices. For example, it revealed:[3]

- Though lid scrubs using diluted baby shampoo traditionally have been a widely accepted therapy,[7-9] one Level 1 study found that a dedicated lid cleanser had reduced ocular surface MMP-9 levels and improved lipid layer quality, and was better tolerated than diluted baby shampoo.[3,10] Baby shampoo also has been associated with reduced ocular surface MUC5AC levels, suggesting it might have an adverse effect on goblet cell function.[8]
- New, proprietary lid cleansing products that use a diversity of delivery mechanisms are recommend-

Eyelid Cleansing: Essential, But Often Overlooked

93% of eye care professionals recommend eyelid cleansing for certain patients[1]

ONLY 19% of dry eye sufferers regularly cleanse their eyelids[2]

ed over traditional lid cleansing strategies.[3] In recent years, many lid hygiene solutions have come to market as marked advancements over baby shampoo.

• Though lid hygiene is widely considered an effective therapy for MGD and blepharitis,[11] compliance with provider instructions is "notoriously poor."[3]

It's clear that modern approaches to managing lid hygiene, such as use of hypochlorous acid, are a more appropriate way to promote eyelid health than older methods using baby shampoo.

Expanding Access to Rx-Strength Solutions

Until recently, two types of eyelid cleansers were available to patients in the following ways, with these traits:

• *Cosmetics:* Not filed with the FDA, with large variation in ingredients and efficacy. Limited availability in retail stores, with most distribution in physician's offices and online.

• *Rx products:* Proven efficacy, but inconvenient due to prescription requirement and potentially very costly depending on insurance.

Realizing the need was great to provide patients with greater accessibility to prescription-strength daily eyelid cleansers, researchers embarked on developing such a product. The result is TheraTears® SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash, now available at a variety of retail stores.

Hypochlorous Acid Reduces Ocular Skin Bacterial Load

One study demonstrated a

>99%

reduction in the staphylococcal load on periocular skin 20 minutes after a solution containing 0.01% pure hypochlorous acid was applied.[1]

Research has revealed the ability of hypochlorous acid to reduce the bacterial load on the surface of the periocular skin shortly after application.[1] Some solutions have even removed staphylococcal isolates resistant to multiple antibiotics.

1. Stroman DW, Mintun K, Epstein AB, et al. Reduction in bacterial load using hypochlorous acid hygiene solution on ocular skin. Clin Ophthalmol. 2017;13(11):707-14.

A Natural Opportunity for Hypochlorous Acid

In today's world, many patients actively seek out products with more natural characteristics. Embodying such qualities, hypochlorous acid is organically produced as part of the cytotoxic myeloperoxidase system in neutrophils.[12,13] This broad spectrum antimicrobial mimics the human system, and has been shown to cause rapid oxidation of nucleotides, inactivation of cell enzymes, disruption of cell membranes, and cell lysis when introduced to various microorganisms in vitro.[14-17]

From a clinical perspective, agents containing hypochlorous acid hold powerful antimicrobial properties that appear to be useful for ongoing use, exhibiting minimal toxic effects to cells in several studies.[4-6] In addition, hypochlorous acid exerts a broad range of anti-inflammatory and immunomodulatory activities, such as those involved in the pruritic cycle of certain dermatologic conditions.[18] For example, in atopic dermatitis, hypochlorous acid may decrease protease binding and modulate interleukins involved in the inflammatory cascade.[18]

A Diversity of Clinical Uses for Hypochlorous Acid

Products with hypochlorous acid have received many FDA and EPA approvals across a broad range of medical markets including dermatology, ophthalmology, dentistry, and wound healing care. They are also widely used in veterinary and ostomy applications.

Clinically Advancing Lid Hygiene Practices

New products featuring hypochlorous acid offer leaps forward in the following clinical areas of eyelid hygiene:

Antimicrobial & Antifungal Efficacy: Research has documented the significant ability of antimicrobial agents such as hypochlorous acid to help maintain healthy skin—including near the eyelids—and to inhibit growth of pathogenic bacteria while promoting the proliferation of symbiotic bacteria.[19] It also has determined the swift, broad-spectrum fungicidal activity of 0.01% hypochlorous acid.[20] One literature review, which evaluated cases of fungal keratitis and endophthalmitis after Boston keratoprosthesis implantation during a 14-year period, noted the ability of 0.01% hypochlorous acid to reduce medically relevant yeast cells or mold conidia by 99.99% within 60

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The first FDA-Accepted antimicrobial eyelid cleanser, TheraTears® SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash (Hypochlorous acid 0.01%), a convenient and affordable over-the-counter solution that is as effective as a prescription:

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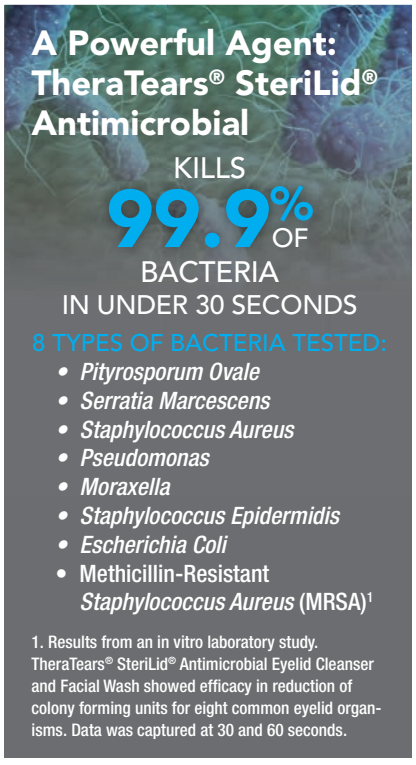
- Accessibility at major retail stores
- Patient-friendly pricing
- 24-month shelf life open or un-opened



seconds, measured by an in vitro time kill assay.[20]

Tolerability: New hypochlorous acid daily eyelid cleansers designed to have low toxicity and pH-balanced formulas may prevent irritation of the delicate skin of the eyelids and eyelid margin.

Patient satisfaction: Rx-strength daily eyelid cleansers featuring non-irritating substances that are available at retail stores offer the possibility of high patient satisfaction and compliance due to greater comfort and easier access to effective eyelid cleansers.

A First in Daily Eyelid Cleansing

An innovation helping eye care professionals to advocate for daily eyelid hygiene, TheraTears® SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash containing 0.01% hypochlorous acid is the first eyelid cleanser to be FDA accepted as a medical device. It has a rinse-free formula, eliminating the need to clean away a residue, and reflects patient-friendly pricing. In addition, the cleanser has a 24-month shelf life, opened or unopened.

My firsthand experience with TheraTears® SteriLid® Antimicrobial Eyelid Cleanser & Facial Wash is that it is easy and quick to use, non-irritating, and effective. It can be applied long-term without irritation. My patients are pleased with how their eyes and lids look and feel after using the product, and I see the slit-lamp improvements.

An eyelid cleanser such as TheraTears® SteriLid® Antimicrobial is especially important for dry eye, pre-operative, and MGD patients, patients who use eye makeup, and those who wear artificial lashes. The MGD Workshop[21] recommends eyelid cleansing twice daily as a treatment starting at the earliest MGD stages, and mentions the advantages of hypochlorous acid as an important and effective ingredient in cleansing solutions.

With the exception of blepharitis/MGD that is graded as trace, or trace to 1+, my first-line therapy for grade 2+ or greater starts with lid hygiene twice daily with an eyelid cleansing solution containing hypochlorous acid. In addition, I also use a host of other products mentioned in the DEWS II and MGD Workshop recommendations.

I have been very pleased with the results of new hypochlorous acid products. Not only are they effective, but they help improve daily eyelid hygiene compliance because patients look and feel better rapidly. Fortunately, we have therapeutic options now such as the TheraTears® system, designed to offer more complete relief of dry eye symptoms for patients.

Dr. McDonald practices at Ophthalmic Consultants of Long Island, Dry Eye Center of Excellence in Lynbrook, New York. *Dr. McDonald has received compensation for the preparation of this article from Akorn Consumer Health, manufacturers of TheraTears®SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash.*

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» DEWS II

The groundbreaking Dry Eye Workshop II (DEWS II) report strongly advised that clinicians utilize newer, more efficacious hygiene solutions available on the market today to improve patient outcomes and compliance when it comes to lid hygiene, as opposed to more traditional strategies.[3]

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Reference: 1. Results from an in vitro laboratory study. TheraTears® SteriLid® Antimicrobial Eyelid Cleanser and Facial Wash showed efficacy in reduction of colony forming units for eight common eyelid organisms. Data was captured at 30 and 60 seconds.

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It's Replacement Time

New tech and treatments are making current practices obsolete. Are you ready to be an early adopter? **By Paul M. Karpecki, OD, Chief Clinical Editor**

Innovation is the lifeblood of our profession, and some practices have differentiated themselves and grown on new technology alone, such as with OCT. More than a decade ago, lecturers said you didn't need an OCT to effectively manage glaucoma. While that's still technically true today, the technology has proven indispensable, and I, for one, wouldn't be confident in my glaucoma management without it. Here are a few other tools and treatments that are making waves.

In With the New

Speaking of glaucoma, corneal hysteresis (CH) is starting to replace pachymetry. Studies show that pachymetry does not accurately correlate with IOP, and it can't be adjusted based on a linear scale of adding or subtracting mm Hg.[1] CH, unlike pachymetry, correlates with visual field loss progression and is a tipping point in the diagnosis for many early cases.[2]

The newest Icare ic100 tonometer increases accuracy compared with Goldman tonometry and allows for easier and more efficient measurements. The Icare Home now allows patients to measure their IOP daily.

Novel post-surgical treatments are starting to replace drops with the recent approvals of Dexycu (EyePoint) and Dextenza (Ocular Therapeutix). Dexycu is a time-released dexamethasone 'pellet' inserted into the anterior chamber, while Dextenza is a dexamethasone-eluting punctal plug. Both have indications for inflammation and pain following ocular surgery. These advances may increase compliance and effectiveness, particularly for those who struggle with drops. Another good option is new injectable solutions such as Dex-Moxi-Ketor (Imprimis).

Enhance, Not Replace

Autorefractors haven't replaced a subjective refraction, and many new tools will follow a similar path. For example, I use ultra widefield fundus imaging (UWFI), but it's not a replacement for a dilated exam. With UWFI, I can better identify suspicious areas that need a closer look with dilation; I've caught retinal tears I would have likely missed without it. But there are times when UWFI may not accurately isolate pathology, which is why it's a complement to, not a replacement for, a dilated exam, especially in cases of flashes and floaters.

Innovation on the Horizon

Several advances aren't here yet, but may make it to your office soon:

The swinging flashlight test, which provides relative afferent pupillary defect (RAPD) measurements, may meet its match this fall. A new technology called EyeKinetix (Konan) measures pupil disparity and provides an accurate reading of an RAPD, including early or subtle levels—in about 30 to 40 seconds.

ObjectiveField (Konan) is an objective visual field test that appears to show similar accuracy (and perhaps greater accuracy in first-time users) than subjective visual field testing. This will likely debut in 2020, potentially replacing subjective visual field testing.

We will likely see adjustable IOLs (RxSight) in late 2019 or early 2020, which allow for adjustment up to 30 days post surgery. Say a patient wishes they had chosen monovision, and you use a contact lens fit on the non-dominant eye to determine that -1.50D is the ideal correction—a UV light laser can adjust that on the IOL.

Also, an implantable artificial capsule (Omega Ophthalmics) will one day allow for an IOL exchange or addition, should a patient require further visual correction.

As tear-based point-of-care testing becomes more sophisticated in measuring inflammation, allergy markers and, eventually, systemic conditions such as diabetes and Alzheimer's, it will replace traditional measures of diagnosis.

Technologies that replace previous diagnostics and treatments are essential to the growth of our profession and the improvement of our patients' vision. It's imperative that we explore these technologies and determine which ones will enhance our practice and patients' lives. Those are worth the investment. ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

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Brand Theater...

It's part of your design, but do you have the right ticket?

Do a quick search for articles on optical design and you'll find many; and many will concur that the trend for optical design is more toward sleek and minimalistic, with displays that are consistent in their look but display differently for select price points. For example, higher priced frames should be given more space, such as open shelves, whereas lower priced frames should be displayed closely and on a different display system such as individual frame holders. When you give more space to a frame this tells the patient it is more of a luxury product. A successful design formula includes minimalist décor which highlights eyewear collections with clever displays.

However, there is an opportunity largely overlooked in the design of an optical. It's called the "brand theater." Every day I work with doctors' practices and I help put together their "brand theater" with merchandising components such as signage and graphics. More often than not, when I visit a practice I see those merchandising components still sitting in the shipping box; not on the "stage" for which they're made.

When a patient comes into your practice they should immediately be made aware of the products you carry, and be able to follow visual elements to them. Today, visual is traveling faster than words (think "photos before forks" - we take photos of our food before dining), so start with branding boards that list the top selling brands you carry to draw the patient into the optical.

Sense of sight provides 83% of human information and Point-of-Sale (POS) remains the primary visual contact for the optical. Your patients are just a Google search away from understanding the products, services and frames you carry, so give them the instant gratification of buying on the spot in your optical and provide the best patient experience possible to them.

With optical retailing gradually falling in line with fashion retailing (I call it "O-tailing"), enhancing the patient shopping experience is key. Your optical and reception is your patient's first and last impression of you and your practice. Doctors may boast about their latest equipment (which is usually not their biggest profit center); meanwhile the front of the practice looks like it's been lost in a time warp. Patients won't be able to tell if you have the top of the line OCT, but they will be able to tell if the displays and furniture look worn and outdated. To remain relevant, because a brick-and-mortar optical is competing with e-commerce, practices need to create inventive ways to entertain and provide a pleasing environment.

Example of displays & design by industry specialist the Eye Designs Group.

Which brings me back to "brand theater." It's part of your design, but do you have the right ticket?

HERE ARE SOME THINGS TO CONSIDER:

- Use large graphics that target your patients, gets their attention and connects with them to create loyalty.
- Integrating video in select areas can be an effective way to inform your patients. Typically one large video monitor is most effective as multiple videos create unnecessary noise. Walk into any high-end designer boutique and you will see one large video highlighting recent fashion shows and introducing new products and styles.
- Consider using music, and if you already have music, make certain it appeals to your patient base. Music can trigger emotional responses when used thoughtfully. This includes your telephone on-hold message.
- Focus on demographics and merchandise to your patients accordingly. Your practice will perform better with improved product turnover ratios compared to having excess inventory of dated products.
- Engage your patients across multiple touch points (signage, graphics, video, music, point-of-sale) and don't forget the contact lens area.
- Doctors have learned that for their patients the shopping experience is not just about providing prescriptions, it's about creating a shopping experience that is fun, exciting and establishes a true brand connection to you as well as the products you sell.

Brand theater, as in regular theater, has a stage, lighting, materials, finishes and props. It conveys a message through multiple dimensions, as should the presentation of your products and services. What is your practice doing to enhance the optical experience using brand theater? Will your patients have, and continue to have, an experience they can't wait to share (think social media)? If so, that's the right ticket!

For more information contact the author:

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Patricia has 30 years of experience working in the ophthalmic industry, branding for a major eye wear company, designing frames and currently designing successful optical environments. She is also a featured speaker at many industry events.

Choose Your Words Carefully

Your team needs a rallying cry. Since "Remember the Alamo" is taken, it's time to get creative. **By Montgomery Vickers, OD**

Does your office have a slogan? In the heat of the battles in optometry, we need something to remind us where the True North of our office may be. Otherwise, it's easy to get lost along the way.

Long-time readers will recall that, in the '80s, I came up with what I thought was a great slogan to make our office stand out. I was so sure of it that I ordered thousands of lens cleaning cloths with the amazing slogan printed on them. I was pretty proud of myself. Years later, my high school-aged son asked me why they said "Ther Area's Best Eye Doctor." Yes, "Ther."

I then changed to an even better slogan: "Our patients are special!"

I liked that this slogan emphasized the patient and not me. It also gently insinuated that if you were not my patient, you were not special. So true. But mostly it was easier for the state board to stomach since they always wondered how I could prove that I was, in fact, "Ther Area's Best Eye Doctor." At least I never claimed I was America's best eye doctor like some people we know. I wonder how they prove that?

From the Vault

Now, my road to verbal perfection is not without other mistakes. Here are some slogans I have toyed with, and discarded, over the years:

- **2CCME** - Sadly, someone already beat me to this vanity license plate.
- ***You deserve a great eye exam*** - But my office was more conveniently located. That counts for something, right?
- ***Free coffee cup for your family members*** - We had to stop because my mom had 57 cups. She passed away a couple of years ago when the caffeine wore off.
- ***We love our patients*** - Sounded better than "We love some patients."
- ***How important are your eyes?*** - Turns out straight teeth take priority.
- ***When was your last eye examination?*** - I sent this to all the local ODs. Hopefully it encouraged them all to at least refract themselves.
- ***Itching and burning? Come see us!*** - Perhaps I should have specified that I was asking about their eyes. Made for some embarrassing moments.
- ***Your mom needs new glasses*** - We had no responses, showing how important moms are.
- ***Are you ever on a computer?*** - Got hacked immediately.
- ***Teeth can be replaced*** - I even included a picture of an eyeball. My dental professional children still won't speak to me.
- ***We make sexy glasses*** - Turns out they rarely help people's love life.
- ***The first thing people see is your eyewear*** - It's the first thing I notice, anyway.
- ***Close your eyes and read this. Did it work?*** - Even my brother thought this was stupid.
- ***Birthdays are the leading cause of blindness. When's your next one?*** - Depressing, but there's only one alternative to birthdays, so keep 'em coming!

Patients pay attention to what you say, especially when it's something dumb. Check your signs. Check your website. You can never take back what they read there, so make sure it makes sense. And, feel free to use "Ther Area's Best Eye Doctor." It's not copyrighted. The state board will love it! ■

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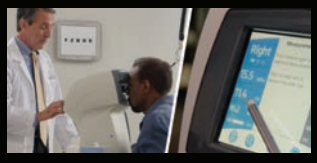

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Hit Hard Against Uveitis

An aggressive but smart strategy can do a lot with a little.

Edited by Paul C. Ajamian, OD

Q A 38-year-old uninsured male presented, stating his right eye had been red for a week. Examination revealed best-corrected acuity of 20/60 OD with a cyclitic membrane, 10% hypopyon and 3+ to 4+ cell and flare. With few resources to pay for meds or labs, how should I treat him?

A Uveitis management begins with an accurate description of the inflammation and often requires additional testing to determine underlying systemic, autoimmune or infectious disease. "A dilated fundus examination is necessary to determine the primary site of inflammation," says Jessica Steen, OD, assistant professor of Nova Southeastern University in Fort Lauderdale.

Clinical clues can help to guide your diagnosis. However, first rule out intermediate or posterior uveitis, or even panuveitis. Since this is the first episode of unilateral anterior uveitis with severe inflammation (cyclitic membrane and hypopyon), Dr. Steen suggests asking about systems associated with HLA B27 positivity, Behcet's disease or herpes.

"My basic laboratory evaluations for this patient would be a CBC with differential, HLA B27, chest X-ray, RPR and FTA-ABS, quantiferon gold (for tuberculosis) as well as consideration of Lyme antibody titer," says Dr. Steen. However, our patient declined these tests because of cost, which we carefully documented.

Treatment

The mainstay of initial therapy in anterior uveitis is a strong topical steroid with an aggressive dosing strategy. While Durezol (difluprednate 0.05%, Novartis) is our first choice, a branded medication may be unfeasible for uninsured patients. Generic prednisolone acetate 1% dosed at least hourly will typically provide adequate treatment.

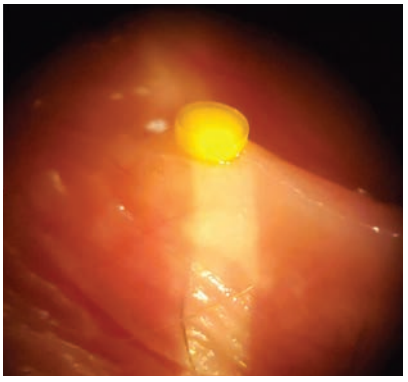

Fig. 1. Dextenza could be a promising alternative or adjunct to drops in treating anterior uveitis.

"Once you have control of the inflammation, determine if the patient is ready for a slow, deliberate taper," Dr. Steen says. Tapering a steroid too quickly could increase inflammation and result in a longer treatment course, increasing the risk of side effects.

Dr. Steen suggests lagging the taper way behind the improvement—only tapering when the 3+ to 4+ cell and flare has significantly improved. Even then, taper slowly.

For patients who show limited improvement with topical steroids, a short course of Medrol Dosepak (oral methylprednisone, Pfizer), or subconjunctival corticosteroid may be necessary. Extended delivery devices, such as the recently approved Dextenza plug (sustained-release dexamethasone 0.4mg, Ocular Therapeutix), can minimize the patient's need for topical therapy; however, it's only approved right now for the treatment of postoperative pain and inflammation (*Figure 1*). Using it alone would most likely not provide the anti-inflammatory effect needed for a primary uveitis.

Manage Pressure and Pupils

With strong steroids dosed frequently, you might get the occasional intraocular pressure (IOP) spike. Continue your anti-inflammatory therapy as planned and add an IOP-lowering medication or combination agent. Dr. Steen's choice is the fixed combination of dorzolamide 2% and timolol 0.5%.

Cycloplegic agents suppress ciliary body spasm, improving comfort and preventing posterior synechiae formation. For mild to moderate iritis, consider homatropine 5% every 12 hours. In advanced cases like our patient's where synechiae have formed, have some 1% atropine and 10% phenylephrine on hand.

We provided our patient with the dilating drops and kept costs to a minimum using generic prednisolone. The hypopyon and cyclitic membrane resolved within a few weeks, and his vision improved to 20/25. Advise patients that it could take several months for complete resolution. We are hoping to run lab tests should he become insured but, meanwhile, will monitor him for any recurrences and re-treat if necessary. ■

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By Paul M. Karpecki, OD, FAAO

Rethinking DED Treatment

Dry eye disease is an underestimated disease in need of new treatment options.

Dry eye disease (DED) is a chronic disease that negatively impacts quality of life comparably to other severe diseases.[1] Symptoms of DED such as feeling of dryness, burning, foreign body sensation or pain are often quite debilitating. Moreover, visual function-related manifestations including fluctuating vision with blinking, blurred vision and difficulty with reading despite normal visual acuity are important and underestimated aspects of the disease.[2]

As many as 5-35% of patients visiting eye care professionals report dry eye symptoms, making it one of the most common ocular conditions seen.[3] And although more than 16 million people are diagnosed with DED in the US,[4] only about 10 percent are treated for the disease.

The fact that 90% of people are not being treated for dry eye means they will likely experience ocular surface damage and a significant drop in quality of life. This situation necessitates a closer look at the unique characteristics of DED, and the way eye care has approached diagnosis and treatment of the disease until now. It also begs the question of whether there might be a better way to care for DED patients.

Characteristics of DED

The International Dry Eye Workshop (TFOS DEWS I and II) classifies dry eye into two major categories: 1) aqueous deficient (i.e., keratoconjunctivitis sicca) and 2) evaporative (i.e., tear-lipid deficient).[5] Research has found that around 10% of dry eye patients have a solely aqueous deficient disorder, in which reduced tear production leads to tear film instability, while 60-90% of patients present with predominantly evaporative dry eye, in which an altered lipid layer leads to tear film instability.[6] About 40% of patients have a mixed presentation.

Meibomian glands on the lower and upper lid play an important role as the sole natural source of lipids for the human tear film. Meibum spreads onto the tear film, promoting its stability and preventing evaporation.[7,8] A large number of DED patients have meibomian gland dysfunction (MGD), either by itself or comorbid with aqueous deficiency. Those suffering from DED with imbalanced tear conditions due to significant MGD represent a symptomatic and large population with great medical needs.

Traditional Treatment of DED

Traditional DED treatment has started with water-based artificial tears and topical lubricants. For moderate to severe cases, topical anti-inflammatory medications, including short two to four-week courses of corticosteroids and longer-term therapy of cyclosporine A and lifitegrast, have been used.[9]

Addressing inflammation is a critical component of treating both forms of dry eye disease. However, available therapies for evaporative DED patients are less optimal, as anti-inflammatory medications often don't address the root cause of excessive evaporation. There is a need for more tolerable and faster-acting agents that have fewer to no side effects, or complication risks.

Treating Inflammation Only One Component of Dry Eye Therapy

It's important to remember that inflammation is just one aspect of the various forms of DED. Although it may be one key to slowing progression, in the case of evaporative DED—making up about 86% of DED—clinicians must also treat the underlying obstructed meibomian glands. At the same time, just treating the obstructed glands of patients with evaporative DED without managing inflammation leads to a potentially quicker return to obstructed glands. Both issues must be handled for optimal outcomes.—*Dr. Karpecki*

Overcoming Limitations

The significant gap between diagnosed and effectively treated patients, as well as the unsatisfying treatment options available for patients require a rethinking of underlying DED mechanisms. It's clear that new therapies are needed.

In response to the need for new DED therapies, Novaliq is developing two topical products for commercialization in the US. Both are based on the company's proprietary water-free EyeSol® technology.

Due to their water-free characteristics, EyeSol® products are preservative- and surfactant-free, and designed to greatly improve tolerability compared with traditional water-based drugs. CyclASol® and NOV03 potentially offer new treatment approaches for both DED segments to improve patients' quality of life.

CyclASol®

CyclASol® 0.1% (cyclosporine A 0.1% in perfluorobutylpentane) is intended as a treatment for patients with moderate to severe DED with an inflammatory component. The use of perfluorobutylpentane as a vehicle for cyclosporine is designed to obviate the need for a preservative, enhance the stability and bioavailability of the active ingredient, and improve comfort.

In the Phase IIb/III ESSENCE study, a multicenter, double-masked trial conducted in the United States, cyclosporine A 0.1% in perfluorobutylpentane met its primary endpoint of change in total corneal fluorescein staining from baseline to week four.[10] A statistically significant improvement in corneal fluorescein staining compared with the vehicle

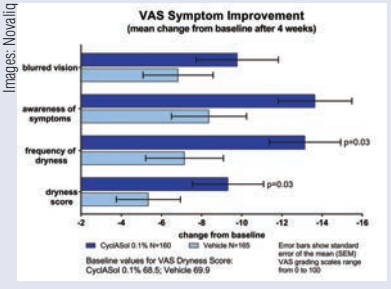
was seen as early as two weeks, and maintained at four, eight and 12 weeks. ESSENCE also significantly met the prespecified symptom endpoint of VAS Dryness Score after four weeks, with a p-value of 0.03. The study included 328 randomly selected patients.

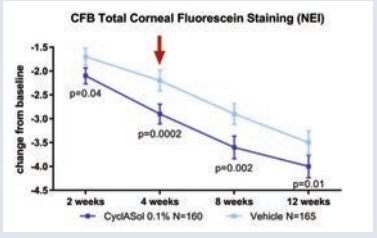
Novaliq believes that CyclASol® 0.1% holds the promise of harnessing cyclosporine A's full potential for the first time in the treatment of DED treatment. It holds the potential to demonstrate the superior benefits of a water- and preservative-free multi-dose formulation, and could enable clinicians to treat more patients suffering from predominantly aqueous deficient DED.[10] This potential is significant because this is not just a higher concentration of cyclosporine A, but rather a vehicle that completely changes how we address the disease in terms of efficacy.

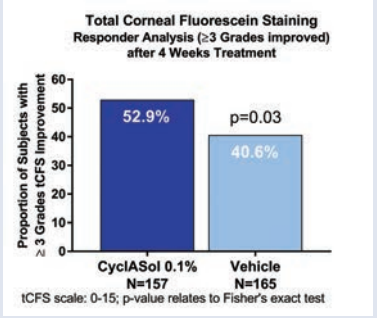
Notably, the drug's water-free nature is designed to result in one of the most comfortable and tolerable dry eye prescriptions we've seen to date. The comfort scores and lack of burning upon instillation in the Phase IIb/III study were remarkably low. This is a very comfortable drop; its water-free characteristics not only help with solubility, but especially with tolerability.

ESSENCE Findings.
Data from the ESSENCE trial.

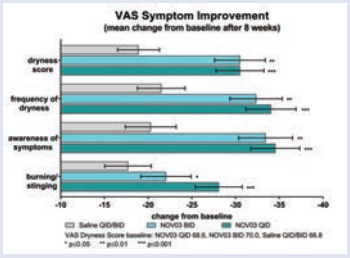
Images: Novaliq

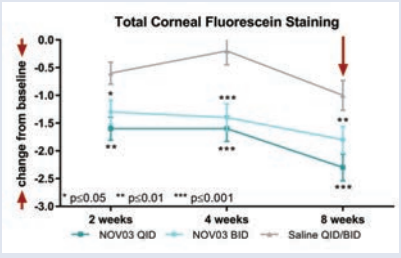






SEECASE Findings. Data from the SEECASE trial.





NOV03

NOV03, a preservative- and surfactant-free product containing 100% perfluorohexyloctane, is being developed as a treatment for patients with MGD-associated DED. Studies show that it helps stabilize the tear film lipid layer and mitigate excessive evaporation. In addition, it has been shown to penetrate into the meibomian gland and liquefy secretions there, improving the quality of the meibum and tear film lipid layer.

NOV03 was investigated in SEECASE, a Phase II, randomized, controlled, double-masked clinical US trial including 336 patients with predominantly evaporative DED associated with MGD. The enrolled patients were highly symptomatic and had a low tear film breakup time, normal Schirmer's scores, and mild to moderate corneal damage. They were randomized into one of four groups—to use NOV03 two or four times daily, or normal saline two or four times daily.

Topline results showed that the study met its prespecified primary endpoint—a change in total corneal fluorescein staining from baseline to week eight.[10] Compared with the control, both dosing regimens of NOV03 showed statistical superiority. The benefit of NOV03 for ocular surface damage was seen as early as two weeks after treatment initiation. The investigational agent was also associated with statistically and clinically relevant improvement in DED-related symptoms.[10]

I'm very excited about the prospect of NOV03. For one thing, it is being investigated to address the most common cause of DED and targets the meibomian glands. Subjects in clinical trials have experienced improvements in a variety of symptoms and corneal staining against control. I would expect we're going to see a significant improvement in vision. NOV03 is also a very comfortable drop, and I like the fact that it is designed to address meibomian gland secretions.

The Future of DED Treatment

Dry eye is a multifactorial disease with respect to its cause and predisposing risk factors, but also in terms of its process. For example, in evaporative DED, factors include obstructed meibomian glands, inflammation secondary to hyperosmolarity, a biofilm or blepharitis component, and a disrupted tearfilm. Having therapeutic agents that address more than one aspect of the disease process and that target different DED segments is extremely valuable for efficacy of treatment, as well as for patient compliance and satisfaction.

Targeted treatment options currently under investigation such as NOV03 and CyclASol®, based on a water- and preservative-free technology, offer hope that new drugs can provide more patients with a satisfying treatment solution—to improve ocular surface damage and DED symptoms, as well as preserve vision and quality of life.

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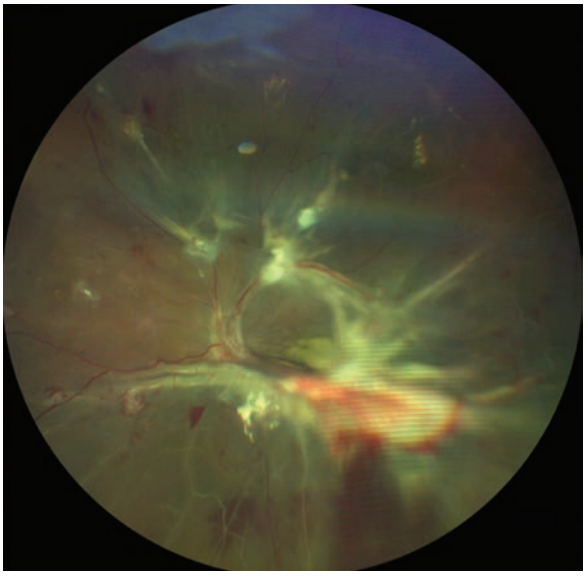
NOV03 and CyclASol are investigational products and not approved, yet.

Pericytes in Peril

Cell dropout is at the root of the microvascular aneurysms characteristic of diabetic retinopathy. **By Bisant A. Labib, OD**

Diabetic retinopathy (DR) is the leading cause of vision loss globally in the working, middle-aged adult population.[1] Nearly all patients with insulin-dependent diabetes develop vascular complications within 15 years of diagnosis, and approximately 34.6% of patients with diabetes exhibit some form of retinopathy.[1,2] Though the precise pathophysiologic mechanism of DR is not completely understood, it has long been associated with damage to the retinal vasculature and loss of pericytes specifically.[3,4]



This patient with proliferative DR shows signs of severe vascular dysregulation.

High Demand

The oxygen requirement for the retina is the highest of all tissues in the human body, exceeding even the brain. To accommodate this demand, the retina requires an extensive—and intact—vascular network to obtain the oxygen and nutrients necessary for adequate function. As such, the retina is supplied by two vascular systems: the central retinal artery nourishes the inner retinal layers, while the choroidal capillaries nourish the outer retinal layers.[3]

Damage from DR, observed at the microvascular level, is split into two categories: non-proliferative and proliferative. Early, or non-proliferative, DR manifests clinically as retinal microaneurysms or microhemorrhages. In later stages, proliferative DR appears as neovascular growth from existing retinal vessels.[3] One of the earliest abnormalities in the diabetic retina, leading to these microvascular aneurysms and hemorrhages, is pericyte dropout.[4]

The Pericyte-Glaucoma Connection

The correlation between pericyte loss and the manifestation of DR is well documented; in fact, DR is perhaps the most studied pathological condition resulting from pericyte loss. However, the impact of pericyte loss has also been implicated as a possible pathological mechanism in glaucoma development. Similar to the mechanisms involved in DR, vascular dysregulation and increased blood-retina barrier breakdown at the level of the optic nerve head can contribute to glaucomatous disease and progression. Further studies are warranted to determine the extent of pericyte involvement in glaucomatous optic neuropathy.[5]

Surface Support

Pericytes were first described in the 1870s by Eberth and Rouget and were initially named Rouget cells. They were later renamed "pericytes" because of their perivascular location relative to the endothelial cells. Pericytes are located on the surface of capillaries and function primarily to stabilize and support the blood vessels. They arise from neuroectodermal neural crest cells and have the highest density in the central nervous system.[5]

Identifying pericytes is often difficult because their cytoplasm may span several endothelial cells and exhibit different morphologies. As such, investigators have studied histological methods, revealing pericyte-specific markers that can aid with recognition. These include platelet-derived growth factor receptor β, NG2, CD13, desmin and vimentin.[4]

Pericytes in the retina are not readily observed on routine testing; they are approximately 7μm and extend distal processes that are even smaller, limiting the use of conventional imaging techniques.

Experimental methods have included *in vitro* and post-mortem histological examinations. More recently, a study using adaptive optics scanning laser ophthalmoscopy showed success in visualizing the retinal cellular structure *in vivo* using mouse models.[6]

Pericytes in the adult retina work as regulators of gene expression to protect the retinal vessels from stress and injury.[4] In healthy eyes, the endothelial cell-to-pericyte ratio is 1:1. In diabetic retinas, this ratio drops to 4:1. Prolonged hyperglycemia and genetic factors in diabetics lead to DR damage through several mechanisms, such as activation of protein kinase C, activation of the polyol pathway, non-enzymatic glycation, inflammation and oxidative stress. These events trigger pericyte death through apoptosis and dropout, leading to retinal microaneurysms, vessel tortuosity, hyperpermeability and capillary non-perfusion.[2,3]

Studies investigating pericytes in mice found that those with reduced numbers of pericytes experienced severe hemorrhage. Furthermore, mice with less than 52% of normal pericyte density developed signs of proliferative DR.[5] Microaneurysm formation in DR is partially due to the over-proliferation of pericyte-deficient endothelial cells in response to vascular endothelial growth factor from hypoxia.[2]

Research also suggests that pericyte loss from retinal capillary walls is responsible for the breakdown of the inner blood-retina barrier (BRB). While research shows that the tight junctions of the endothelial cells and the retinal pigmented epithelial cells make up the inner and outer BRB, many studies have concluded that reduced pericyte numbers also lead to increased permeability and BRB breakdown.[5]

Valuable and Vulnerable

Pericytes—small, specialized cells on the surface of capillaries—have a significant impact on vascular homeostasis. A better understanding of their prominent role in the pathogenesis of DR, and possibly other ocular diseases, can help spur the development of potential therapeutic targets to delay or treat this leading cause of vision loss. ■

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Me and My OCT

Maximize your technology—for the good of your patients and your practice.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Rapid technological advances are changing the way optometrists deliver clinical care in a big way. Earlier, more accurate diagnoses, better monitoring of chronic conditions and practice building opportunities are all great reasons to invest in new technology. But choosing which technology will help you deliver better care while maintaining a healthy bottom line is rarely easy. Let's focus on OCT as an example.

OCT has been a mainstream part of optometric practice for nearly a decade. Some would even argue that is has become the standard of care for glaucoma and macular disease. Not surprisingly, in 2012, CMS updated the coding for OCT from a single code (92135) to three to further refine and define OCT usage:

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

While these codes describe typical OCT functionality, other capabilities often built into the device require a better understanding of the coding rules and regulations. The three I hear about most often are:

1. Screening Exam

If your OCT has a specific screening mode, you may be able to use it to provide a non-medically necessary screening of the retina. Coding for this screening procedure would be defined as S9986 - Not Medically Necessary Service (patient is aware that service not medically necessary). Being a Level II HCPCS code, no reimbursement is typically associated with this code.

If your OCT doesn't have a screening mode, you and your specific OCT manufacturer must find a clinically valid method for performing a procedure less detailed and invasive than a regular OCT. You cannot perform a full OCT capture, eliminate the interpretation and report and call it a screening.

2. Corneal Thickness and Contact Lens Fits

OCTs are already clinically indicated for the measurement of the angles or the crystalline lens, but many can also measure corneal thickness and help with contact lens fittings. While OCT may do a great job in measuring corneal thickness, the scan cannot be coded as corneal pachymetry, CPT 76514, which is defined as "ophthalmic ultrasound, diagnostic; corneal pachymetry, unilateral or bilateral (determination of corneal thickness)." Instead, you must use CPT code 92132, for which there is generally no diagnosis that would support the procedure; you may also find that 92132 is often considered non-covered as experimental and investigational by many insurance carriers.

If you use your OCT for fitting a contact lens, you are also obligated to use CPT code 92132.

3. OCT-angiography

If your OCT can perform angiography, you may be tempted to code for and bill the added angiography component separately as CPT code 92499 (unlisted ophthalmological procedure). However, not only is this inappropriate, but it's also against the CPT definition and interpretation. Instead, CPT code 92134 alone incorporates the angiography component in its base definition of the code, and no additional code is necessary. In fact, coding with both CPT 92499 and CPT 92134 represents a coding and billing error. If paid for both, you would be obligated to return the 92499 payment to the carrier, or to the patient if they paid the extra. An ABN is not appropriate to use, as it cannot be used to split a single service into two parts for the purpose of collecting additional payment from the patient.

With technology swamping the field of optometry, we must carefully evaluate which tools are worth incorporating into our practices—and knowing both the opportunities for better clinical care and the limitations of the coding definitions and rules is the key. ■

Send your coding questions to rocodingconnection@gmail.com.

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Author: Eric T. Brooker, OD, FAAO

Chronic Blepharitis

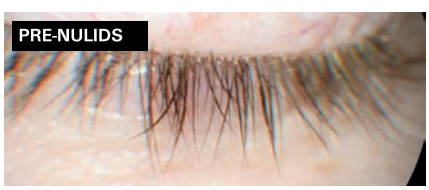
A better approach

Dr. Brooker is the founder of the Advanced Vision Institute in Las Vegas, NV which features a dry eye specialty clinic. He is an adjunct clinical professor for the Southern California College of Optometry, where he received his doctorate of optometry. He is a Fellow of the American Academy of Optometry and has published numerous professional papers, and lectured around the world to ophthalmic surgeons and optometrists on advanced surgical treatments for permanent vison correction, dry eyes, and advanced ocular healing. He also acts as an international ophthalmic consult to several medical device and pharma companies with influence in the eye care industry.

Introduction: Chronic blepharitis is an ocular condition, which is primarily a chronic inflammation of the eyelid margin and also the cause of chronic ocular inflammation. Blepharitis can be anterior affecting the lid margins and skin and the base of the lashes or it can be posterior affecting the Meibomian glands and their orifices. Meibomian Gland Dysfunction (MGD) is chronic changes to the Meibomian glands which are caused by duct obstruction and changes to the meibum secretions. The changes result in tear film alterations that lead to evaporative dry eye and eventually chronic tissue damage. Some signs of blepharitis are chronic redness of eye and lids, loss of eyelashes, distorted lid margins, granular tissue formation, marginal keratitis, limbal opacification, neovascularization of cornea, prominent vessels at lid margin, frothy discharge at eyelid margin, blocking of Meibomian orifices, turbid or absent excretions from Meibomian glands, trichiasis and eventual atrophy of Meibomian glands. Symptoms can present as red eyes, itchy eye lids, swollen eye lids, eyelashes sticking together, burning eyes, fluctuating vision, foreign body sensation, redness of the eyelids, or reoccurring styes. Doctors report 37-47% of patients present with some form of blepharitis and 50% of these patients have an associated dry eye condition.

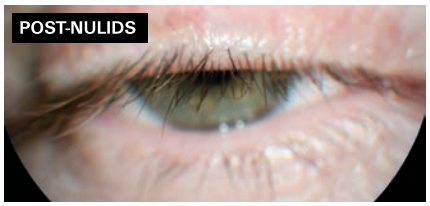
Patient History: This is a 62 year old white male. He presented with complaints of fluctuating vision and occasional redness of eyelids and redness of the nasal conjunctiva. Also reported occasionally eyelashes feel sticky in the morning. He had not tried any additional treatments for this condition nor had he been diagnosed with any ocular diseases in the past.

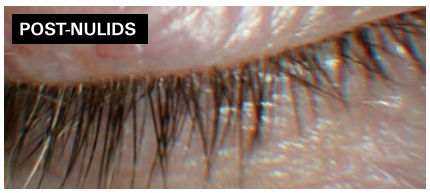
Initial Assessment: On initial examination that patient was found to have UCDVA of OD 20/25 and OS 20/20 in the distance. Biomicroscopy examination revealed 3+ blepharitis superior OD with collarets and a biofilm on the superior and inferior lid. Examination OS revealed 3+ blepharitis with collarets with a biofilm on the superior and inferior lid. Nasal conjunctiva presented with 1+ injection and nasal conjunctival sodium fluorescein staining bilateral in both eyes. He also had an early progressive pterygium nasal in both eyes with trace superior punctate staining bilateral. His TBUT in the right eye was 8 seconds and 9 seconds in the left eye. Patient was diagnosed with chronic blepharitis



with an associated evaporative dry eye condition that has begun to cause chronic tissue damage.

Treatment Strategy: This patient had recently retired and being prior military was very regimented in his lifestyle. He vowed that he was going to eliminate the blepharitis from his eyelids. He was started on a tea tree based lid scrubs QHS OU following his normal face watching regimen along with preservative free Oasis Tears . He was also instructed to change his pillow cases once a week. After one week of dedication to using lids scrubs the patient returned to clinic. At this visit he had improved the condition to 2 + blepharitis and the biofilm was still present. We all concurred that this was a chronic condition that was not going to be easy to control. The manual lid scrubs despite the consistent usage were not effective at maintaining his condition. We recommended the patient undergo an in office blepharoexfoliation treatment with our lead doctor to get rid of the biofilm and get the patient back to baseline. Following the patient's consent the procedure was performed successfully and he was discharged in good condition. He was also started on Tobradex drops bid OU to keep down inflammation and attempt to improve the presentation of his ptergium that had developed over several years. He was also instructed to continue with manual lid scrubs bid OU. Upon his 2 week return visit the patient had improved significantly but the blepharitis had already returned to a level 1+ bilaterally in the upper lashes. Patient education was given regarding the chronic aggressive nature of his condition. The doctors explained the need for a more aggressive solution that





could address his blepharitis while also promoting improvement of his Meibomian gland secretions. We recommended NuLids automated blepharitis treatment combined with Hypochlorous gel. Due to the regimented nature of this patient we knew that equipped with the appropriate at home treatment he would be successful.

Outcomes: Following 1 week of treatment with the NuLids device prior to sleep, the patient returned to clinic for a follow up assessment. On assessment the patient was found to have completely eliminated the blepharitis on both the superior and inferior lids of both eyes and his conjunctival injection had reduced. His TBUT had increased to 16 seconds in both eyes. The patient reported that his eyes felt rejuvenated and more moist and were no longer stuck together in the mornings. This case report demonstrates the effectiveness and superiority of automated lid hygiene versus manual lid scrubs

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42nd Annual Technology Report

12 Ways to Get More Out of Your OCT

This handy tool can help you assess everything from peripheral retinal lesions to the tear meniscus. Here's how.

By Carolyn Majcher, OD, Jeannette Wong-Powell, OD, and Amy Conner, OD, MBA

OCT can add value to any optometric practice; it can provide you with an extra boost of confidence in your diagnosis and decrease your referral rates. Even if you are already using OCT in your practice, you might not be maximizing its utility. These novel clinical applications of both anterior and posterior segment OCT will help you get the most out of your investment.

1. Detect Objective Ocular Torsion

For patients complaining of vertical diplopia, OCT may aid in detecting and quantifying objective ocular torsion. In cases of ocular torsion, optic nerve OCT imaging shows a separation, or shifting, of the nerve fiber layer "TISNT" graphs of the two eyes, assuming that your OCT model does not automatically correct for torsion effects (*Figure 1*).

When a vertical tropia or phoria is present, excyclotorsion of the hyper eye is suggestive of superior oblique palsy, unlike skew deviation, which would be expected to exhibit incyclotorsion. Clinicians should compare the amount of excyclotorsion noted with subjective measures (double Maddox rod testing) and objective measures to aid in the differentiation between decompensated congenital superior oblique palsy and truly acquired cranial nerve IV palsy. If the amount of subjective excyclotorsion is far less than the amount of objective excyclotorsion, a decompensated congenital palsy is likely. However, if the amount of subject torsion equates the degree of objective excyclotorsion, an acquired palsy in need of additional investigation in the absence of trauma in likely the cause.

2. Image the Lamina Cribrosa

Incorporating these images as part of your glaucoma evaluation can add tremendous value. Enhanced-depth-imaging (EDI) OCT is a method of preferentially imaging deeper structures such as the choroid or the lamina. Many OCT instruments automatically incorporate EDI-OCT or have an EDI "check-box" option. You can also enhance your view of deeper structures using one-line raster scans that maximize imaging averaging. Two measures of the lamina can be of predictive value: laminar thickness and lamina cribrosa depth (*Figure 2*).

The biomechanical theory of glaucoma postulates that ganglion cell axons are crushed as the lamina distorts and bows posteriorly due to intraocular pressure (IOP) elevation.[1,2] Theoretically, thinner or weaker laminas will bow more with even modest IOP elevation. Investigations using EDI-OCT laminar imaging show that the lamina tends to be thinner in eyes with primary open-angle glaucoma (POAG)

compared with normal eyes and laminar thinning correlates with the degree of structural and functional glaucomatous damage.[3-5] One study found a mean laminar thicknesses of 234µm, 179µm and 156µm in eyes with early, moderate and advanced stage glaucoma, respectively.[3] Another study found that a faster rate of nerve fiber layer thinning was associated with a laminar cribrosa depth greater than 490µm, and smaller lamina cribrosa thickness in POAG eyes. Therefore, EDI-OCT measures may have predictive value for glaucomatous progression.[6]

3. Assess Peripheral Retinal Lesions

Don't be afraid to image peripheral lesions with off-axis OCT raster scanning, as it can help you to differentiate between lamellar and full thickness peripheral retinal holes within areas of lattice degeneration (*Figure 3*). It can also help you measure the extent of subretinal fluid surrounding atrophic holes and monitor progression. Along with scleral indentation and Goldmann 3-mirror evaluation, peripheral OCT imaging can aid in distinguishing a vitreoretinal tuft from a full thickness flap tear that requires barrier laser treatment (*Figure 4*). This OCT technique also allows you to differentiate retinoschisis from a retinal detachment (*Figure 5*).

4. Determine Macular Status in Retinal Detachment

In rhegmatogenous retinal detachment (RRD), the status of macular attachment dictates the urgency of referral for surgical repair, as macula-on RRDs constitute emergent referral. However, when a detachment is shallow, the true extent of the detachment and its proximity to the macula can be difficult to assess clinically. Additionally, the macula can be challenging to assess clinically in tractional retinal detachments resulting from proliferative conditions such as diabetic retinopathy. Concurrent vitreous hemorrhage may result in decreased macular visibility with ophthalmoscopy. OCT can help to delineate the extent of retinal detachment and the degree of macular involvement (*Figure 6*). Although the vitreous hemorrhage can limit OCT imaging, depending on the extent and location, even a poor quality OCT in these cases can

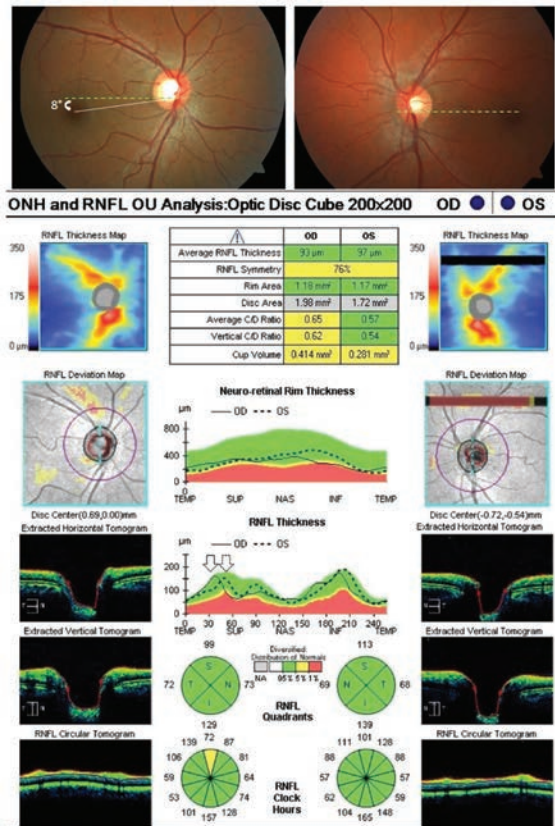

Fig. 1. Optic nerve OCT shows objective excyclotorsion OD (leftward shifting of the right eye TISNT graph in relation to the left) in a patient complaining of intermittent vertical diplopia with a low magnitude right hyperphoria. No subjective torsion was noted via double Maddox rod testing, suggestive of a congenital superior oblique palsy.

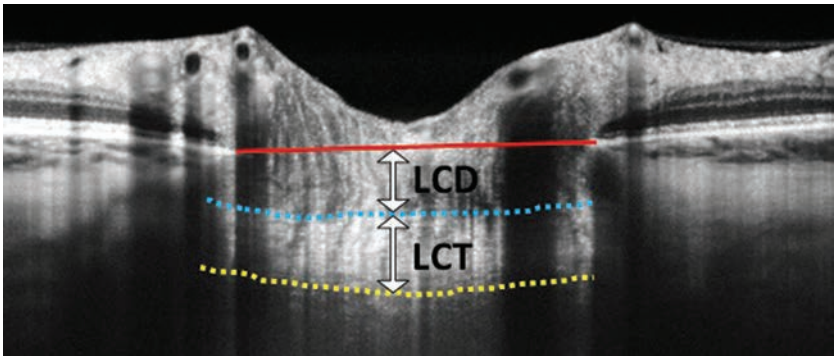

Fig. 2. In this EDI-OCT through the optic nerve, the lamina cribrosa thickness (LCT) is measured from the anterior border (blue dashed line) to the posterior border (yellow dashed line) of the lamina cribrosa. Lamina cribrosa depth (LCD) is measured along a perpendicular line from the anterior border of the lamina to a reference line that connects the edges of Bruch's membrane (red solid line).

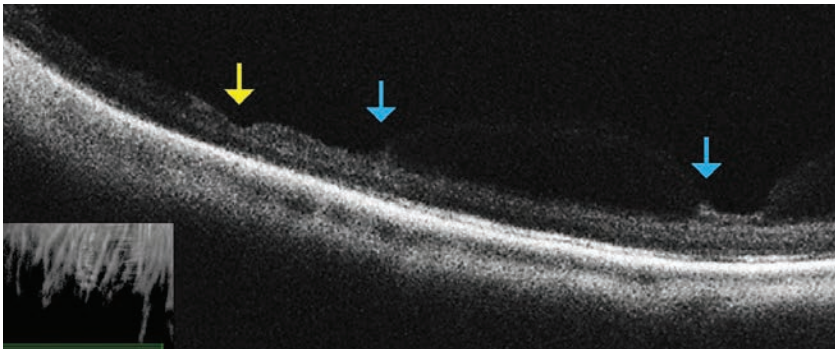

Fig. 3. This OCT raster scan taken through an area of lattice degeneration in the inferior peripheral fundus shows retinal thinning (yellow arrow) bordered by vitreous condensation/vitreoretinal traction. OCT does not reveal any full-thickness breaks.

provide substantial information that is relevant when determining referral urgency (*Figure 7*).

5. Identify Posterior Shaffer's Sign

OCT can image small cells within the vitreous that take on the appearance of "falling ash" (*Figure 8*).[7] Cells in this region may represent pigmented cells suggestive of a retinal break in the setting of an acute posterior vitreous detachment (PVD), similar to "tobacco dust" seen in the anterior vitreous, or inflammatory cells in cases of vitritis. They may also represent red blood cells in the instance of vitreous hemorrhage. If seen in the presence of an acute symptomatic PVD, they suggest that a retinal break is likely present and the patient should have a through fundus examination that includes scleral depression and Goldmann 3-mirror retinal assessment. Clinicians should be careful to distinguish between age-related vitreous floaters and cells on OCT—vitreous cells are more numerous and smaller than age-related floaters.

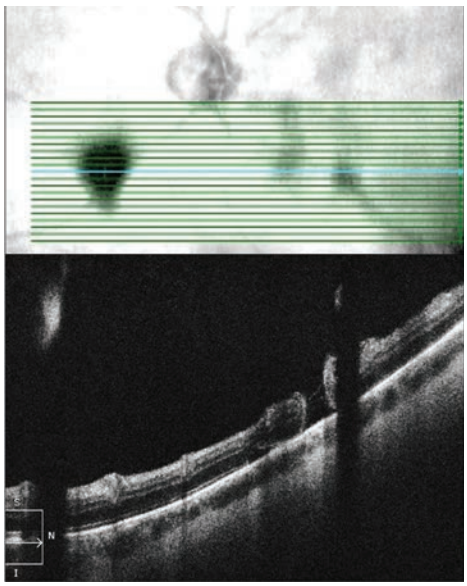

Fig. 4. OCT raster scan reveals a subclinical retinal tear with minimal surrounding subretinal fluid in a patient with acute onset vitreous hemorrhage and proliferative DR.

6. Differentiate Nonproliferative from Proliferative Diabetic Retinopathy

Widefield, montage OCT angiography (OCT-A) is an invaluable tool for detecting early neovascularization (*Figure 9*). A recent study found that approximately half of eyes graded as severe nonproliferative diabetic retinopathy (NPDR) based on fundus photography and clinical exam had areas of preretinal neovascularization when imaged with widefield OCT-A.[8] Making the distinction between proliferative and nonproliferative DR is important for management and monitoring, especially as practice trends are moving toward earlier anti-VEGF therapy. Patients with significant, widespread peripheral nonperfusion should be monitored more closely than those without.

7. Assess the Anterior Chamber and Image the Angle

Anterior segment OCT (AS-OCT) imaging gained popularity in 2005 and 2006 with the FDA approval of two commercial devices. Both were specifically designed to image the anterior segment with a longer wavelength (1,310nm) than posterior segment OCT (about 800nm) to provide deeper imaging of anterior tissues, including the scleral spur. Several parameters were proposed to use AS-OCT as screening device for anatomically narrow angles.

Adaptive lenses were soon developed to allow posterior OCT devices to also capture the anterior segment. Today, most devices have a built-in anterior segment imaging module, usually capable of scanning 6mm wide areas to provide limbus-to-limbus imaging of the anterior chamber as well as localized scans of the cornea and conjunctiva.[9]

8. Image Suspected Anterior Segment Tumors

On ultra-high resolution OCT, ocular surface squamous neoplasia shows three distinctive features: a hyper-reflective thickened epithelial layer, an abrupt transition from normal to abnormal epithelium and a distinct plane between the lesion and the tissue beneath.[10] Ultra-high resolution OCT can image down to just 3µm, while most commercially available equipment has a resolution of 4µm to 5µm, making this application possible only in devices with above-average resolution. AS-OCT

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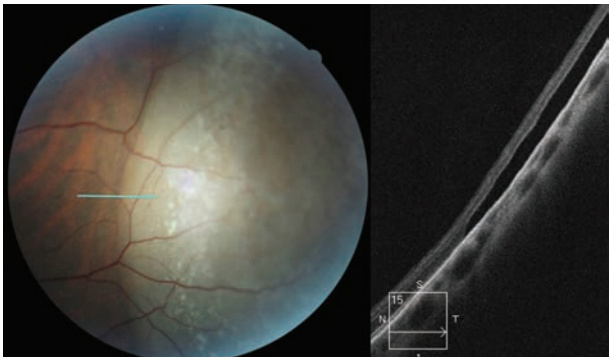

Fig. 5. OCT raster scan taken though an elevated peripheral retinal region in a 26-year-old female diagnosed with retinoschisis one year prior. OCT reveals a full-thickness RRD caused by a combination of inner and outer layer holes in a preexisting retinoschisis.

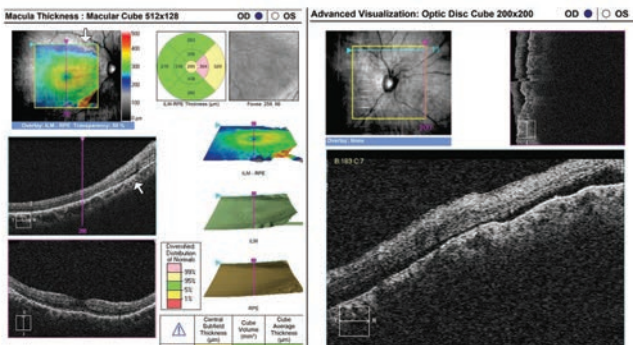

Fig. 6. This 35-year-old with a history of blunt trauma OD and 20/30+ acuity was referred for evaluation of IOP elevation. OCT reveals a very shallow RRD involving the superior aspect of the optic nerve head cube (right) and superior macula (left, arrows). Referral was emergent.

can also image small hypopigmented tumors, although ultrasound biomicroscopy has superior penetration and should be used in highly pigmented and ciliary body suspected tumors.[11,12] Researchers are currently investigating OCT-A as a noninvasive and cost-effective method to assess vasculature characteristics in potentially malignant iris lesions.[9]

9. Assess The Anterior Segment Peri- and Postoperatively

AS-OCT is the only single instrument that can image all five corneal layers—epithelium, Bowman's layer, corneal stroma, Descemet's membrane and endothelium—making it a valuable diagnostic and perioperative tool. AS-OCT images are routinely used to assess intracorneal ring placement, Descemet's stripping endothelial keratoplasty (DSEK) and refractive surgery outcomes.[13-16] Specifically, AS-OCT allows for accurate measurement of flap thickness, residual stromal bed thickness, and detection of flap-related complications for preoperative planning. After myopic LASIK, clinicians can use AS-OCT to calculate the corneal power and accurately select the intraocular lens power in cataract surgeries.[9,16]

AS-OCT is also useful as a prognostic predictor following trabeculectomy. Successful filtering blebs exhibit certain slit-lamp examination features such as moderate elevation, diffuse extent, scarce vascularity and conjunctival microcysts. AS-OCT imaging shows that failed blebs tend to have a highly reflective bleb wall surrounding the internal fluid-filled cavity, leading to the belief that a thinner bleb wall may allow for better aqueous flow and lower IOP.[17] As a noninvasive imaging tool that carries no risk of infection, AS-OCT is advantageous over conventional ultrasound biomicroscopy bleb examination.[18,19]

When a laser peripheral iridotomy (LPI) is small or placed far in the periphery of the iris under arcus, assessing patency can be a clinical challenge. However, a simple AS-OCT raster scan can quickly and easily assist the clinician in confirming LPI patency (*Figure 10*).

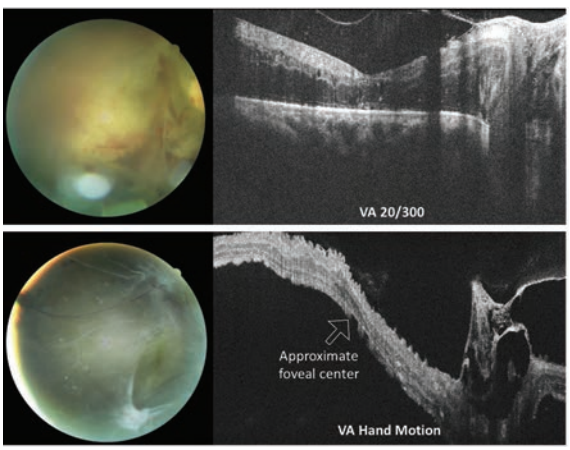

Fig. 7. OCT raster scans through the optic nerve and macula in two different patients with proliferative diabetic retinopathy with vitreoretinal traction of the right eye. In both instances, macular involvement was uncertain based on clinical examination alone. The top eye suffers from severe macular edema of an attached macula. Conversely, the macula of the bottom eye is detached. Acuities were top 20/300 (top) and hand motion (bottom).

10. Image Corneal Disorders

Assessing the depth, size and affected layers of corneal lesions with AS-OCT offers many advantages over slit-lamp examination or photography. With a minimum resolution of up to 5µm, depending on the instrument, you can determine the exact

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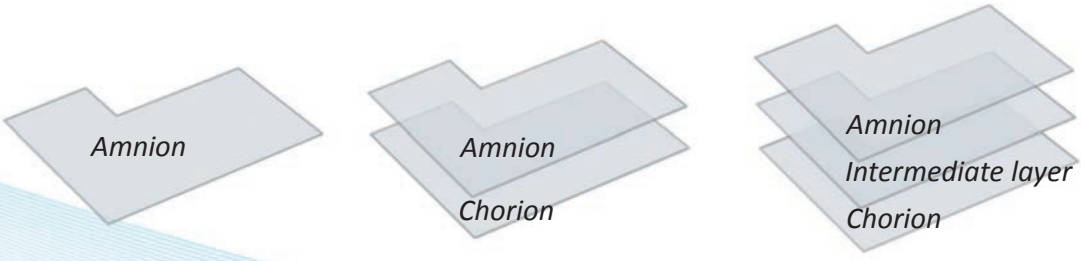

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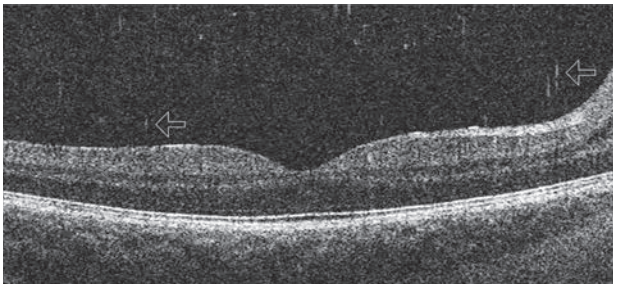

Fig. 8. This posterior Shaffer's sign shows cells in the posterior vitreous that appear as "falling ash" (arrows).

involved layer and depth of any corneal lesion with certainty. This can be particularly important in the management of corneal infections, corneal foreign bodies and keratoconus.[19,20] One of the most severe complications of advanced keratoconus is corneal hydrops, stromal edema, due to a tear in Descemet's membrane. Without AS-OCT, it can be difficult to assess corneal integrity of posterior corneal layers by conventional slit-lamp examination in the presence of scarring or haze anterior to the layer in question (*Figure 11*).

Features of microbial keratitis on AS-OCT include infiltrates that appear as hyper-reflective areas in the corneal stroma. Researchers also note that you can see anterior chamber inflammatory cells with AS-OCT.[21] In addition, you can closely monitor corneal and infiltrate thicknesses when measured with the caliper function.

OCT can help to establish the depth of a demarcation line that occurs following corneal collagen crosslinking, and you can use it as a keratoconus screening tool to detect focal thinning.[22-25]

Corneal dystrophies are categorized relative to the affected corneal layer: epithelial-subepithelial, epithelial-stromal, stromal and endothelial dystrophies. Researchers have found that OCT images correlate with histological sections, and can serve as a reference to confirm a clinical diagnosis and track changes over time. As an example, Reis-Bückler's corneal dystrophy shows a hyper-reflective material at the level of Bowman's layer on OCT.[26]

11. Tear Meniscus and Dry Eye Evaluation

Although Schrimer's scores and tear break-up time are consistently used as baseline for dry eye disease, investigators note that tear meniscus height (TMH) taken with OCT can more precisely label dry eye (*Figure 12*). TMH should be greater than or equal to 0.2mm (or 200µm)—anything below this number is suspicious for dry eye.[26]

12. Corneal Epithelium Thickness Mapping

Epithelium thickness mapping (ETM) with OCT recently received FDA approval.[27] This scanning tool will allow you to follow irregularities in dry eye, epithelial shape changes (i.e., epithelial bogging under a contact lens tear reservoir) under a contact lens, detect early keratoconus changes and any anterior surface deformities, such as Salzmann's nodules.[27,28]

In Conclusion

This versatile instrument, and its ever-progressing iterations, has become integral to many aspects of

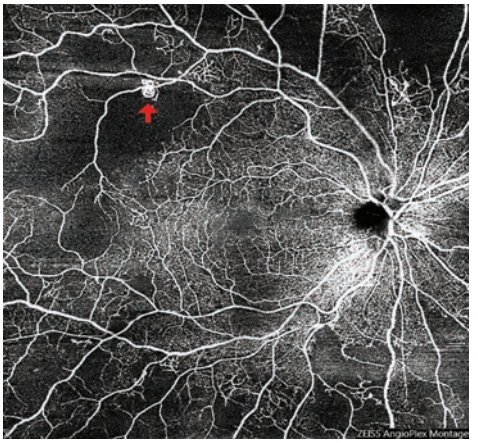

Fig. 9. Widefield OCT-A of an eye with subtle neovascularization (arrow) led to a diagnosis of early proliferative diabetic retinopathy. Also note the significant peripheral retinal nonperfusion.

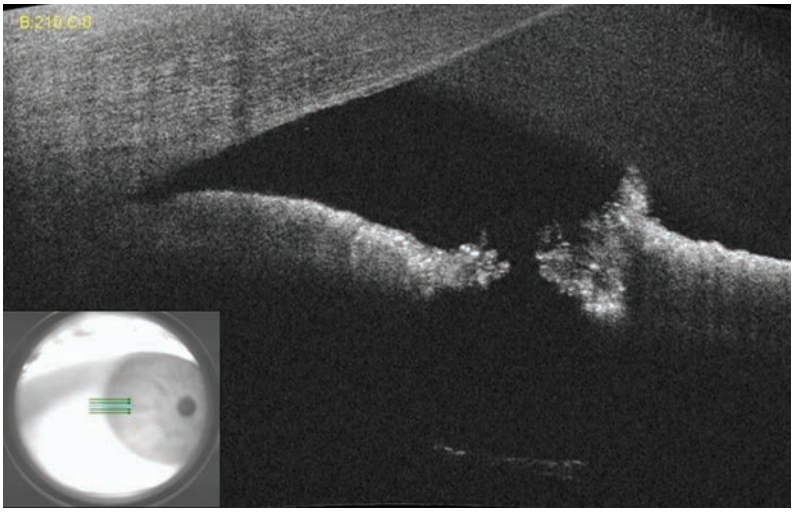

Fig. 10. AS-OCT of a small laser peripheral iridotomy reveals a full thickness hole. Lens zonules can be seen in the bottom scan due to light penetration through the patent iridotomy.

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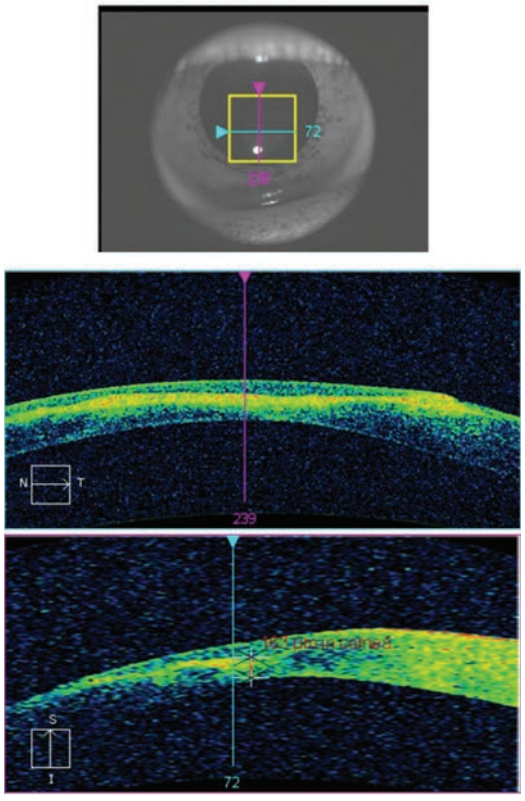

Fig. 11. This AS-OCT scan of a 28-year-old Asian patient with advanced keratoconus and moderate-to-severe pain demonstrates intact endothelial and posterior stromal layers while showing increased reflectivity associated with stromal scarring. In this case, AS-OCT confirmed an intact endothelium and Descemet's membrane. As an added advantage, corneal thickness was measured accurately at the thinnest location using the caliper tool, thereby providing a reliable baseline for follow-up comparison. In light of the anatomical state of the cornea, the patient's symptoms were attributed to epithelial disruption overlying the apex of corneal steepening.

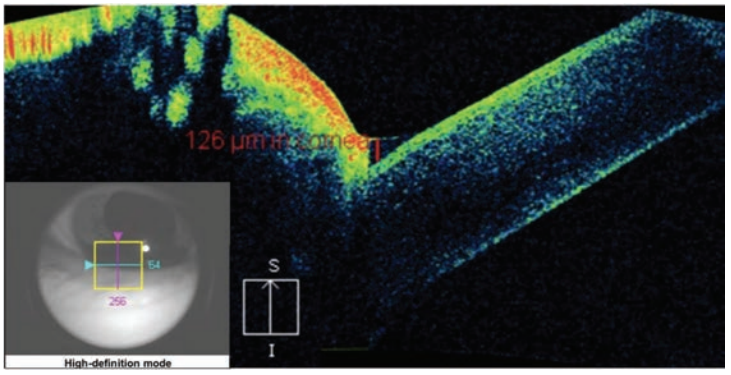

Fig. 12. The crosshair of this anterior segment cube scan taken at the lid margin shows the tear meniscus height at the fornix of the globe and the palpebral conjunctiva. The measurement of 126µm is considered dry eye.

the ocular exam. While it will never replace your clinical acumen, it can augment your clinical findings and help you diagnose and track any number of anterior and posterior conditions. It's time to take your relationship with your OCT to the next level. ■

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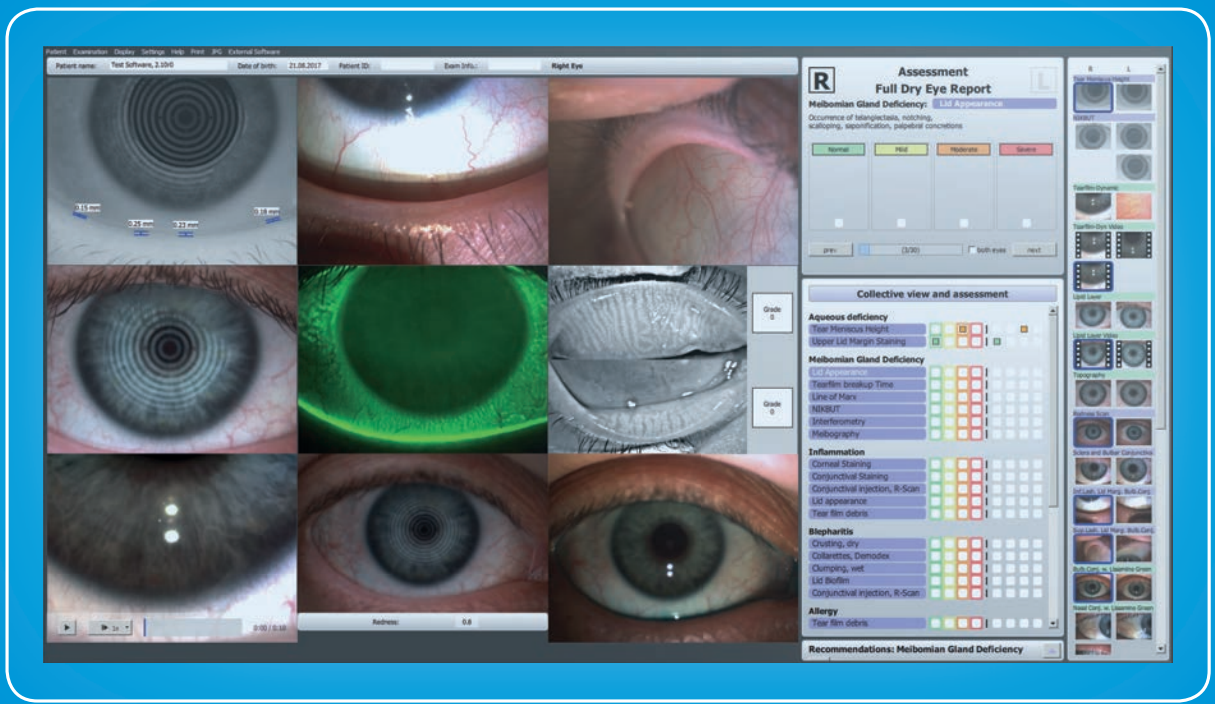

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42nd Annual Technology Report

Fit Specialty Contact Lenses With OCT Precision

New capabilities for your tried-and-true tool can help you find the right fit the first time. **By Amy Conner, OD, MBA**

Anterior segment OCT (AS-OCT) has come a long way in recent years. Clinicians are now using it to image everything from the tear film to the conjunctiva and cornea. Not surprisingly, many optometrists now use it to help them assess specialty contact lens fits in particular. As specialty lenses, such as scleral contact lenses and hybrid lenses, continue to gain in popularity, AS-OCT can be increasingly helpful in the lens assessment process. AS-OCT makes what was once a challenging clinical encounter a quick, easy and successful experience.

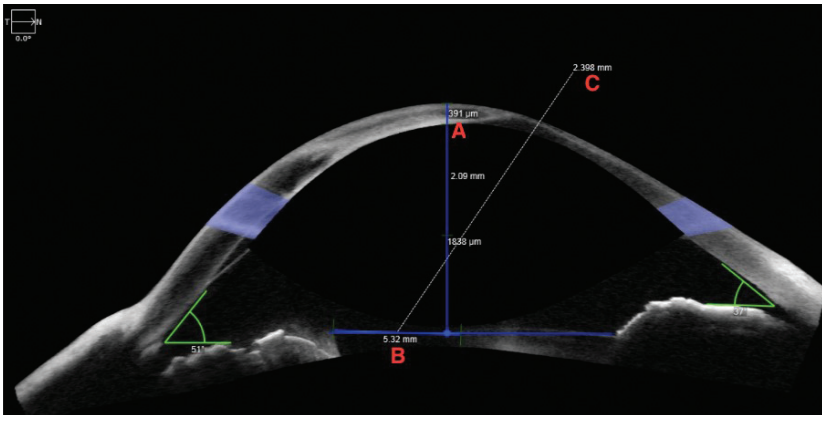

Fig. 1. This AS-OCT, taken with an anterior chamber adapter lens, shows a sagittal depth of 391µm (A). Pupil size is 5.32m (B), and angle Kappa is 2.398m (C).

One study found complications in addition to infectious keratitis associated with specialty contact lens wear could cause hypoxia, inflammation, mechanical influence, deposition and visual blur.[1] These complications can be better understood using an AS-OCT map of the contact lens in relation to the cornea. Using AS-OCT to image a specialty contact lens on the cornea can help practitioners decrease any adverse events related to contact lens wear.

Initial Lens Choice

Clinicians can use their AS-OCT to better determine several initial lens parameters, including base curve and the need for back toricity:

Base curve. AS-OCT can aid contact lens practitioners in fine-tuning an initial starting point for pulling a properly fit lens from a fit set based on sagittal height or base curve. Clinicians can use the precise sagittal depth (sag) generated from the tip of the cornea down to the 15mm horizontal chord to get a better idea of the necessary vault height. The sag can be used to match an initial fitting lens from the fitting set. This 15mm chord measurement can also show pupil size and angle kappa, which may be helpful in designing optic zones or multifocal contact lenses (*Figure 1*).

Back toricity. When fitting a scleral contact lens,

most practitioners do not know when to choose a back surface toric lens (toric haptic) versus a spherical back surface lens. Back surface toricity can help to stabilize the lens for comfort and best vision, while front surface toricity is only for optical outcome.[2] Spherical scleral landing zones on a toric sclera would exhibit a poor-fitting, with-the-rule appearance; the lens will touch the horizontal meridian and lift off in the vertical meridian.[3] Landing on both meridians can be achieved only with rotationally asymmetric toric haptic scleral landing zones, which are necessary to ensure lens stabilization and centration.[4] AS-OCT can be an excellent tool to use when deciding to use a toric haptic in fitting scleral lenses, as it can help you decide on a starting point. Currently, most scleral fitting sets come with a toric haptic component.

The average peripheral corneal, limbal and scleral angle is approximately 38.4° in normal eyes.[5] If a patient's angle measures landing angle is greater than or equal to 41° at horizontal chords between 10mm and 15mm, a scleral lens is likely to have limbal bearing and scleral toricity (*Figure 2*). These large insertion angles may warrant a toric haptic lens with additional vault added to the limbal zones.[5]

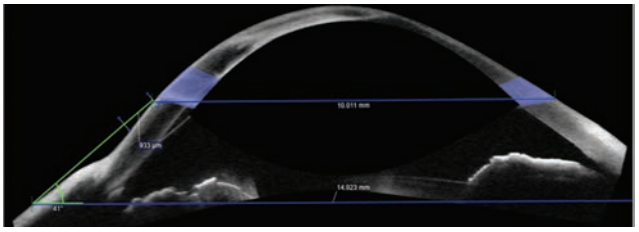

Fig. 2. The angle of the peripheral cornea, limbus and sclera shows a steeper temporal corneo-scleral insertion and flatter nasal landing zone. The angle caliber tool is measured between chord 10mm and 15mm. The 41° here suggests adding more limbal clearance and a toric back surface lens.

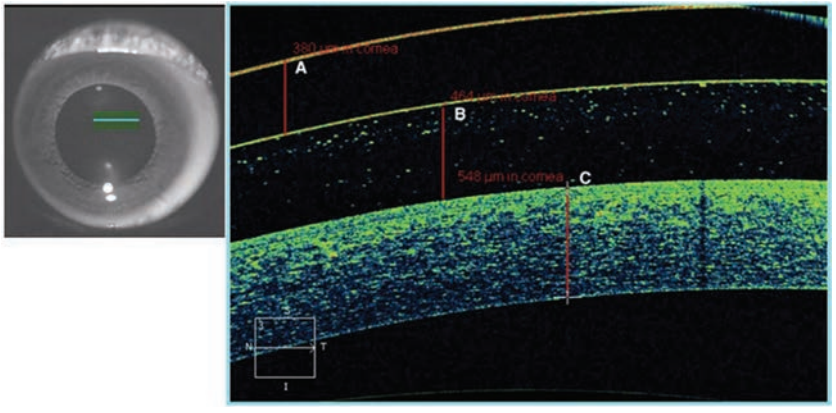

Fig. 3. This anterior segment 5-line raster shows tear clearance below a scleral contact lens. The center thickness of the lens vaulting the cornea is 380µm (A). The tear clearance of 464µm shows midday fogging particles trapped in the tear layer (B). The center corneal thickness is 548µm (C).

Tear Layer Clearance

One of the most widely used views to evaluate specialty lenses with an OCT is the central clearance of the lens vaulting over the cornea. Research suggests a well-fit scleral lens should have a 100µm to 200µm tear layer in the center and 10µm to 50µm over the limbus.[1,6,7]

Central clearance is important because of its impact on a patient's visual outcome and ocular health. One study found excessive tear clearance is associated with increased leukocytes, causing midday fogging and hypoxia.[6] Every 50µm increase in clearance has an associated 2.24 times higher odds for midday fogging and possible hypoxia.[6] A patient with a tear clearance of 464µm, for example, is 20.79 times more at risk of midday fogging (*Figure 3*). In contrast, minimal tear layer clearance can put the patient at risk of scarring or discomfort from bearing on the cornea. AS-OCT can help image touch of the contact lens on the cornea, an optimal tear clearance after settling and a tear layer in excess that could result in hypoxia (*Figure 4*).

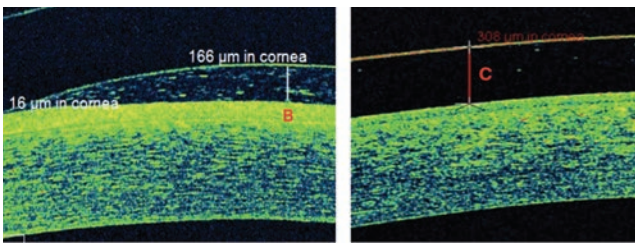

Fig. 4. This anterior segment 5-line raster of the tear clearance below a scleral contact lens shows touch (A), good clearance of 166µm (B) and excessive clearance of 308µm (C).

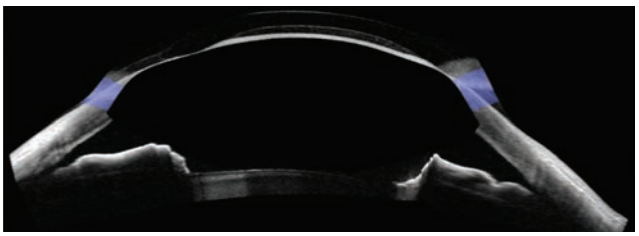

Fig. 5. This AS-OCT shows a poor-fitting hybrid lens with decentration, which is causing touch.

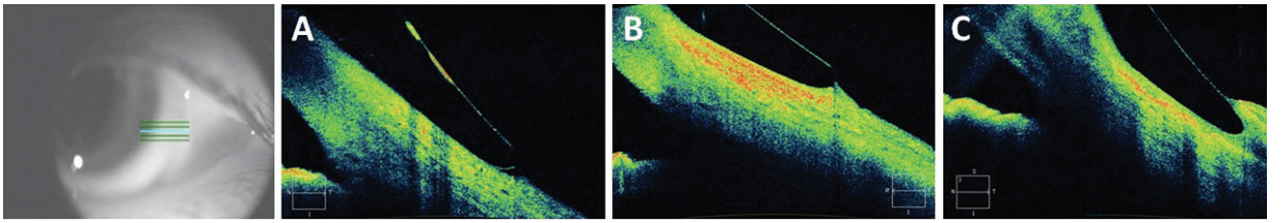

Fig. 6. Anterior segment 5-line raster images show a scleral lens edge that lands too flat and lifts away from the conjunctiva (A); good, stable landing aligned with conjunctiva (B); and impingement into the conjunctiva with a steep landing zone edge (C).

Computing the theoretical oxygen transmissibility for contact lens material, contact lens center thickness, plus the amount of tear layer thicknesses is of future clinical interest to determine lens-tear system characteristics that will alleviate corneal hypoxia.[7] For now, controlling tear layer surface area is the one way to decrease risks of hypoxia.

Fit vs. Corneal Shape

Using an overall anterior chamber view can benefit a specialty contact fit as it helps the practitioner get a better sense of the relationship between the shape of the eye and how the contact lens settles (*Figure 5*). This view allows practitioners to see where adjustments need to be made to mimic the overall globe shape.

Pachymetry

Clinicians can use this anterior segment tool to rule out any corneal edema induced by contact lens wear. Corneal thickness should be measured before specialty contact lens wear and within six months after wear to rule out any edema caused by the contact lens. If the central corneal thickness number increases over time, then the corneal edema is induced by contact lens wear.

Current research suggests 5% is an acceptable amount of edema for daily wear contact lenses.[8] Beyond 5% edema, striae appear in the posterior stroma.[9] Edema greater than 15% is considered pathological, and visual acuity becomes cloudy. Pathological edema can lead to infiltrates, epithelial microcysts, pain, neovascularization and destruction of endothelial pumps.[9]

Peripheral Scleral Lens Landing

Scleral lenses need to distribute their weight along the conjunctiva. Lenses that fit loosely or flat at the scleral landing zone can cause excessive movement, lid irritation and vision fluctuation (*Figure 6A*). Lenses fit too tight or steep in the scleral landing zone can cause pain and lack of tear exchange (*Figure 6C*). Landing zones should land parallel to the conjunctiva for best comfort and fit (*Figure 6B*).

Limbal Clearance

The human eye can only see 50µm to 60µm under evaluation with a slit lamp.[10] Using an OCT can precisely show by how many microns the scleral lens clears the limbus, with an indeal clearance of 10µm to 50µm (*Figure 7*). Excessive limbal clearance can cause corneal bogging and conjunctival prolapse. Conversely, hypoxia, low tear exchange and discomfort can occur when the limbus is not cleared properly.

In fitting and evaluating specialty contact lenses, AS-OCT may be your new best friend. With a little practice, these new imaging techniques can show you the exact relationship between the lens and the eye—giving you the information you need to get the fit right. ■

Dr. Conner works as a therapeutic glaucoma optometrist at Dallas Eye and Ear in Dallas.

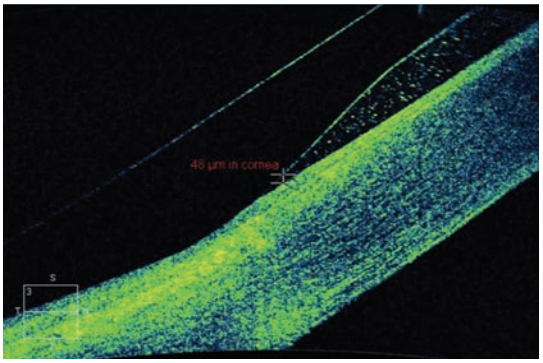

Fig. 7. Anterior segment 5-line raster shows limbal clearance of a scleral lens vaulting the limbus of the cornea to scleral junction.

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42nd Annual Technology Report

At the Crossroads of Technology and Tradition

Tried-and-true retinal exam techniques are still relevant, even with the inclusion of ever-more precise gizmos. **By Shreya Jayasimha, OD**

The pace of technological advancement has granted optometrists a more targeted, more robust and more comprehensive ability to diagnose, evaluate and treat a plethora of ocular pathologies. Specifically, new retinal devices may supplant or enhance their technological predecessors, all of which serve to benefit optometrists and patients alike.

Where ODs once had to rely on sometimes invasive techniques, we now have non-invasive options. Ocular examinations that previously required a considerable amount of time can now be done instantly. Some diagnostic and monitoring procedures that had in years past required comanagement with ophthalmology are now in the hands optometrists alone.

Technology isn't only changing what optometrists can accomplish; it's changing what optometry is.

This article delves into the nuances and benefits of the machines that are guiding the way, the traditional techniques ODs still must master and how a combination of the old and the new can be used in the modern clinic.

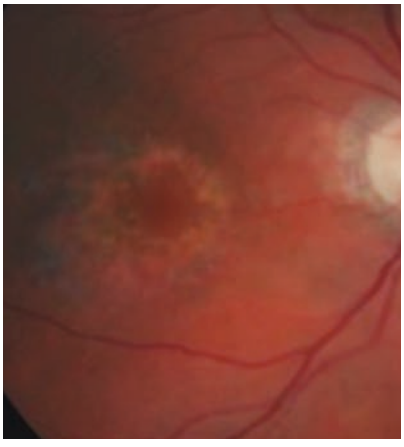

Photo: Marion Demeritt, OD

This photo of a dilated fundus exam portrays a patient with parafoveal ring-shaped "bull's eye" retinal pigment epithelium defects secondary to Plaquenil toxicity.

Dilated Fundus Exam vs. Widefield Retinal Imaging

The one practice that predates every mode of technology is a dilated fundus examination. A dilated exam remains the standard practice, and its use is not overridden by the development of new technologies. But that doesn't mean it's perfect. This traditional method expands the ability to view the retina in its entirety and allows for a better appreciation of the depth and contour of many clinical entities. However, dilation can take approximately 15 minutes to take effect and can last anywhere from two to four hours, rendering the patient temporarily light sensitive and unable to see up close.[1] Some patients may even experience "tightness" or "heaviness" of their eyelids, which may serve to hinder their ability to drive immediately following their eye examination.[1] According to the National Eye Institute, it is recommended to have a dilated eye exam annually after the age of 40, which also means experiencing these side effects on an annual basis.[1,2]

Nevertheless, dilated eye exams play an integral role in delineating

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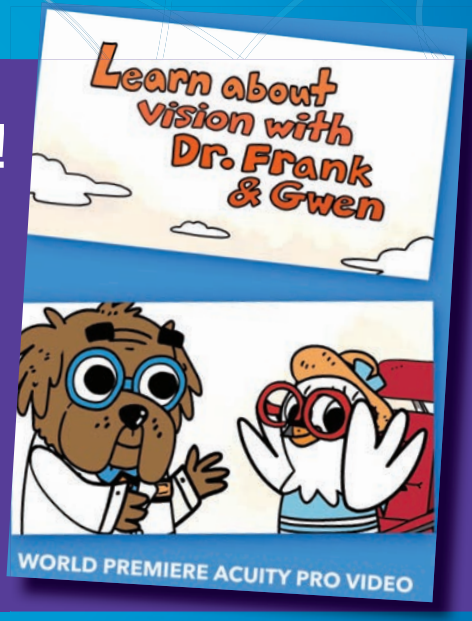

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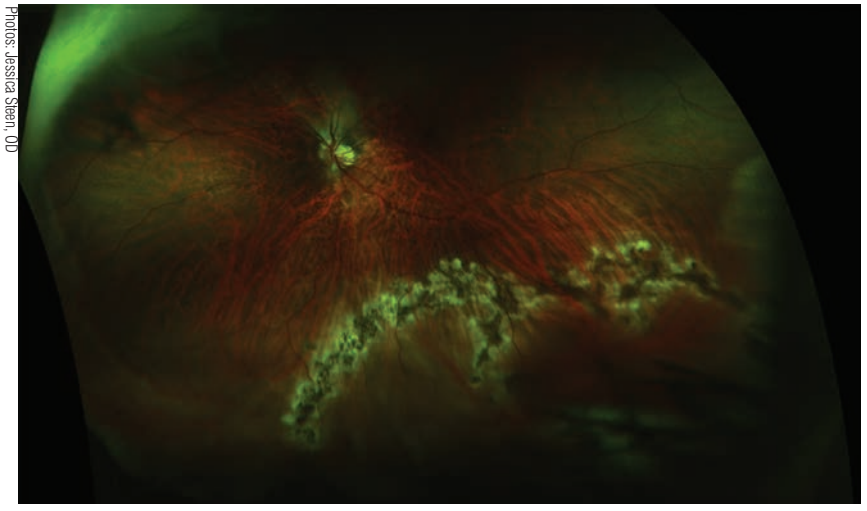
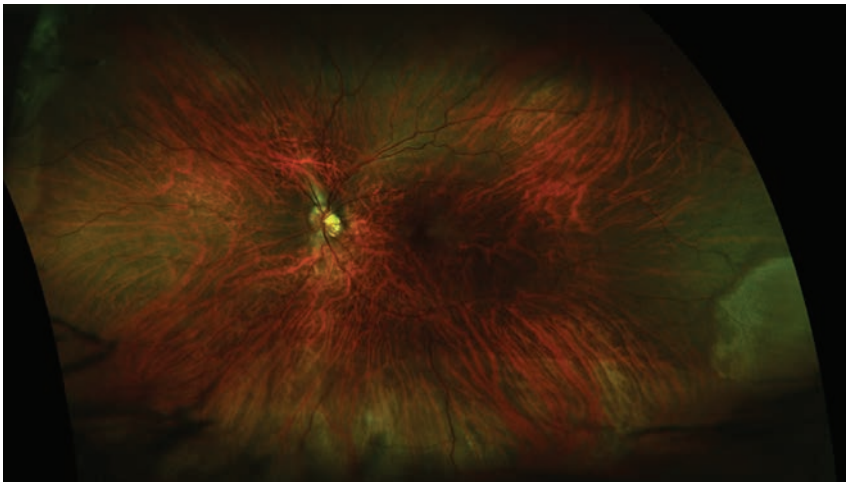
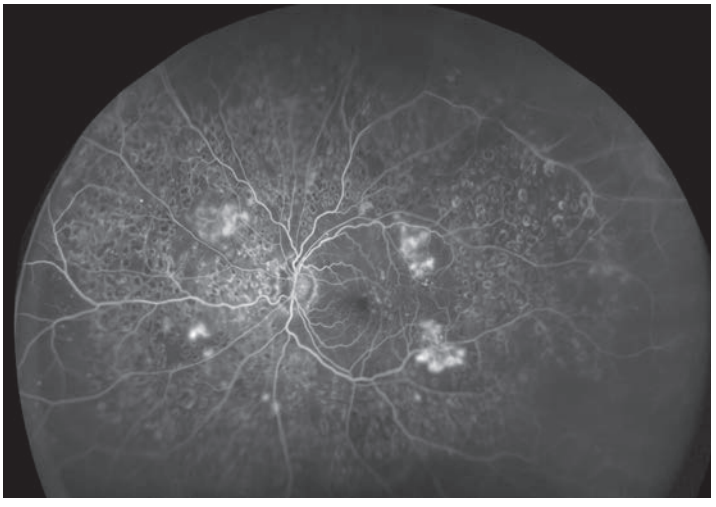


This retinal break with retinal detachment, seen post-treatment (above), was diagnosed using dilated funduscopy, but was not apparent pre-treatment (below) using ultra-widefield imaging.





This ultra-widefield image shows an expansive view of a retina with severe diabetic retinopathy.

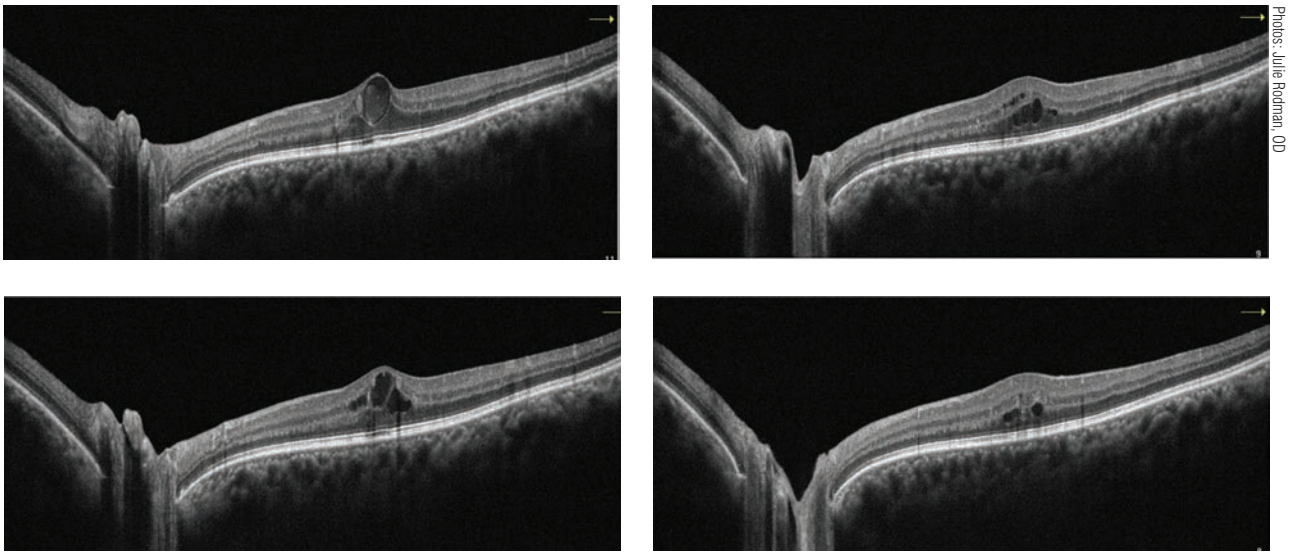
potential problems that imaging devices may flag. Many retinal imaging modalities may produce false positives and false negatives that can skew our management decisions in the wrong direction. As such, dilation allows for further, accurate analysis that will yield the best possible ocular and systemic outcomes for all patients alike.

As a supplement to dilated exams, ultra-widefield high-resolution retinal imaging can non-invasively help to evaluate, diagnose and document the peripheral retina and may also capture peripheral retinal pathology, such as capillary non-perfusion, neovascularization and retinal detachments, that may otherwise go undetected through traditional techniques. The technology is equipped with multiple imaging options including color, red-free, choroidal and autofluorescence features.[2,3] Imaging views can also be modified to include standard 200˚ single capture, auto montage and stereo imaging.[3]

These imaging devices serve to benefit the clinical practice and patients in several ways. To begin, it can be used as a visual aid that can facilitate treatment and management discussions, both of which serve to increase patient compliance. One study suggests that widefield imaging may be 30% better at locating peripheral retinal lesions such as holes, tears, nevi and hemorrhages due to its ability to provide 200-degree temporal and nasal imaging.[2,3]

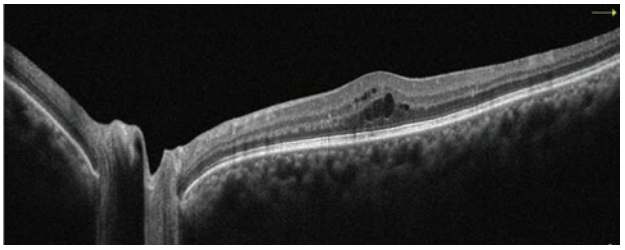
In addition, widefield imaging allows for visual documentation. This can track the progression or resolution of retinal abnormalities, which can crucially affect management protocol.

Finally, widefield imaging is time-efficient as it can be used to screen the retina for possible



Photos: Julie Rodman, OD

This collection of OCT images examplify how the technology is able to portray a diabetic retinopathy patient through the course of several visits.



abnormalities, which can be further analyzed through traditional dilating techniques.

Caveats to widefield retinal imaging include patient cooperation, possible artifacts, training needed to capture good retinal images and poor quality of images due to media obstruction.[3] Additionally, this form of imaging, while convenient and useful, may give patients the idea that it surpasses or even replaces dilated exams. With that said, if optometrists are going to offer this service, it's vital to explain to patients that these images are used to supplement rather than replace dilated fundus examinations.

Slit Lamp Indirect Ophthalmoscopy vs. Optical Coherence Tomography

Slit lamp indirect ophthalmoscopy is another facet of a complete dilated fundus examination. With the use of high-plus power lenses, such as 78D or 90D lenses, indirect ophthalmoscopy at the slit lamp produces inverted images of the retina with a higher resolution and a wider field of view.[4] In fact, the lower the dioptric power of the lens, the higher the axial resolution and the better the stereopsis.[4] This, in turn, allows for easier detection of various posterior pole pathologies including cystoid macular edema, subretinal fluid secondary to choroidal neovascularization, diabetic changes and more. In addition, careful examination of the slit lamp beam as it hits the retina can differentiate between various contours of a retinal lesion.

Advantages of this diagnostic skill include a comfortable working distance between the patient and the practitioner, various magnifications that can be obtained and creating an image independent of the refractive power of the eye.[4] Finally, slit lamp indirect ophthalmoscopy can also allow for a sufficient view through small pupils or pupils that do not dilate well.

However, limitations to this procedure can include patient discomfort at the slit lamp, hazy views due to media obstruction, lack of peripheral retinal views and most importantly, difficulty appreciating the depth of a retinal lesion.[4] This, in turn, may limit the description of the retinal lesion, including its thickness, level of extension and point of origination.

In contrast, optical coherence tomography (OCT) is a non-invasive imaging technique. It uses low-coherence light waves to produce high-resolution, cross-sectional images of the retina, retinal nerve fiber layer and optic nerve head.[5,6] Enhanced-depth imaging (EDI) is a feature available on most OCTs that allows for better visualization of structures underneath the retinal pigment epithelium (RPE) such as the choroid.[6,7] OCT is increasingly being used to analyze the retinal morphology and quantify changes in various disease states.

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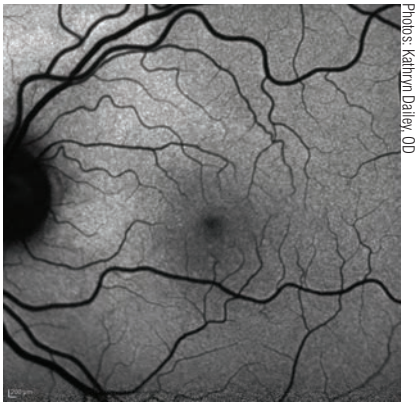
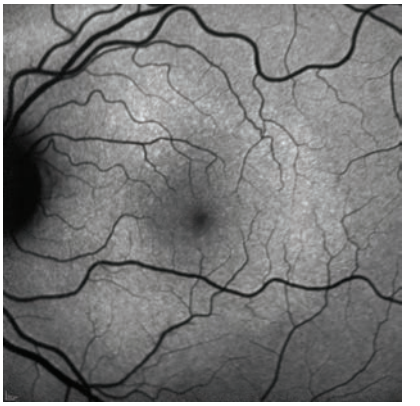
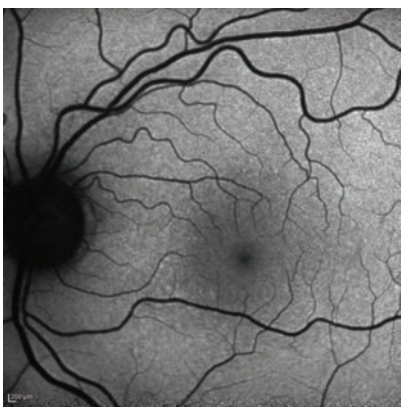


Photos: Kathryn Dailey, OD





These FAF photos show a patient with with the changes in the photoreceptor integrity line secondary to neurosyphilis. The top left shows the patient at initial presentation; top right shows them at first follow-up; at left is their second follow-up. Note the extension of the hyperfluorescence inferior to the optic nerve and macula.

These include, but are not limited to, macular degeneration, diabetic retinopathy and central serous retinopathy.[7] The impetus for its introduction into medical practice is to document and monitor subtle retinal changes over time in an efficient manner, thereby enhancing the level of care provided.

OCT is routinely used as it is time-efficient in clinical practice, which serves to benefit doctors and patients alike. Each volumetric scan takes no more than six seconds, far less than the time it would take to complete other imaging modalities such as fluorescein angiography.[7] It provides many cross-sectional images, which not only allows ODs to monitor subtle retinal changes but also serves as a visual aid to the patient.

Despite these benefits, patient motion and blinking might introduce artifacts into the imaging results and ultimately skew the quality of the image produced.[7] In addition, the image quality may be limited by media opacities like cataracts or blood in the vitreous cavity.[6,7] OCTs also tend to be costly and have limited field of view and limited lateral resolution, making it difficult to scan peripheral retinal lesions.[7] While OCT may not be able to fully substitute a traditional funduscopic examination, it is a valuable tool in diagnosing, documenting and following a variety of ocular pathologies.

Fundus Exam vs. Fundus Autofluorescence

New retinal imaging techniques can provide an in-depth analysis and a unique perspective of the retinal tissue and RPE. Specifically, fundus autofluorescence (FAF) was developed to provide functional information about the retina by imaging and assessing the underlying RPE.[8] It is a non-invasive,

time-efficient imaging technique that detects fluorophores, which are naturally occurring molecules that absorb and emit light of specified wavelengths.[8,9] FAF employs blue-light excitation and then collects emissions within a certain spectra (typically between 500µm and 700µm) to form a brightness map reflecting the distribution of lipofuscin, a dominant fluorophore within the RPE.[8] For this reason, FAF has become a useful tool to use when diagnosing and monitoring several diseases, including macular degeneration, central serous chorioretinopathy, vitelliform lesions, macular telangiectasia, medication toxicity and more.

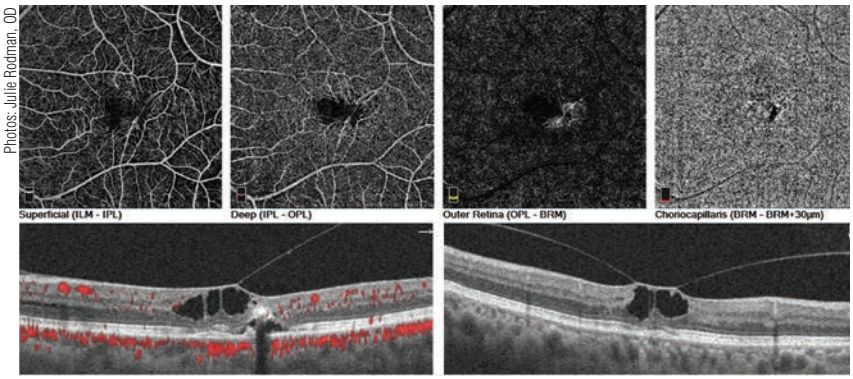
To further understand its application, let's look at how FAF highlights the differences between wet age-related macular degeneration (AMD) and vitelliform deposition. Choroidal neovascularization in wet AMD patients often presents with corresponding hypo-autofluorescence, which may be surrounded by a thin layer of hyper-autofluorescence reflecting a hyperplastic proliferation of RPE cells surrounding the lesion.[9] Hemorrhages and exudation are initially hypo-autofluorescent due to light absorption but may eventually become hyper-autofluorescent.[9] Vitelliform deposition, on the other hand, reflects an accumulation of lipofuscin underlying the macula, which creates a distinct area of hyper-autofluorescence.[9,10] Subsequent RPE atrophy following vitelliform deposition corresponds to areas of hypo-autoflourescence.[10]

One of the most prominent advantages of this imaging modality is that it may allow for the identification of retinal diseases when they are not otherwise evident during dilated examinations. This is particularly helpful when managing patients with unknown vision loss or a positive family history of hereditary retinal diseases. For instance, early AMD encompasses alterations at the level of the RPE, which can become readily apparent in FAF imaging while appearing normal on funduscopy.[9]

FAF is also useful in tracking pathophysiological mechanisms. For instance, FAF can highlight areas of arterial occlusion as they inhibit proper autofluorescence of the RPE due to the increased thickness.[10] Knowing this can allow for an objective analysis of a patient's response to therapy and help monitor the natural reduction in the severity of the disease.

In addition, FAF allows for a better measurement of the progression or regression of retinal disorders compared with earlier standards of measurement. In fact, for a more precise measurement, autofluorescence can be averaged between different retinal zones or even acquired at specific desired points.[10] Additionally, FAF is valuable in predicting the progression of geographic atrophy in AMD patients.[9] Research shows an increased autofluorescence surrounding an area of geographic atrophy precedes its extension, a feature that cannot be appreciated during a traditional dilated examination.[9]

FAF, however, does have its limitations. At present, only a limited number of reference databases exist to consistently classify normal and pathological FAF phenotypes.[8,9] The inter-individual and intra-individual variability of media opacities, refractive error, genetic expression and cellular lipofuscin content make the possibility to develop this database challenging.[8] Differences in image acquisition not only limit producing objective autofluorescent measurements but also make the ability to compare



These images showcase OCT angiography's ability to image macular telangiectasia.

images from different patients and operators difficult.[8] Nevertheless, fundus autofluorescence is quickly being integrated into clinical practice as a complementary tool in diagnosing, following and treating retinal disorders.

OCT Angiography vs. ICG Angiography

Indocyanine green (ICG) angiography is a diagnostic procedure that employs a water-soluble dye to examine the blood flow of the choroid.[11] Similar to fluorescein angiography, ICG dye is injected into the vein of the arm and photographs are taken as the dye passes through the choroidal vasculature.[11] Infrared light, given off by the ICG dye, has a longer operating wavelength compared with the light emitted from fluorescein dye, thereby allowing it to be better imaged through pigment, fluid, lipids and hemorrhages.[11,13] This increases its ability to detect abnormalities at the level of the choroid that may otherwise be blocked by an overlying hemorrhage or hyperplastic RPE.

Additionally, ICG dye does not readily leak out of choroidal vessels, whereas fluorescein dye does.[13] This gives it an advantage over intravenous fluorescein angiography (IVFA) for many choroidal pathologies. ICG angiography can also highlight abnormal aneurysmal outpouchings of the inner choroidal vascular network seen in idiopathic polypoidal choroidal vasculopathy.[12]

Indications for the use of ICG angiography include, but are not limited to: choroidal neovascularization, pigment epithelial detachment, polypoidal choroidal vasculopathy, central serous retinopathy, intraocular tumors and occult retinal disease.[12] As an ancillary test, this dye is highly protein bound, which equates to less leakage through the choriocapillaris, reducing allergy concerns.[12,13] In addition, dynamic ICG angiography uses confocal scanning laser ophthalmoscopy (SLO) to detect smaller feeder vessels, which can be valuable in establishing the role of ICG-guided therapy in medical practice.[14]

ICG angiography, however, does have disadvantages as well. In addition to the allergy concerns, it is invasive, expensive, time-consuming and not always readily available.[12] It also has little to no benefit in other retinal vascular disorders such as diabetic retinopathy and retinal vein occlusion.[13]

Nevertheless, clinical applications of ICG angiography continue to expand as it allows for the analysis of choroidal blood flow in both normal and diseased eyes.[14] Ultimately, this not only allows physicians to enhance current treatment strategies but also establish new management protocol for various disease states.

The retinal and choroidal vasculature can be a breeding ground for many ocular pathologies and dye-based angiography has been the gold standard diagnostic test for assessing these disorders. However, they have their limitations. OCT angiography (OCT-A), in contrast, is a novel technique that may be able to overcome some of these limitations. OCT-A is a non-invasive imaging technique that allows for the detection and three-dimensional reconstruction of the retinal capillary network.[16] OCT-A images are produced by comparing, on a pixel-to-pixel basis, repeated B-scans acquired at a particular retinal location in rapid succession.[16]

This imaging modality has the ability to analyze each vascular capillary network individually, from the superficial to the deep plexus, which becomes crucial when studying various retinal vascular disorders such as macular telangiectasia, retinal artery and vein occlusions.[15]

Additionally, OCT-A has a superior ability to precisely delineate vessels surrounding the foveal avascular zone.[15,16] Being able to visualize this has provided insight into the mechanisms of various disorders. For instance, a recent study demonstrated that the perifoveal intercapillary area on OCT-A increases in size as the level of diabetic retinopathy progresses.[15,17]

Another important application of OCT-A is for patients with macular telangiectasia, as it is able to highlight the abnormal vessel growth in both the superficial and deep plexus, which may otherwise be unnoticed on fundus examination.[15]

Possibly the most important and common application is identifying different choroidal neovascularization morphologies and its response to anti-VEGF therapy. Overall, its sensitivity in cross-sectional and *en face* imaging make it a crucial tool in monitoring various ocular pathologies.

In its current configuration, however, there are a few limitations of OCT angiography. One drawback is that it has a restricted field of view.[15] The scan protocols currently available are 2x2mm, 3x3mm, 6x6mm and 8x8mm, rendering it ineffective for peripheral retinal disorders.[15,17] OCT-A is also prone to acquiring various artifacts. It works by detecting erythrocyte motion, so any extraneous motion by the patient during image acquisition can result in motion artifacts.[16] In addition, low amounts of blood flow below the device threshold may go undetected.[16] Media opacities can lead to signal attenuation and shadowing, thereby producing poor quality images.[16,17] Superficial blood vessels may obscure abnormalities in the deeper retinal network, possibly leading to incorrect diagnoses if not promptly identified.[15,16] Aside from being extremely motion sensitive, OCT-A imaging is expensive to perform.

While it is a non-invasive technique that can provide insight into the diagnosis of a retinal condition, its role in therapeutic monitoring remains unclear. Nevertheless, OCT-A is promising as it allows for the simultaneous assessment of both structural integrity and vascular flow. It is also safer, faster and more comfortable for the patient compared with dye-based approaches, making it invaluable in both clinical practice and research studies.

A Delicate Balance

Dilation continues to be the standard of care in comprehensive optometric examinations. However, with so many new technologies on the market, optometrists can enhance their level of care and grow the profession itself. Keeping the traditional in balance with the cutting edge is key to optimizing patient care. Overall, while each piece of equipment gains traction and popularity, it is an optometrist's duty to know how and when to employ them. ■

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42nd Annual Technology Report

Living With Blue Light Exposure

The sun is your biggest enemy, and digital devices aren't as bad as you think. Here's the current research and recommendations.

By Mark Rosenfield, MCOptom, PhD

The impact of blue light on the eye has gained increased interest in recent years due to the explosion of devices and lighting sources emitting wavelengths between 400nm and 500nm.[1,2] General lighting, desktop computers, laptops, tablets, electronic reading devices and smartphones all expose the eye to blue light. Nevertheless, clinicians must remember that the amount of light emanating from artificial sources is a fraction of the radiation emitted from the sun—a typical LED used for general lighting emits around 50 to 70 lux, while sunlight provides approximately 100,000 lux.[3]

The Real Risk

A number of ocular conditions are associated with blue light exposure (*Table 1*). However, due to the proximity within the electromagnetic spectrum of blue and ultra-violet (UV) radiation, it is unclear precisely which are the damaging wavelengths. Given that UV exposure is associated with eyelid malignancies, such as basal cell and squamous cell carcinomas, photokeratitis, pterygia and cortical cataracts, researchers speculate this may be the damaging radiation rather than visible blue light.[4] While the cornea, aqueous and vitreous are largely transparent to wavelengths between 300nm and 400nm, the natural crystalline lens absorbs much of the ultraviolet A (UVA) range (320nm to 400nm), thereby shielding the retina from its potentially toxic effects.[5]

Table 1. Ocular Conditions Associated With Blue Light Exposure

- Age-related macular degeneration
- Basal cell and squamous cell carcinomas
- Photokeratitis
- Pterygium
- Cortical cataract
- Damage to the retinal pigment epithelium
- Eye strain during and after sustained near-work

No clear answer exists as to what constitutes excessive exposure. Single, high amounts are damaging but so may be long-term, low-level exposure to the eyes and skin. For example, UVA radiation damages keratinocytes in the basal layer of the epidermis, which is the site of most skin cancers. Ultraviolet B (UVB, 290nm to 320nm) exposure can lead to sunburn, photokeratitis, cataracts and retinal lesions. This wavelength tends to damage the skin's superficial epidermal layers and plays a key role in the development of skin cancer, as well as a contributory role in tanning and photo-aging.

For radiation to damage the posterior segment of the eye, it must be transmitted through the ocular media. While most blue light does reach the retina of a young, healthy eye, the natural yellowing of the crystalline lens with increasing age creates a blue-blocking filter, thereby obstructing passage of these wavelengths. But even the clear crystalline lens absorbs some wavelengths between

400nm and 420nm.[6] Additionally, retinal illuminance is reduced further by the pupil, given that pupillary constriction is greater when the eye is exposed to blue light compared with an equal amount of green light.[7]

The photoreceptors within the macula are directly exposed to light, as they have no other cell layers covering them. Within these photoreceptors, the antioxidative pigments lutein and zeaxanthin normally filter out blue light due to their yellow color. The xanthophylls have a protective role against retinal oxidation through the absorption of damaging blue light, neutralization of photosensitizers and reactive oxygen species and scavenging of free radicals.[8,9] We obtain these antioxidants through our diets, and they are included in the AREDS-2 formulation designed for the prevention of age-related macular degeneration.[10]

Fig. 1. This is how a computer screen appears when viewed through either a filter that blocked 99% of blue light (right) or an equiluminant 0.3log unit neutral density filter (left). This image, which shows the bright yellow appearance of the blue-blocking filter, is for illustrative purposes only. In the study, only one of the filters was present for each experimental trial.[26]

Although a number of animal studies show direct evidence of retinal damage following blue light exposure, almost all of them used radiation levels far in excess of natural conditions.[11,12] Blue light damage has been observed following both *in vitro* and *in vivo* studies.[13-16] One investigation observed a significant loss of photoreceptors in the superior retina of albino rats following 24 hours of exposure to LED light sources at 6,000 lux through a dilated pupil.[11] In contrast, cyclic exposure (12 hours on/12 hours off) to LED light sources at 500 lux without pupil dilation for one month did not produce any significant retinal cell loss in pigmented (non-albino) rats.[11] Additionally, significant retinal damage was observed when albino mice were continuously exposed to white light of high intensity (5,000 lux) for seven days.[12] Given the extremely high levels of radiation necessary to produce retinal damage, naturalistic exposure levels are unlikely to be large enough to cause significant tissue impairment.

Digital Eye Strain

We currently live in a society where electronic devices are deeply embedded into daily life. Ninety percent of families in the United States own at least one computer, smartphone or tablet, while the typical American family has five or more of these devices.[17] Furthermore, between 40% and 60% of individuals experience visual or ocular symptoms while viewing electronic displays for prolonged periods of time.[18,19] These symptoms—including eye fatigue, ocular irritation, burning, eye strain, redness, dryness, blurred and double vision—are collectively termed digital eye strain (DES).[20] Although the symptoms are typically transient and disappear soon after device use ceases, some individuals experience ocular discomfort for a sustained period after prolonged viewing of an electronic screen.

Many have speculated that the high levels of blue light emitted from digital displays may be responsible for the development of DES symptoms. For example, blue light contributes more than one-third of the emission spectrum of an Apple iPhone 7.[21] However, the evidence to support this association is minimal. Nevertheless, many ophthalmic lens manufacturers market blue-blocking filters as a treatment paradigm for DES. One study examined the effect of low-, medium- and high-density blue filters (in the form of wraparound goggles) worn during computer work in dry eye and normal subjects.[22] The researchers observed a significant reduction in symptoms in the dry eye group, but not in the non-dry eye subjects, for all of the filter densities tested. However, the study did not include a control condition, so a placebo effect cannot be ruled out. Further, the wraparound goggles may have reduced tear evaporation in the dry eye subjects, thus increasing ocular comfort.

Subsequently, other investigators evaluated the effect of blue-blocking lenses on symptoms of DES and the critical fusion frequency (a parameter previously associated with eye fatigue) following a two-hour computer task.[23-25] The study authors determined that the high-blocking filter, which blocked around 60% of the blue light, produced a significantly greater post-task change in critical fusion frequency compared with a low-blocking blue filter (which blocked approximately 24%) or control lenses that blocked approximately 3.2% of blue light. Based on the critical fusion frequency findings, the authors reported that subjects wearing high-blocking

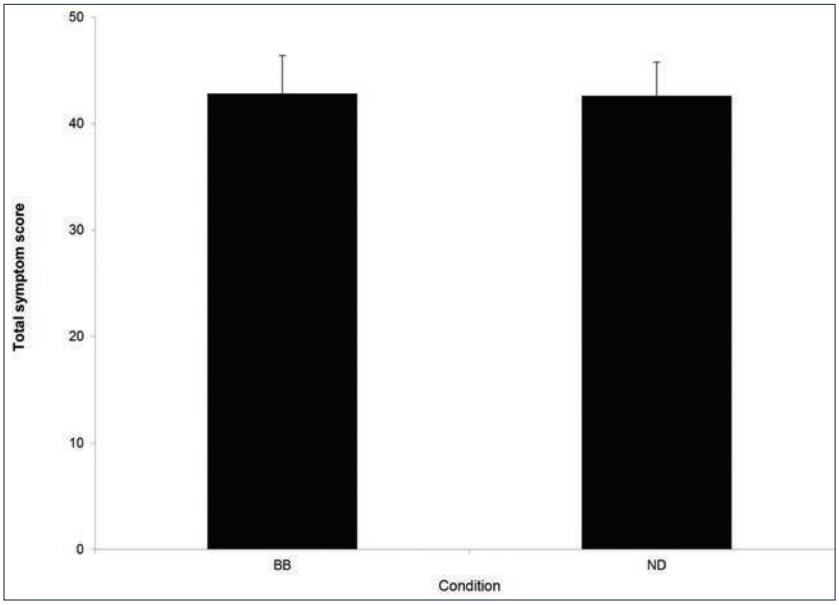

Fig. 2. Mean total symptom scores for the blue-blocking (BB) and neutral density (ND), i.e., control conditions immediately following a 30-minute reading task from a tablet. Error bars indicate one standard error of the mean. No significant difference in symptoms was observed for these two conditions.[26]

filters had less fatigue after the two-hour task than before they started the trial. As for subjective symptoms, the high-blocking filters produced a significant reduction in pain, heaviness and itchy eyes but not in other previously noted DES symptoms, such as eye fatigue.[20] However, the various filter conditions were performed on different groups, so the reduced symptoms observed in the high-blocking filter group may have been a consequence of those particular individuals, rather than the effect of the filters.

Two studies from our laboratory do not support the proposal that DES symptoms are associated with exposure to visible blue light. In the first investigation, we compared symptoms after sustained reading from a tablet computer.[26] The screen was covered either with a filter that blocked more than 99% of blue light or an equiluminant, neutral-density filter (*Figure 1*). We observed no significant difference in post-task symptoms between the two conditions (*Figure 2*). The study does have some limitations, as it was not performed on a double-blind basis, and most commercially available filters only block between 10% and 20% of blue light, rather than the 99% level tested here.[27]

In a subsequent investigation, we compared three commercially available lenses having an identical, clear appearance using a double-blind protocol. Twenty-four subjects performed a 20-minute reading task using a tablet computer while wearing lenses containing either a blue-blocking filter (TheraBlue 1.67 or TheraBlue polycarbonate) or a CR-39 control lens.[28] While we observed a significant increase in symptoms immediately following the near vision task, no significant difference in symptoms was found between the three lens conditions (*Figure 3*).

Accordingly, there is little evidence at this time to support the use of blue-blocking filters as a clinical treatment for DES. Management of other ocular factors, as well as the creation of an optimal environment for screen viewing, is more likely to provide greater success in minimizing symptoms. For instance, most smartphones now include a night setting that reduces the magnitude of short wavelengths emitted from the screen. While this shift is unlikely to reduce DES symptoms, it may attenuate any difficulty in falling asleep after sustained viewing of digital screens.

Circadian Rhythm

Blue light exposure can affect the physiological circadian rhythm. The natural sleep-wake cycle is controlled by the release of the hormone melatonin from the pineal gland.[29] Typically, melatonin secretion increases soon after the onset of darkness, peaks in the middle of the night (between 2am and 4am)

Blue-blocking Intraocular Lenses

If the natural lens is removed surgically (e.g., cataract extraction), the question arises whether it should be replaced with a clear or yellow (i.e., blue-blocking) intraocular lens (IOL). A Cochrane systematic review on the effect of blue-filtering IOLs noted that a yellow IOL does not produce any significant reduction in best-corrected visual acuity or contrast sensitivity. However, the same review also reported no significant difference in the proportion of eyes that went on to develop late-stage age-related macular degeneration after three years of follow-up, or any stage of AMD after one year of follow-up. The authors concluded that the use of blue-blocking IOLs to alter the risk of developing AMD is "speculative."

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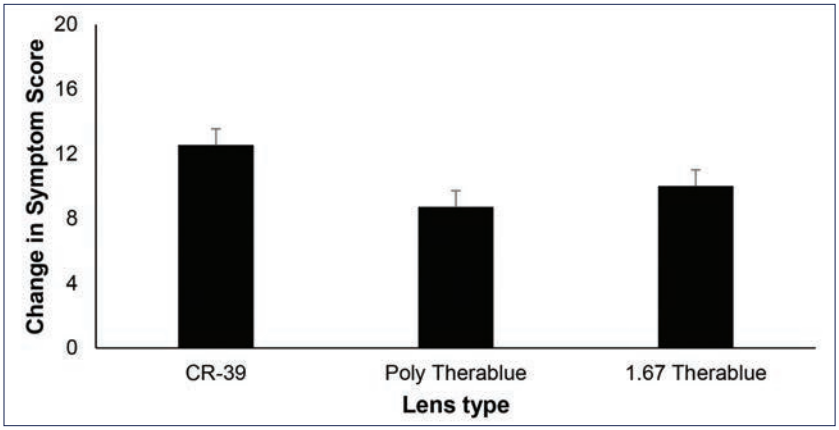

Fig. 3. Mean post-task change in symptom score following a 20-minute reading task from a tablet for three lens conditions. CR-39 = clear lens with no blue-blocking filter. Both the polycarbonate (poly) and 1.67 Therablue lenses included a clear, commercially available blue-blocking filter. Error bars indicate one standard error of the mean. No significant difference in symptoms was observed for these two conditions.[28]

and gradually falls during the second half of the night. Exposure to any visible light, but especially blue light, suppresses the secretion of melatonin. When comparing the effects of 6.5 hours of blue light exposure to green light of comparable brightness, the blue light suppressed melatonin for about twice as long and doubled the shift in circadian rhythms (three hours vs. 1.5 hours).[30]

Exposure to blue light sources in the evening will affect one's ability to fall asleep. Subjects reading from an electronic reader take longer to fall asleep and have reduced evening sleepiness, melatonin secretion and morning alertness and later timing of their circadian clock when compared with subjects who read a printed book.[31] Similarly, the use of short wavelength-blocking glasses at night increases subjectively measured sleep quality and duration.[32,33] Therefore, clinicians should recommend patients avoid using electronic digital devices for two to three hours before bedtime.

However, blue light exposure isn't always a bad thing. Evidence shows that the use of blue-enriched, white fluorescent lighting (17,000K) in an office setting improves alertness, positive mood, concentration, ability to think clearly and evening fatigue when compared with white fluorescent lighting (4,000K).[34]

Table 2. Range of Digital Device Blue Light Exposure[35]

Type of Device	% of ICNIRP limit
Desktop	0.71 - 1.26
Laptop	0.63 - 1.97
Tablet	0.43 - 2.38
Smartphone	1.78 - 4.09

How Much is Too Much?

In evaluating safe levels of blue light exposure, the International Commission on Non-Ionizing Radiation Protection provided guideline levels below which adverse health effects were considered unlikely. Their recommendations state that detailed assessments of white light sources are not required for luminance values below 104cd/m^2.[35]

By applying this criterion to everyday conditions, we see that staring at the sky in the United Kingdom on a clear day in June or a cloudy day in December represents about 10.4% and 3.4%, respectively, of this standard. The emission of blue light from digital displays barely reaches 4% of this limit (*Table 2*). Thus, clinicians may conclude that the magnitude of exposure from digital devices does not approach dangerous levels.[35] Interestingly, a new policy on outdoor light pollution, recently issued by the government of France, specifically restricts the emission of blue light. The decree requires that, in all instances, the correlated color temperature of light should not exceed 3,000K (equivalent to that of a tungsten halogen light bulb).[36]

Real-world Recommendations

By far the most significant source of low-wavelength radiation comes from sunlight, and excessive sun exposure is a well known risk factor for age-related macular degeneration, carcinoma, photokeratitis, pterygia, cataract and retinal pigment epithelium damage.[37] Patients with high exposure to sunlight need to be counseled on the use of visors and brimmed headwear, UV-blocking lenses with a wrap-around design, small vertex distances and lenses that cover a large area.

While lenses may help to protect the eye against exposure to dangerous wavelengths, the skin (including the eyelids) may still be exposed, highlighting the importance of sun-protective clothing. Additionally, blue light's effect on the body's circadian rhythm can interfere with sleep patterns, and exposure should be minimized two to three hours before bedtime. However, minimal evidence supports the use of blue light-blocking filters as a treatment for DES, and they are not necessary for the majority of individuals. ■

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Retina Care "Plus"

How Primary Eye Care Can Elevate Patients' Retinal Health

Kerry Gelb, OD
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Retinal health and function has always been an essential part of primary eye care. Optometrists examine the entire eye from the front to the back, and this, of course, includes the retina, its structure, and its function. Retinal examination is a fundamental part of the optometric evaluation.

Thanks to improvements in retinal technology, eye care professionals are now able to move beyond routine assessments of the retina and advance to what I call a 'retina care plus' model; that is, retina care *plus prevention*, in order to broaden the scope of their optometric care. This novel approach enables eye care professionals to address their patients' retinal health and function, with the aim of catching any potential damage early and preventing progression to disease.

Optometrists, because of the amazing technology available to them, can detect potential disease long before the primary care physician, and recommend strategies to reduce mortality and morbidity, and lower healthcare costs. This capability has become even more profound with our aging population.

Contemporary imaging modalities enable clinicians to, for example, more easily observe subtle macular pigment changes, early drusen, microaneurysms in the retinal capillaries, and tiny retinal hemorrhages. Such findings offer optometrists a more complete picture of the eye's health and the opportunity to manage patients more impactfully through functional, preventive medicine approaches that address root causes.

Proactive and preventive care is not a new concept for optometrists in other areas of practice. Adapting this approach in the retinal realm can steer patients toward better retinal and overall health, and strengthen the doctor-patient relationship. Eye care professionals who go beyond their usual responsibilities to provide guidance on improving overall nutrition and exercise habits, reducing stress levels, smoking cessation, and proper macular supplementation can lead to life-changing improvements for patients, reinforcing their loyalty to the practice.

The Right Technology to Intervene

To move to the next level in retina care, it's key that optometrists have the right imaging technology in their practices. State-of-the-art retinal cameras and optical coherence tomography technology (OCT) systems are essential tools to assess retinal structures, and help monitor disease progression and therapeutic responses.

Retinal cameras offer the clinician a better view of blood vessel changes that might indicate diabetes or macular degeneration, while OCT provides structural details of the retina, resulting in quantitative and qualitative information that is useful in tracking diabetic macular eye disease. The OCT is sensitive down to approximately 8

Link Between Retinal Microaneurysms & Diabetes

The association between findings of retinal microaneurysms and future diabetes is strongly correlated. One study I co-authored and published in *Diabesity* looked at how insulin resistance negatively affected the retinal vessel health of individuals at risk of diabetes.[1]

Using highly sensitive 580nm multispectral retinal imaging, our group found that subclinical retinal microaneurysms correlated with insulin and pancreatic function, and IR measures more closely than fasting glucose tests. We concluded that diabetes research should focus on retinal microaneurysms and IR as actionable pre-diabetes and pre-retinopathy risk factors. *—Dr. Gelb*

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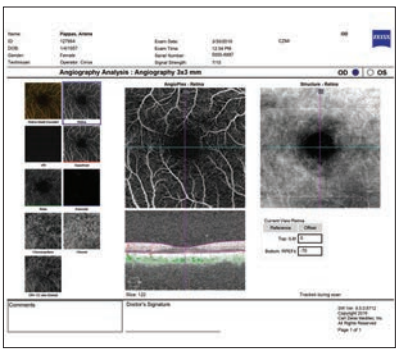


OCTA Images. Microaneurysms in a diabetic patient. Images: Kerry Gelb, OD

microns, allowing the optometric physician to zero in on the smallest retinal blood vessels, providing excellent ability for very early detection of hidden problems.

More recently, advanced OCT angiography (OCTA) has become available to produce depth-resolved images of abnormal new blood vessels at the macula without the need for contrast agents, while ultra-widefield imaging is able to capture up to 200 degrees of retinal view in a single shot.

Catching Disease Early

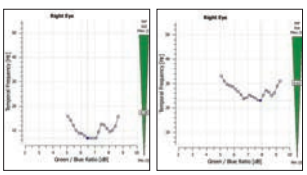
New technology can illuminate subtle harbingers of disease sooner than direct ophthalmoscopy. For example, it can help optometrists detect early drusen (signs of age-related macular degeneration) or retinal pigment epithelium stacking (signs of geographic atrophy) that are not appreciable by more traditional means. In addition, OCTA and advanced retinal cameras can reveal microaneurysms and intraretinal hemorrhages that aid clinicians in identifying individuals at risk for diabetic retinopathy or diabetes. This capability is paramount, as research has borne out a strong connection between the presence of microaneurysms and future diabetic disease.[1]

In my own clinical experience, I have had a number of patients present with hemorrhages imperceptible on direct ophthalmoscopy that led to eventual diagnoses of diabetes. So having access to advanced imaging technology that can pinpoint early signs is invaluable for me as a doctor and for my patients.

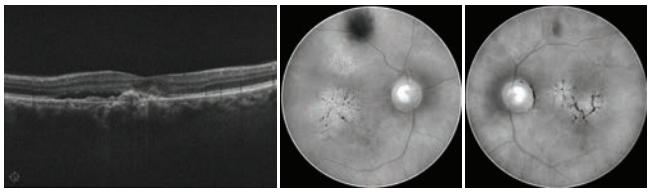
Using Nutraceuticals to Improve Retinal Health & Vision

In addition to leveraging advanced imaging, I also test patients' macular pigment density to evaluate retinal health. If I see evidence of abnormal macular pigment loss, I recommend macular supplementation—specifically MacuHealth with LMZ (which I also use daily).

The macula relies on three diet-derived pigments—lutein, zeaxanthin, and meso-zeaxanthin (MZ)—to function optimally. Numerous studies have shown that people with low macular pigment are at greater risk for macular degeneration, and that boosting pigment levels can slow damage from natural aging and oxidative stress that frequently leads to disease.[2-6] Another reason why macular supplements are so important is that most of us are not getting adequate nutrients from our diets, partially because the nutrient content of fruits and vegetables has declined over time.[7,8]



Macular Pigment Improvement. Macular pigment optical density improvement seen after supplementing with MacuHealth with LMZ, using the QuantifEye MPOD Measurement Instrument.



OCT and Fundus Imaging. Macular degeneration is evident.

Since I've been recommending macular supplements over the last 15 years, I have observed transformative improvements in my patients' retinal health and functional status. I only recall one patient progressing to wet macular degeneration among the hundreds who have supplemented and made positive dietary changes.

Macular supplementation is also exceptionally beneficial for healthy patients. Since the macula makes up the central 4% of the retina, and mediates central and color vision, optometrists can actually enhance visual function in healthy patients through proper nutritional supplementation.[2] Studies show that increasing the density of the macular pigment layer can improve contrast sensitivity, decrease glare, and aid in photostress recovery.[9,10]

Rationale for Adding the Next Level of Retina Care

The reason I added preventive retinal care to my practice is because I wanted to help my patients *before* they were diagnosed with disease. Since I have a way to support patients' eye health and vision for a lifetime—through proper macular supplementation—I want to start taking action immediately. The decision to expand into preventive retina has paid off exponentially for my patients' eye and vision health, as well as for my practice in the form of ongoing referrals from extremely happy patients. In fact, my happiest, most enthusiastic patients are the one I speak to about lifestyle medicine.

It's true that optometrists are busy seeing patients and running practices on a day-to-day basis, and the idea of adding another clinical responsibility might not be top of mind. Also, some optometrists could be uncomfortable about the idea of selling or promoting nutritional supplements. However, if eye care providers take the time to educate themselves on the enormous benefits of macular nutrition to patients, the decision to recommend supplementation is an easy one.

The best part about expanding retina in the eye care practice is that it's a win-win—for patients' health and the practice's growth. Many ocular and systemic diseases that manifest in the body show early signs in the retina, so the opportunity for optometrists to extend care beyond their existing scopes of practice is considerable. But even more importantly, I firmly believe that to be a great, comprehensive optometrist, you have to be great at retina.

Clinical Strategies to Address the Whole Patient

Along with recommending macular supplementation to patients who exhibit low macular pigment levels or early signs of AMD, I prescribe omega-3 supplementation and refer for vitamin D blood tests. If vitamin D levels are low, I encourage patients to get out in the sun more often or take a vitamin D3 and K2 supplement to improve cardiometabolic function and overall health.

Often, I will assess DNA methylation, a process that can modify gene function, and test for MTHFR gene mutations—which can affect the body's ability to use folic acid or folate, and increase disease risk. If patients test positive for mutations, I'll prescribe a B-complex supplement.

I also spend time discussing lifestyle improvements such as diet and exercise strategies with patients. *—Dr. Gelb*

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42nd Annual Technology Report

Today's Tech for Assisting the Visually Impaired

Determining which devices serve a patient's goals makes intervention more feasible.

By Parres M. Wright, OD

Visual impairment is included among the top 10 most prevalent causes of disability in the United States.[1] Low vision affects more than two million Americans and ranks only behind arthritis and heart disease as the reason for impaired daily functioning in Americans older than 70.[1] In addition, blindness ranks third as one of peoples most feared disorders.[1]

All eye care providers can overcome the challenges of visually impaired patients seeking assistance, through simple low vision intervention techniques and strategies.

The numbers of low vision patients is expected to increase, according to the National Eye Institute, and the prevalence of low vision is expected to almost triple by the year 2050.[2] Practitioners from every state and mode of practice will likely encounter patients who are visually impaired and seeking quality care. This is especially true because the majority of patients with vision impairment have moderate vision loss with median Snellen values ranging from 20/71 to 20/80.[3]

Most often practitioners will encounter patients who seek care because they are having difficulty with specific tasks, most commonly reading.[4] Having difficulty reading is a major consequence of vision loss for Americans with low vision.[5] In addition to reading, driving and household chores, self-grooming and meal preparation are common complaints in my practice. The loss of these abilities burdens the patients and their loved ones. Current technological advances in assistive devices and the smartphone allows practitioners to make timely in-office suggestions and implement strategies for immediate use, thereby increasing the quality of life of their clients.

Many practitioners may be tempted to refer these patients immediately to a low vision provider without attempting to make any interventions themselves; however, all optometrists are capable of performing a basic low vision evaluation and providing the patient with vision impairment some initial tools to get started on their journey of improved functioning.

Subsequently, the patient learn as much as they can and then be referred for more extensive low vision exams and training as needed or if vision worsens.

Current Advances in Tech

The equipment and technology that is available in low vision rehabilitation is vast and ever-changing, provid-

ing optometrists myriad options when providing care to visually impaired patients. The newest technology includes advanced autofocused telescopic devices, portable electronic magnifiers, wearable devices, character readers, apps and built-in smartphones and tablet accessibility options. These options make it easier for eye care providers to assist their patients in living fuller, more productive and more independent lives.

Telescopic Devices

These options range in complexity from low tech to high tech. Telescopic options may be handheld or mounted, monocular or binocular and full diameter or bioptic. A single handheld monocular telescope is oftentimes a good device for distance spotting and enhancing safety during mobility, thus making mobility easier. A focusable monocular telescope in moderate power from 2x to 6x may be used for spotting streets signs and signals and for viewing distance objects or events for short periods of time.

Monocular telescopes are made by a variety of manufacturers and are readily available. They are a cost-effective alternative to more high-tech options in the "tech adverse" subset of patients. When considering prescribing a telescope for patients, practitioners must first determine what magnification a patient will require to perform tasks. Generally, the minimal target acuity is 20/40, for most distance tasks due to this is the minimum distance acuity that is required for unrestricted driving in most states.

A quick way to determine the magnification required is with a simple formula which does not require a calculator: **Magnification = Best Corrected Visual Acuity ÷ Target Acuity**

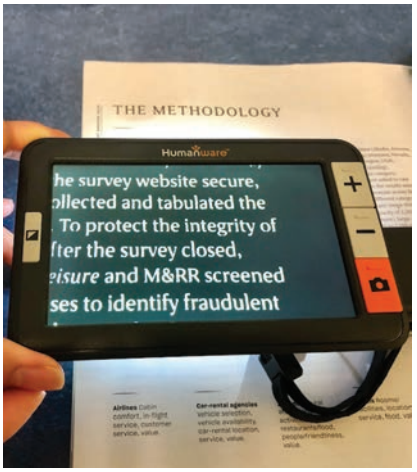
For example, if a patient has an entering best-corrected distance acuity of 20/100 and the target acuity is 20/40, the magnification is calculated as follows: 100/40=2.5x. Thus a 2.5x telescope will provide the ability for the patient to see the goal acuity of 20/40 in this example. When choosing a telescope, it is also helpful to understand how telescopes are labeled. Generally, traditional monocular telescopes will have several numbers on them such as 4x12. The first number represents the magnification, in this example 4x. The second number indicates the diameter of the objective lens in this example 12mm. The objective lens is the lens closest to the target which effects the brightness of the image ultimately.

A binocular option for distance viewing that many low vision clinicians recommend is the Eschenbach MaxTV. This is a great binocular telescopic option for multiple reasons. It is practical and easy to maneuver with a minimal learning curve for most patients and patients often get immediate gratification after working with this device. It is made for sedentary, distance viewing tasks such as watching television. Practitioners must warn patients not to walk around in these types of devices due to the risk of falling due to increased magnification. It provides approximately two times magnification and allows each eye to be focused individually, this allows for focus on objects from 10 feet and farther.[6] It is good device to consider not only for TV but also for patients who want to watch sporting events, go to movies or who have taken up hobbies such as bird watching.

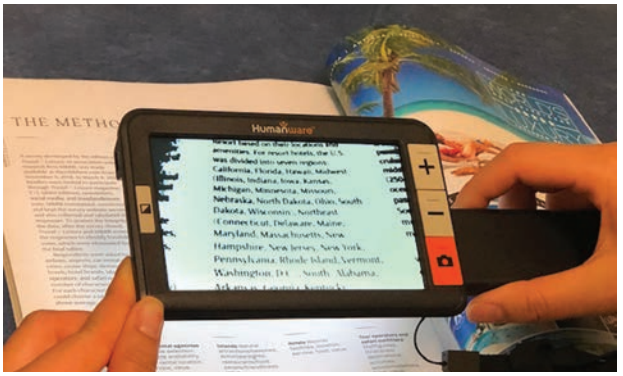
Binocular telescopic devices can be helpful for sedentary, distance-viewing activities.

Patients often complain of their inability to enjoy outings with family to sporting events or being able to watch their grandchildren in extracurricular activities. They appreciate this device because it allows them to return to enjoying these activities.

Another telescopic device that practitioners will find helpful and easy to incorporate into practice is the VES-Falcon Autofocus Bioptic Telescope. The VES-Falcon is available in several power options and covers a wide range of prescriptions.[5] It provides the user with hands-free, immediate clear vision at almost any distance and covers refractive error ranging from +8.00D to -8.00D.[5] This wide array will encompass most patients who present for a low vision exam making this an easy to fit and train device for most eye care practitioners. It is lightweight and comes with a rechargeable battery that lasts up to eight hours on a single charge, providing patients



Electronic magnifiers such as these allow the visually impaired to have a portable, customizable device to assist in their visual tasks.



who use it to drive with the peace of mind that it will be ready to use, especially at the end of a work day. This is an option for patients a who have difficulty manipulating dials and buttons due to arthritis or neuropathy.

Portable Electronic Magnifiers

When convenience and portability are a priority, a tablet or an electronic video magnifier is a good place to start. Small portable electronic magnifiers are a great way for technologically savvy, visually impaired users to have portability and variable magnification in one piece of equipment. These devices are ideal for patients who require more assistance than optical aids such as high-powered reading glasses can provide, but do not necessarily need or want a large stationary closed circuit television (CCTV).

This category of devices can be moderately priced and can be used in a variety of settings. Furthermore, electronic video magnifiers as a whole provide a wide range of magnification, contrast and enhancement modes allowing for customization by the user.

With all the devices that are available currently, it can be a daunting task to help patients decide which may be the best device for their needs. Practitioners must take into consideration the patient goals, best-corrected visual acuity, size of magnification and portability including size and battery life.

With these considerations, there are some options such as the Explore 5 (New England Low Vision and Blindness), the Ruby XL HD (Freedom Scientific) and the Pebble HD (Enhanced Vision). The Explore 5 may be used simply handheld like a cellular phone, with the attached folding handle or on a tabletop.[7] It offers magnification extending from 2x to 22x on a five-inch screen.[7] It can store images for viewing at the user's convenience and also be connected to a television to display both pictures and text. The Ruby XL HD offers a built-in reading line to help users keep their place on a page while reading, which is a common complaint amongst people with visual impairment in my experience. Its large color-coded buttons with easy to decipher markings make it simple for patients to adjust the level of magnification.[8]

The Ruby XL is useful to a broad spectrum of users, including those with central vision loss and monocular patients, due to its ability to enlarge objects of interest two to 14 times the original size, decreasing strain and fatigue many patients with low vision experience. Moreover, it is compatible with computers.

Lastly, the Pebble HD has a real-time clock and calendar and provides audible feedback.[9] These features can help users keep track of appointments, scheduling and time management. This device allows practitioners an option for those requiring either a little or a lot of help with visual tasks as well as those needing a convenient portable option.

Wearable Technology

These assistive technologies are among the newest options available to eye care practitioners and patients. This branch of technologically advanced options is growing rapidly and extending the list of choices to consider when implementing a low vision device as part of a treatment plan.

As a group they offer extensive magnification ranges and great portability. They are also becoming less bulky and more aesthetically pleasing to potential users. Eye care professionals may want to consider wearable devices in patients with tremors, paralysis or muscle weakness and for long-term tasks.

One such device is the OrCam MyEye 2.0. The OrCam is a wearable pair of glasses with an accompanying camera. It is easy to use and responds to simple hand gestures.[10] It is capable of reading printed text as well as text displayed on electronic screens such as comput-

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ers, tablets or phones. It affords the user the ability to recognize faces and audibly announces the person in real-time.[10] OrCam does not need an Internet connection and can be controlled with more than 20 voice commands.[10]

IrisVision is one of the newest wearables among low vision devices. It may be used in patients with a variety of ocular diseases including age-related macular degeneration and retinitis pigmentosa, which cause central vision loss and peripheral vision loss respectively. This device is able to give patients an individualized experience with the unit. The IrisVision allows the operator to enjoy hobbies, read and recognize faces with a 70-degree field of view. [11] It uses the power of a headset along with a smartphone camera to capture the scene.

The Jordy (Enhanced Vision) holds a battery life of eight hours and an autofocus camera that works at distance, intermediate and near.[12] It gives the user clear vision through a wide range of tasks and can be worn like glasses.[12] The Jordy also has CCTV with docking station available for users who may be interested in a stationary desktop device in the home, giving two-in-one convenience.

Devices in this category are exceptional options for practitioners looking for portable, versatile and hands-free suggestions for patients with vision loss. This area is rapidly growing and is becoming a mainstay of low vision intervention. These devices allow users to regain and maintain an active lifestyle and independence.

Apps and Accessibility

Regardless of age, many patients that optometrists encounter have some working knowledge of smartphones and tablets and often use these devices daily for work, entertainment or school. These common devices are obvious choices for practitioners to recommend as part of their low vision treatment plan. Two paramount reasons for this are that most patients already possess the hardware with some built-in accessibility options and that many very useful, applications (apps) are free and accessible within minutes. Whether the patient is an Apple user or an Android devotee, both operating systems have an "accessibility" menu of settings where these built-in features can be enabled and adjusted to the user's preference.[13]

These built-in accessibility features are often intuitive and simple to use, making them a good first-line rehabilitation tool for patients. Optometrists can easily do this training as part of a basic low vision exam. Often, this is one of the first things I go over with patients when inquiring about their previous use of low vision devices. If a patient is unaware of the built-

in features, I do some training during their first visit with the phone they already own.

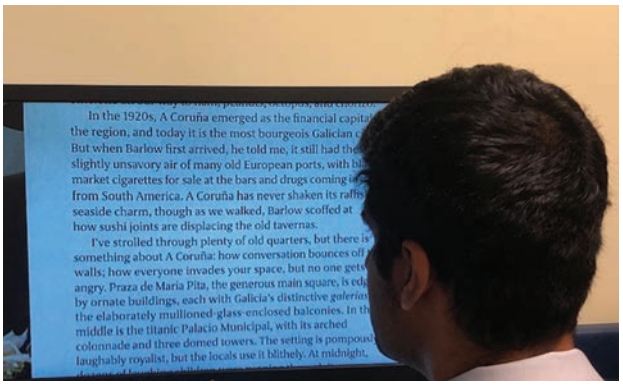
Some of the most useful built-in features include voice-command, enlarged text, VoiceOver and a built-in zoom capability. Voice-command assistants are available on multiple platforms, most popularly Siri for iPhone. This feature allows users to vocally request for multiple tasks such as making phone calls, reading/sending text messages or making list and reminders. This common feature is invaluable to users for navigating their commonly used devices. Teaching visually impaired patients how to use this feature effectively allows for more independence and control while using technology.

Many of the devices in this genre include the ability to enlarge text, photos and images displayed on the screen through finger gestures, eliminating the need for additional devices or magnifiers for visual tasks. The VoiceOver option allows the device to verbally speak word, numbers, or icons displayed on the screen.[13] Navigating the plethora of options available can be tricky and many owners of smartphones and tablets do not realize that they are available and can instantly make many daily activities easier to manage.

Along with included features, apps are another area of growth in the assistive technology space. Apps allow for personalization of a user's smartphone or tablet to assist with daily tasks such as splitting a check at a restaurant or managing bank accounts. A currently promising and useful app is Seeing AI by Microsoft. It helps describe environments and people in the space around the user, including both indoor and outdoor spaces. Furthermore, the app reads aloud text, reads documents and handwriting, recognizes currency, recognizes people and reads barcodes for product recognition.[14] This one app encompasses multifunctionality, offer-

Hotline for Low Vision Consumers

Patients suffering from vision loss need all the allies they can get. Now there's a phone number they can call to seek help in finding products that can make their lives a bit easier. The Accessible Products Hotline (316-252-2500) connects low vision patients with professional advice about purchasing and operating products especially suited for their unique accessibility needs. Items highlighted by the hotline, such as memo recorders, microwave ovens and headsets, were selected for their strong accessibility features and were recommended by other users who are blind or have low vision. The hotline was recently launched by Envision, an advocacy group for the visually impaired, based in Wichita, KS.



Becoming familiar with devices for low vision patients, such as a monocular telescope (left) or a stationary CCTV (right), can help your practice assist in a wide range of their visual needs.

ing the operator the capacity to move seamlessly and comfortably through environments where multiple types of visual tasking is required.

The Visor-Magnifier app essentially turns the user's current device, (iPhone or iPad) into a portable electronic magnifier. This app in conjunction with any built-in accessibility features provides a robust experience for the visually impaired user without the cost of purchasing a new piece of equipment.[15]

Aira is an innovative app and service that can either be used with an existing smartphone or with Aira smart glasses. The app along with a smartphone camera allows live video stream to be sent to a real person, who can be immersed into the user's environment in real-time to provide assistance. The service's agents have access to maps, search engines and ride share services to facilitate the users experience.[16] The app is free and many of the features may be used for free in certain locations such as drug stores, airports and federal buildings. The service is available anytime 24 hours a day/seven days a week. This service, via its app provides the ultimate in independence, safety and freedom for visually impaired individuals.

All eye care providers can overcome the challenges of visually impaired patients seeking assistance, through simple low vision intervention techniques. With the help of these quick tips and resources, most practitioners will successfully be able to implement basic, cost-effective and timely low vision strategies. With these strategies, practitioners may return functioning and hope to those experiencing vision loss.

Consider simple telescopic devices, portable electronic devices, wearables and apps as viable options for low vision patients when implementing a low vision plan of care. In addition, embrace an interdisciplinary approach—including occupational therapists, low vision therapists, orientation and mobility specialists and mental health professionals will aid in the care of the "whole" patient, which is the future of healthcare at its best. Using these tools and technologies will broaden your patient base and enhance the quality of care provided to your patients and their families. ■

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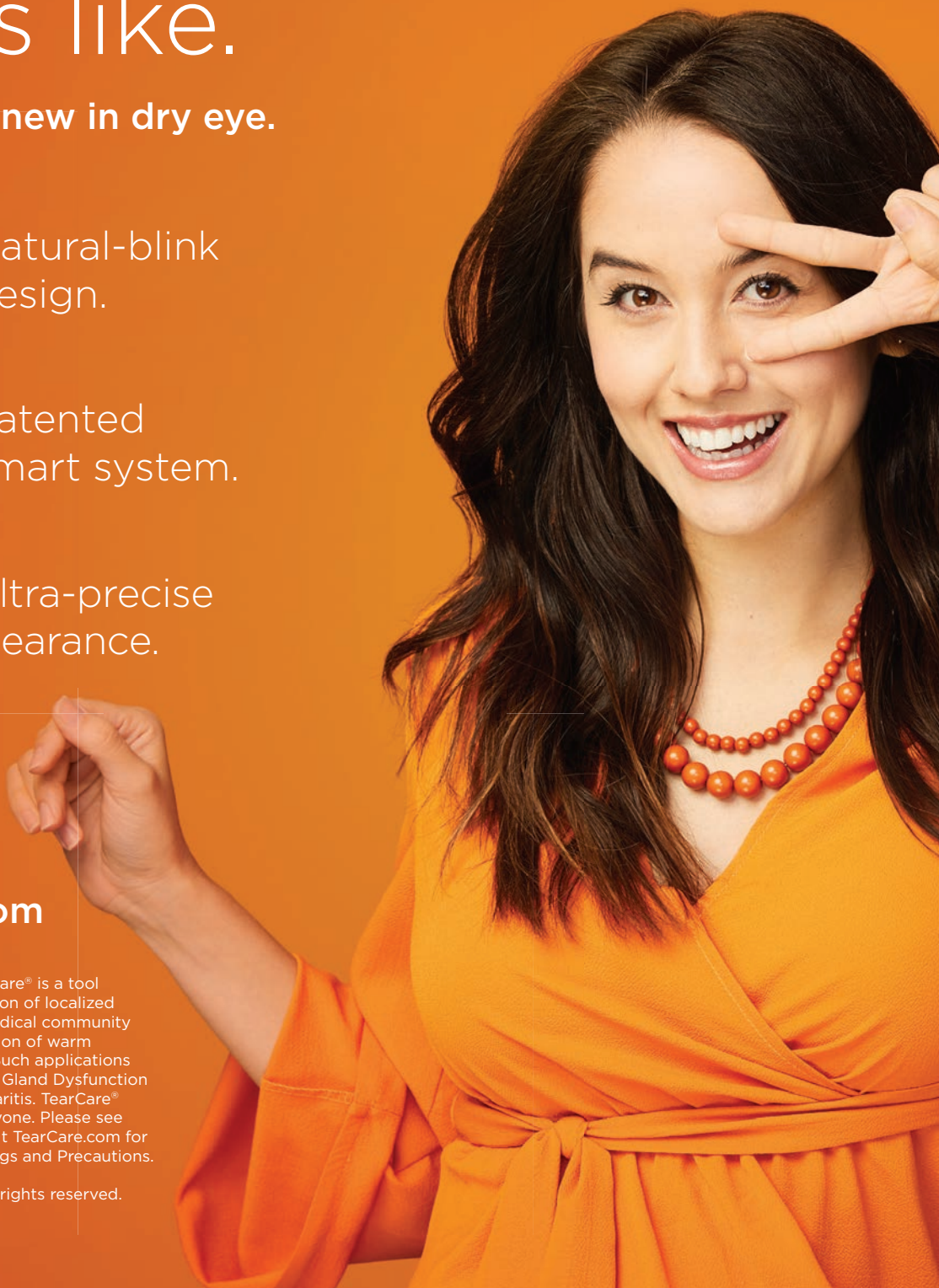

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Pinnacles of Awareness in AMD

How milestones in our understanding of disease mechanisms are shaping modern standards of care, allowing for meaningful intervention prior to vision loss.

BY JOSEPH J. PIZZIMENTI, OD, FAAO

It was the 1980s and, as a third-year intern, I was feeling proud of myself for detecting and correctly diagnosing macular drusen in one of my older patients. Later, at an end-of-the-day debriefing, my preceptor asked me to briefly explain how age-related drusen come about. While I was able to toss out a few sentences about normal macular function, when it came to abnormal retinal metabolism in AMD, I was at a complete loss for words.

The next day I received several printed articles in my student mailbox with a note to the effect of: "Joe, if you are serious about your patients' retinal health, you need to understand both normal *and* abnormal functional anatomy. Be able to intelligently talk about theories of AMD pathogenesis the next time I see you." I took these words to heart.

One of the publications I found in my mailbox was a landmark review of AMD pathogenesis by R. W. Young.[1] As a result of reading this and the other articles, I learned that the clinical and histopathological features of AMD involve a complex relationship—with age, heredity, environmental and systemic factors, thickening of Bruch's membrane, and the formation of basal laminar and basal linear deposits, pigmentary disturbances and drusen all having significant roles.[1]

After graduation, and for many years following, I continued my investigation into the pathogenesis of AMD and grew increasingly passionate about how various diagnostic approaches might help us detect—and ultimately manage—the disease before it causes irreversible

Release Date: September 15, 2019
Expiration Date: August 26, 2022
Estimated time to complete activity: 2 hours
Educational Objectives: After completing this activity, the participant should be better able to:
• Describe the histopathology of AMD.
• Evaluate strategies, tools and technologies that can aid in the diagnosis and monitoring of patients with AMD.
• Discuss modern approaches to diagnosing and managing AMD.
• Understand the practical challenges associated with diagnosing AMD using only traditional subjective clinical assessment and structural tests.
• Outline the benefits of a functional diagnostic test for AMD.

Target Audience: This activity is intended for optometrists engaged in the care of patients with AMD.

Faculty/Editorial Board: Joseph J. Pizzimenti, OD, FAAO, Rosenberg School of Optometry University of the Incarnate Word

Continuing Education Credit: This activity, COPE Activity Number 118000 is accredited by COPE for continuing education for optometrists. This course is COPE approved for 2 hours of CE credit. Course ID is 64461-PS. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

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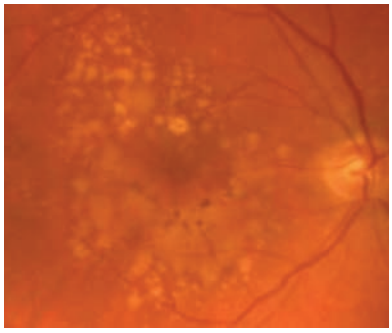

Images: Joseph J. Pizzimenti, OD, FAAO

Figure 1. Large drusen are rich in lipids, protein, and cholesterol.

damage and loss of visual acuity. It was a slow and arduous climb, but we have finally reached a peak in our understanding of AMD. Equipped with this broader view of what causes AMD, we've been able to develop appropriate tools and improve diagnostic and management standards to a degree that was unimaginable only a few short years ago.

WHY PATHOGENESIS MATTERS

When we think of AMD, our minds automatically race to choroidal neovascularization (CNV), geographic atrophy (GA), severe vision loss, and a lifetime of intravitreal injection therapy. We're so fixated on what happens at the end of the road that we fail to recognize and appreciate what optometrists can do along the way to minimize vision loss. But to truly appreciate how we can affect change in AMD, it's essential to understand what causes it in the first place.

Obviously, age-related changes occur in the eyes of each and every one of our patients. But what makes AMD different and what causes some patients to turn the corner and others to remain relatively healthy? In short, AMD represents a *pathologic* stage of an otherwise normally occurring deteriorative process. Therefore, our challenge as clinicians is to determine which patients are going to fall off that cliff and develop true AMD and which ones will never progress to a pathologic state.

This used to be exceedingly difficult since age-related structural changes take place in the eyes of almost all of our older patients. Fortunately, newer methods are allowing us to detect functional markers earlier in the disease continuum, which allows us to intervene early and with confidence.

HISTOPATHOLOGY AND LIPID DEPOSITION

Retinal health is contingent upon the relationship between photoreceptors and the retinal pigment epithelium (RPE).[2] Indeed, the accumulation of photo-oxidized debris within and under the RPE is now considered the initiating cause of AMD.[3] The primary lesion appears to reside in the RPE, likely resulting from its high rate of molecular degradation. RPE cells, in response to many negative stimuli, go through morphological changes such as hypertrophy, atrophy and intraretinal migration.[4]

Beginning early in life, and continuing throughout the decades, RPE cells gradually accumulate pockets of molecular debris.[1] These residual bodies (lipofuscin) are remnants of the incomplete degradation of abnormal molecules that have been damaged within the RPE cells or derived from phagocytized rod and cone membranes.[1]

Cholesterol deposition in the RPE/Bruch's choriocapillaris complex is the forerunner to drusen. More specifically, drusen can be described as extracellular deposits of lipids and proteins under the RPE. Furthermore, cholesterol in a druse acts in the same way as cholesterol found in carotid arteries of patients with atherosclerosis, as both involve lipoprotein retention. In AMD, lipidation of Bruch's membrane impairs transport of compounds necessary for the health of the RPE and photoreceptors.[5]

Histopathological studies have shown that the RPE cells deposit locally generated cholesterol beneath the RPE cell layer (basal laminar deposits) and in Bruch's membrane (basal linear deposits) before drusen are formed.[6,7] This cholesterol accumulation impairs normal transport of vital nutrients, including vitamin A, across Bruch's membrane and causes oxidative stress, inflammation and a localized vitamin A deficiency. Although these deposits may not become visible drusen for several years after their formation, this is where they begin. In other words, when cholesterol becomes sufficiently deposited, damage is well underway as it builds over time to become a visible druse. So what does it mean to have subclinical AMD?

In subclinical disease, a condition has no (or only minimally recognizable) clinical findings. While it is not yet clinically detectable, subclinical disease is destined to become clinical disease. This is not the same as preclinical disease. A preclinical disease is in the stage of progression immediately before clinical symptoms begin.[8] Not all diseases have a recognizable preclinical stage. But in those that do, it refers to the time when early changes are taking

AMD and Cataracts

Structural signs of macular degeneration can be difficult to view or detect through a cloudy crystalline lens, but early disease identification is imperative, especially in patients who are considering a presbyopia-correcting IOL. Loss of contrast sensitivity is present even in mild forms of AMD, making implantation of a multifocal IOL a relative contraindication[1]—or at least a reason to proceed with extreme caution. Since both macular degeneration and multifocal IOLs reduce patients' contrast sensitivity, one would face a compounded reduction in contrast sensitivity and perhaps decreased visual outcomes.[2] And this happens more often than you might suppose.

In a study presented at the XXXIV Congress of the European Society of Cataract and Refractive Surgeons,[3] researchers asserted that screening is needed. Their retrospective chart review identified 193 patients who underwent dark adaptation testing within a 13-month period. A total of 27 patients had both a normal fundus exam and normal corneal topography, making them candidates for multifocal IOLs. Of these 27 eyes, 17 (63%) had normal dark adaptation and 10 (37%) had abnormal dark adaptation. In other words, if all 27 of these patients opted to undergo cataract surgery with a multifocal IOL, more than one-third of them would likely experience problems that could not have been anticipated if dark adaptation had not been measured preoperatively.

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place (for example, in the retina) that are detectable through certain tests but not yet showing as a clearly recognizable condition. Whereas pre-clinical disease will usually soon progress to the clinical stage with characteristic signs and symptoms, a subclinical condition may remain latent or not progress until some future time.[8] That's great news for patients who are diagnosed with AMD at a subclinical stage.

Later, the basal laminar/basal linear deposits and subsequent drusen may trigger the further contamination of the RPE/Bruch's membrane/choriocapillaris complex. In eyes that continue to progress over time, loss of vision results from cellular death due to degeneration and atrophy of the RPE, and/or from the effects of neovascular membranes that invade from the choroid.[1]

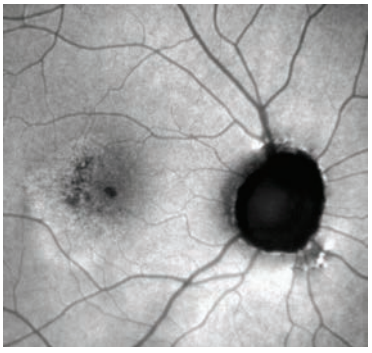


Figure 2. Non-exudative AMD on fundus autofluorescence showing (dark) areas of RPE atrophy.

In AMD, an inflammatory response to the accumulated lipid material may likewise ensue.[5,9] This activates complement factors and other immune system components, leading to RPE atrophy and/or induction of a pro-angiogenic state and choroidal neovascularization (CNV). For this reason, elimination of large, lipid-rich, sub-RPE drusenoid deposits is regarded as a potential benefit to patients.[5,9,10]

RODS, CONES AND THE MACULA

The macula is a relatively large area, 6mm in diameter, or 21.5 degrees of visual angle.[11] The small, cone-dominated fovea is only 0.8mm in diameter (2.75 degrees). The central 300µm of the fovea, or foveola, is totally rod-free. So why do rods matter in AMD? First, the cone-rich fovea is surrounded by a rod-dominated parafovea (1mm to 3mm from the fovea or 3.5 to 10 degrees from fixation).[11] In healthy, young adults, rods outnumber cones in the macula by 9:1. Therefore, the macula may be described as cone-enriched but rod-dominated.

Rods are responsible for our most sensitive motion detection, peripheral vision and night vision.[12] Curcio and colleagues reported that in maculae of older adults without visible drusen and pigmentary disturbances, the number of cones was stable through the ninth decade. However, the number of rods in the maculae of the same eyes decreased by 30%, with the greatest loss occurring in the parafovea.[13]

Further research found that the foveal cone mosaic of eyes with thick basal laminar deposits and large drusen appeared surprisingly similar to that of age-matched controls, and the total number of foveal cones was normal.[14] By contrast, in the parafovea of those same eyes, the cones appeared large and misshapen, and very few rods remained. In fact, in those eyes with late AMD, virtually all surviving photoreceptors in the macula were cones, a reversal of

the normal predominance of rods.

It appears that in AMD, there is preferential loss of rods over cones. Why does this occur? Psychophysics may help to uncover some answers. The classic dark adaptation function describes the recovery of retinal sensitivity after a bright flash of light. It consists of an early portion exclusively mediated by cones, a transition to rod function (rod-cone break), and a later portion exclusively mediated by rods.[15]

Studies of photopic and scotopic sensitivity strongly correlate to histopathologic findings showing that rods are at risk for degeneration in both aging and AMD.[16,17] Researchers also found slowing of the rod-mediated component of dark adaptation and earlier involvement of rods relative to cones in aging and AMD.[17] Mean scotopic sensitivity within 18 degrees of fixation was significantly lower in early AMD patients as a group than in age-matched controls without AMD. This finding was most severe within 9 degrees of fixation, suggesting that scotopic sensitivity deficits within the parafovea may be an early sign or, at least, a harbinger of disease.[17]

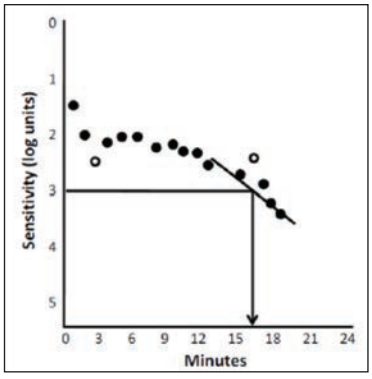


Figure 3. A rod-mediated dark adaptation (or rod intercept) time of 16 minutes. RIT greater than 6.5 minutes indicates a high likelihood of degenerative activity.

Why the rods of the central retina, which share a common light exposure and support system with the neighboring cones, are preferentially vulnerable to aging still remains to be determined. Fortunately, tests of rod function, particularly those that evaluate dynamic properties, may enable detection of AMD at its incipient stages.[16,17]

MEASURING SUBCLINICAL DISEASE

Standard color fundus photography, although historically useful for classifying the stage of AMD, may not readily identify some common early and intermediate manifestations.[18] Indeed, alterations to the RPE may not be clinically detectable by funduscopy or photography. Consider that drusen and subretinal drusenoid deposits become clinically visible at 30µm, while changes in RPE cells are substantially smaller.[19]

Despite these limitations, we must endeavor to find novel ways to improve our capacity to detect disease before it's too late and somehow overcome the current grim statistics. Up to 78% of AMD patients have substantial, irreversible vision loss in one eye at first treatment.[20,21] Why aren't we doing a better job of catching disease earlier?

In short, while we may be excellent diagnosticians, evaluating the fundus for small drusen and early pigmentary disturbances can be quite challenging. A recent study published in *JAMA Ophthalmology* revealed that optometrists and ophthalmologists failed to diagnose AMD about 25% of the time—even when they knew their findings would later be reviewed.[22]

The cross-sectional study included 1,288 eyes (644 adults). Each patient in the study had digital color fundus photos taken, which were reviewed by masked, trained graders who determined the presence or absence of AMD findings according to the Clinical Age-Related Maculopathy Staging (CARMS) system.[23] The results revealed that one of four eyes studied was not diagnosed with AMD during the dilated fundus examination, despite these eyes having macular characteristics indicative of AMD in the fundus photos. Furthermore, 30% of the undiagnosed eyes in the study had large drusen, a well-known risk factor for progression to advanced disease.[22]

Make no mistake, there are significant practical challenges associated with diagnosing AMD using only traditional subjective clinical assessment. For this reason, it is imperative that we begin to look beyond what we can visualize structurally. One way to do this is with risk assessment, such as advanced age, a history of smoking, heredity (genetics) and Macular Pigment Optical Density (MPOD) measurement. Another is with functional diagnostic testing using dark adaptometry.

Because rod deterioration happens in the earliest stages of AMD, dark adaptation becomes affected much earlier than visual acuity declines.[24] Impaired dark adaptation identifies subclinical AMD at least three years before it can be seen with imaging, OCT or clinical examination.[25] Furthermore, impaired dark adaptation is a functional biomarker of true subclinical disease, as opposed to a less definitive indication

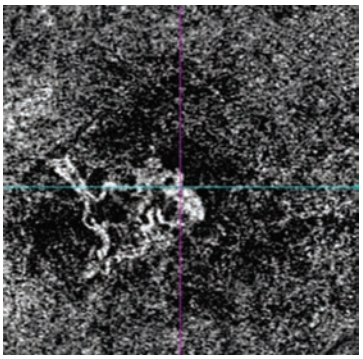
of risk. In fact, the updated preferred practice patterns of the American Academy of Ophthalmology[26] indicate that an initial history should consider difficulties in dark adaptation.

Dark adaptometry measures how long it takes for the eye to adapt from bright light. Dark adaptation assessments proved to be highly sensitive (90.6%) and highly specific (90.5%) to the development of AMD.[27]

EVIDENCE FOR THE BENEFITS OF EARLIER MANAGEMENT

Vision loss from AMD can be functionally and emotionally debilitating, as it can make it difficult, or even impossible, to read, drive, enjoy certain hobbies and maintain an independent lifestyle.[28] So imagine if we could detect subclinical AMD in patients before clinical signs present. Would you want to get them started on nutraceuticals and lifestyle and dietary modifications as early as possible in an effort to slow or delay disease progression? Aside from supplementation, current medical treatment options for AMD are only indicated in patients whose disease leads to the development of CNV. However, there's plenty we can do for patients before they reach that stage. Most critically, we can monitor them very closely so that prompt co-management with a retinologist for appropriate treatment with antiangiogenic agents can be initiated promptly should CNV develop. Consider how much better second eye outcomes are compared to first eye outcomes.[29] More frequent examinations and consistent structural and functional testing can make a meaningful difference. When you know a patient has AMD—however early the stage—moving from a 12-month follow-up interval to a six-month (or even shorter in some cases) may be useful for monitoring disease progression.[30]

Just as in pre-diabetes, our patients with subclinical AMD can implement nutrition and lifestyle measures in an effort to protect against sight-threatening disease. But which ones are appropriate in early-stage AMD? As was discussed above, based on our current understanding of AMD pathogenesis, the stages of subclinical, early and intermediate AMD represent different clinical manifestations of the same underlying disease process. Therefore, all of the following non-medical treatments should be considered at first detection, regardless of the disease stage:



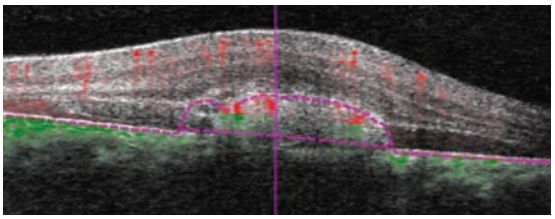


Figure 4. A CNV lesion, as revealed with Cirrus 5000 OCT with Angioplex (Zeiss).

• ***Smoking cessation.*** Smoking is the largest modifiable risk factor for the progression of both CNV and GA,[31] so it is critical to emphasize this with patients. Surprisingly, in a recent study, 90% of patients with AMD said they were not advised to stop smoking.[32]

• ***Nutritional supplements.*** Although there is debate about which supplements are most appropriate at various stages of disease, the evidence suggests prescribing a high-quality, broad-spectrum antioxidant formula. In fact, nutritional trials and related studies have demonstrated a slowing of progression for established AMD.[33-36]

• ***Lifestyle modification.*** Following a healthy diet rich in leafy greens, fresh produce and omega-3 rich fish, exercising regularly and maintaining overall health are sound goals[37] that may act synergistically to prevent or delay onset or progression of AMD. Research shows that individuals who followed a healthy diet, engaged in physical exercise and avoided smoking had substantially lower risk of early AMD compared with those who did not follow these healthy lifestyles.[38,39]

• ***Systemic disease management.*** Cardiovascular disease, diabetes, hypercholesteremia and obesity have been associated with increased risk of AMD and/or progression of AMD.[40,41] High body mass index and abdominal obesity are independent risk factors for progression to advanced AMD.[40]

• ***Retinal light protection.*** Epidemiological evidence suggests that chronic sunlight exposure

increases the risk of incident AMD and its progression.[42] As such, it is advisable for patients at risk for, or with early signs of, AMD to wear quality sunglasses that significantly reduce the transmittance of high energy visible light (HEVL).

IT'S TIME TO ELEVATE AMD CARE

An understanding of normal functional anatomy, as well as abnormal retinal physiology helps us provide optimal care because it expands our view beyond end-stage disease and clarifies how we can make a meaningful impact before it's too late. A microscopic cholesterol layer is a hallmark defect of AMD, and even though it is not yet visible in living eyes using current diagnostic tools, it is detectable using functional tests.[6,24]

It's time to look forward instead of backwards. The current AMD grading scales, though useful in research and for documentation purposes, won't help optometrists raise standards in caring for this devastating disease. Furthermore, these staging schemes were created before we had information about the link between impaired dark adaptation function and AMD. This is now well established and must therefore drive our clinical practice protocols.

We can make a meaningful difference in countless lives and families. Clinical AMD is more prevalent than glaucoma and diabetic retinopathy combined.[43,44] How many of these patients are we diagnosing while they still have 20/20 vision? It's time to raise the bar and set our sights higher.

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CE TEST

You can obtain continuing education credit through the Optometric Study Center. Complete the test form and return it to: Jobson Healthcare Information, LLC, Attn.: CE Processing, 395 Hudson Street, 3rd Floor New York, New York 10014. You can also access the test form and submit your answers at Review Education Group online, www.reviewsce.com.

You must achieve a score of 70 or higher to receive credit. Allow four weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of credit from UAB School of Optometry.

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

1. Which of the following statements is accurate?
a. AMD represents a pathologic stage of an otherwise normally occurring deteriorative process
b. All stages of AMD are part of the normal aging process
c. AMD is a normally occurring deteriorative process
d. Any age-related deteriorative process is considered pathologic

2. Age-related structural changes take place in the eyes of _____.
a. Less than 10% of older patients
b. 25% of older patients
c. 58% of older patients
d. Almost all older patients

3. The accumulation of _____ within and under the RPE is considered the initiating cause of AMD.
a. Vitamin A
b. Cone membranes
c. Photo-oxidized debris
d. Blood

4. Cholesterol deposition in the RPE/Bruch's choriocapillaris complex _____:
a. Always develops after drusen appear
b. Is a forerunner to drusen
c. Is a sign of late stage AMD
d. Is clinically meaningless

5. Drusen can be described as:
a. Extracellular deposits of lipids and proteins under the RPE
b. A sign of AMD if they are 63-124 microns in diameter
c. A sign of AMD if they are greater than 125 microns in diameter
d. The most reliable indicator of early AMD

6. What is another name for locally generated cholesterol beneath the RPE cell layer?
a. Basal linear deposits
b. Basal laminar deposits
c. Drusen
d. CNV

7. What is another name for locally generated cholesterol in Bruch's membrane?
a. Basal linear deposits
b. Basal laminar deposits
c. Drusen
d. CNV

8. Basal laminar deposits and basal linear deposits:
a. Are unrelated to the formation of drusen
b. Occur before drusen are formed
c. Occur after drusen are formed
d. Begin to occur after CNV

9. Which of the following best describes subclinical AMD?
a. At least one drusen that is 63 microns in diameter
b. Two or more druse that measure at least 63 microns in diameter
c. At least two AMD risk factors and any small drusen
d. Basal laminar and basal linear deposits that are not yet clinically visible

10. Subclinical disease is _____:
a. The same as preclinical disease
b. In the stage of progression immediately before clinical symptoms begin
c. Destined to become clinical disease
d. Not a reliable way to determine whether a patient will develop a disease

11. The central 300µm of the fovea is _____:
a. Cone-dominated, with no rods
b. Cone-dominated, rod-enriched
c. Rod-dominated, with no cones
d. Rod-dominated, cone-enriched

12. The macula as a whole is _____:
a. Cone-dominated, with no rods
b. Cone-dominated, rod-enriched
c. Rod-dominated, with no cones
d. Rod-dominated, cone-enriched

13. Rods are responsible for _____:
a. Sensitive motion detection
b. Peripheral vision
c. Night vision
d. A, B, and C

14. The number of _____remains stable, regardless of age and drusen formation.
a. Rods
b. Cones
c. Rods and cones
d. Rods, cones and basal laminar deposits

15. Tests of _____may enable detection of AMD at its incipient stages.
a. Rod function
b. Rod volume
c. Cone function
d. Cone volume

16. Drusen and subretinal drusenoid deposits become clinically visible at a size of _____:
a. 10µm
b. 20µm
c. 30µm
d. 40µm

17. Up to _____of AMD patients have substantial, irreversible vision loss in one eye at first treatment.
a. 18%
b. 38%
c. 58%
d. 78%

18. In a study of 1,288 eyes that was published in *JAMA Ophthalmology*, optometrists and ophthalmologists who were told that their findings would later be reviewed, failed to diagnose AMD about _____of the time.
a. 5%
b. 10%
c. 15%
d. 25%

19. Which of the following is a functional diagnostic test?
a. Dark adaptometry
b. OCT
c. MPOD
d. Direct ophthalmoscopy

20. Impaired dark adaptation identifies subclinical AMD _____:
a. Only after it can be seen with fundus imaging
b. Only after it can be seen on a clinical examination
c. Only after it can be seen with OCT
d. Before it can be seen with retinal imaging, OCT or clinical examination

Examination Answer Sheet

Pinnacles of Awareness in AMD

Valid for credit through August 26, 2022

Online: This exam can be taken online at www.reviewsce.com. Upon passing the exam, you can view your results immediately and download a real-time CE certificate. You can also view your test history at any time from the website.

Directions: Select one answer for each question in the exam and completely darken the appropriate circle. A minimum score of 70% is required to earn credit.

Mail to: Jobson Healthcare Information, LLC, Attn.: CE Processing, 395 Hudson Street, 3rd Floor New York, New York 10014

Credit: This course is COPE approved for 2 hours of CE credit. COPE Activity Number is 118000. Course ID is 64461-PS.

UAB School of Optometry has sponsored the review and approval of this activity.

Processing: There is a four-week processing time for this exam.

Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ
5. Ⓐ Ⓑ Ⓒ Ⓓ
6. Ⓐ Ⓑ Ⓒ Ⓓ
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8. Ⓐ Ⓑ Ⓒ Ⓓ
9. Ⓐ Ⓑ Ⓒ Ⓓ
10. Ⓐ Ⓑ Ⓒ Ⓓ
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16. Ⓐ Ⓑ Ⓒ Ⓓ
17. Ⓐ Ⓑ Ⓒ Ⓓ
18. Ⓐ Ⓑ Ⓒ Ⓓ
19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

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

21. Describe the histopathology of AMD. ① ② ③ ④ ⑤
22. Evaluate strategies, tools and technologies that can aid in the diagnosis and monitoring of patients with AMD. ① ② ③ ④ ⑤
23. Discuss modern approaches to diagnosing and managing AMD. ① ② ③ ④ ⑤
24. Understand the practical challenges associated with diagnosing AMD using only traditional subjective clinical assessment and structural tests. ① ② ③ ④ ⑤
25. Outline the benefits of a functional diagnostic test for AMD. ① ② ③ ④ ⑤
26. Based upon your participation in this activity, do you intend to change your practice behavior?
(choose only one of the following options)
Ⓐ I do plan to implement changes in my practice based on the information presented.
Ⓑ My current practice has been reinforced by the information presented.
Ⓒ I need more information before I will change my practice
27. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ☐

28. If you plan to change your practice behavior, what type of changes do you plan to implement? (check all that apply)

ⓐ Apply latest guidelines ⓑ Change in pharmaceutical therapy ⓒ Choice of treatment/management approach ⓓ Change in current practice for referral ⓔ Change in non-pharmaceutical therapy ⓕ Change in differential diagnosis ⓖ Change in diagnostic testing ⓗ Other, please specify: _____

29. How confident are you that you will be able to make your intended changes?

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

30. Which of the following do you anticipate will be the primary barrier to implementing these changes?

ⓐ Formulary restrictions
ⓑ Time constraints
ⓒ System constraints
ⓓ Insurance/financial issues
ⓔ Lack of interprofessional team support
ⓕ Treatment related adverse events
ⓖ Patient adherence/compliance
ⓗ Other, please specify:

31. Additional comments on this course:

_____
_____
_____
_____
_____

Please retain a copy for your records. Please print clearly.

First Name
Last Name
E-Mail

The following is your: ☐ Home Address ☐ Business Address

Business Name
Address
City State
ZIP
Telephone # - -
Fax # - -

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

Signature _____ Date _____

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

32. The content was evidence-based. ① ② ③ ④ ⑤
33. The content was balanced and free of bias. ① ② ③ ④ ⑤
34. The presentation was clear and effective. ① ② ③ ④ ⑤

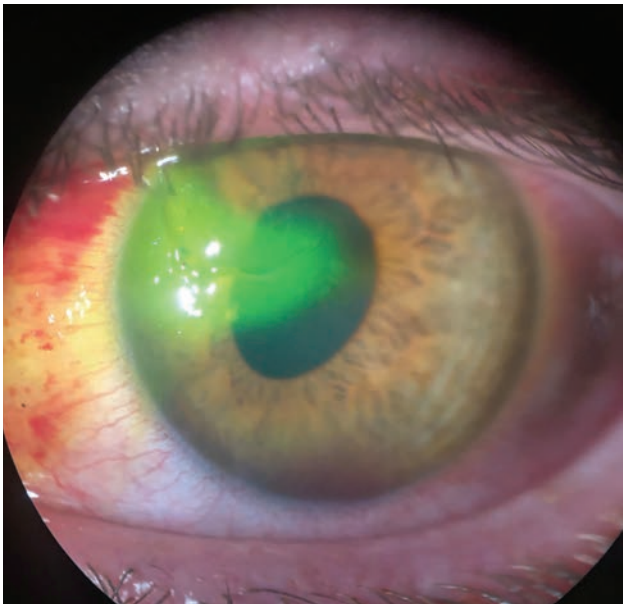
Developing a Pain Pill Protocol in Optometry

Now that ODs in most states can prescribe them, learning the nuances of this class of drugs is a must. **By Tracy Offerdahl-McGowan, PharmD, BPharm, RPh, P. Scout McGowan, Greg Caldwell, OD, and Marissa Mangoni**

Treating pain has long been a difficult task for practitioners, particularly with so many drugs on the market. Further complicating this topic is the subjective nature of pain, as there is no objective way to assess the true level of a patient's pain. Several pain scales are available, including a numerical scale, a color scale and a facial grimace scale; however, these are somewhat subjective as a patient may underestimate or overestimate their pain intensity.

The two main types of pain are nociceptive and neuropathic pain. Nociceptive pain is the normal processing of pain, where pain is in response to signals conducted from a normal, intact nervous system. Neuropathic pain occurs when signals are sent to a damaged nervous system.[1,2] In quantifying pain using the numeric scale, mild pain is 1-3, moderate pain is 4-6, and severe pain is 7-10.

Pain that presents in the optometric patient will generally be acute and nociceptive in nature and will be effectively treated with non-opioids and opioid medications. Painful ocular conditions include trauma, infection, uveitis corneal abrasions or ulcers or scleritis. In choosing an agent to manage acute pain, use one appropriate for the level and type of pain (i.e., inflammatory vs. non-inflammatory). It should have a fairly rapid onset, an extended half-life, while causing the fewest drug interactions and side effects possible.[3,4] With or without a DEA license, optometrists can effectively treat most patients with eye pain with currently available over-the-counter and prescription agents.



This patient suffered a traumatic corneal laceration after a mishap with a screwdriver. Note the sodium fluorescein being pulled into the stroma and the oblong pupil.

Mild to Moderate Pain

Tylenol (acetaminophen, Johnson & Johnson) may cause rather mild inhibition of prostaglandin synthesis. It has no useful anti-inflammatory potential.[6] Make sure your patients choose a product that has acetaminophen as a single ingredient, as many over-the-counter products contain acetaminophen with unnecessary ingredients, such as antihistamines, decongestants and

expectorants. While the FDA still lists the maximum daily dose at 4,000mg, some practitioners prefer to limit that to closer to 3,000mg since antihistamines can cause unwanted dry eyes and drowsiness.[9]

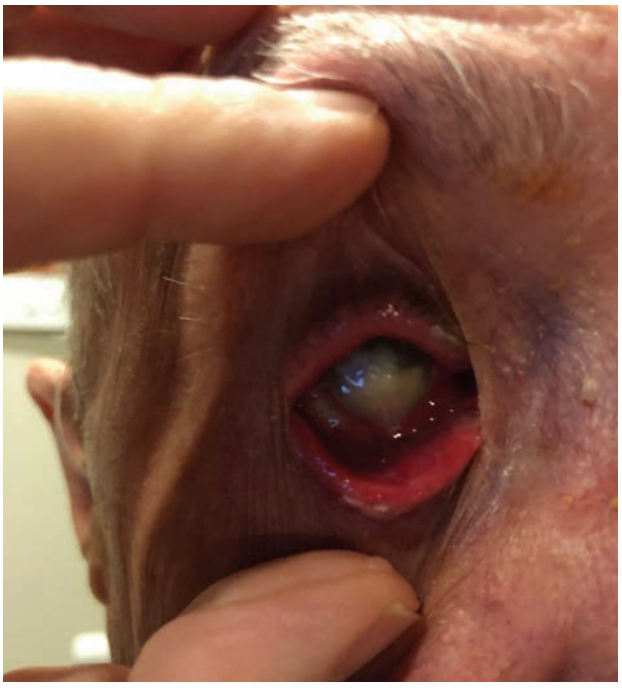
Side effects associated with acetaminophen are fairly mild as long as patients follow the appropriate dosing guidelines. Potential adverse effects include hepatotoxicity. In fact, in the United States, acetaminophen is one of the leading causes of acute liver failure requiring a transplant.[7] The Food and Drug Administratio- suggests that patients who consume more than three alcoholic beverages per day are at an increased risk for hepatotoxicity. Usually, hepatotoxicity is unintentional, where patients do not follow appropriate safe dosing guidelines or where they mix acetaminophen with other pain medications that also contain acetaminophen (i.e., Vicodin, Percocet).[8] In 2011, the FDA asked manufacturers of prescription products that contain acetaminophen, to reformulate all products to contain no more than 325mg of the drug per dosage unit.[9] Acetaminophen is generally safe in pregnancy and breastfeeding.[2,3,9]

Nonsteroidal anti-inflammatory drugs (NSAIDs) include a variety of agents that differ in structure as well as in side effect potential. In the general pain management of an optometric patient, Advil (ibuprofen, Pfizer) or Motrin (ibuprofen, Johnson & Johnson) and Aleve (naproxen sodium, Bayer) are typically the only agents needed to effectively control mild-to-moderate pain, and they have the added benefit of having anti-inflammatory potential, which may be beneficial in patients with inflammatory eye pain. Aspirin is also an NSAID; however, its use is generally limited to A low dose as a cardiac protectant in select patients, mainly because of side effects like hypersensitivity, tinnitus, and gastrointestinal erosion in all patients, and Reye syndrome in susceptible children.[10]

Mechanistically, traditional NSAIDs reversibly inhibit cyclooxygenase-1 and 2 (COX-1 and 2) enzymes, which decrease the formation of prostaglandin precursors.[10-12] This prostaglandin inhibition gives us a decrease in pain, inflammation and fever; however, it also produces the gastrointestinal and renal side effects that are associated with NSAIDs. Additionally, aspirin irreversibly inhibits thromboxane A2, which is what results in the cardiac protection.[10-12] NSAIDs do not and instead result in a potential increase in cardiovascular risk.[10-12] The dosing of NSAIDs varies greatly; however, the anti-inflammatory potential of these agents is generally only seen at the higher end of the dosing range (*Table 2*).[3,11]

Mild-to-moderate and Moderate Pain Meds

- **Non-opioids:** Acetaminophen
- **NSAIDs:** Ibuprofen, Naproxen sodium
- **Cyclo-oxygenase inhibitors:** Celecoxib, Meloxicam



This patient presented with severe pain secondary to endophthalmitis that he developed after being noncompliant following a corneal transplant.

The side effects of traditional NSAIDs are well documented. Because NSAIDs reversibly inhibit prostaglandins, platelet aggregation effects may increase the cardiovascular risk, particularly when used long-term or in those with underlying cardiovascular/cerebrovascular disease. Additional toxicities include gastrointestinal (GI) erosion, renal vasoconstriction, hypersensitivity (rash, respiratory, anaphylaxis) and hematologic abnormalities.[12-15] Upper GI complications are the most common manifestation in the gut and sometimes concomitant use of a proton-pump inhibitor, such as omeprazole or esomeprazole may be warranted. Additionally, all NSAIDs should be taken with food to add a layer of protection between the drug and the lining of the gut. Food is not a fool-proof way to protect against GI issues, as the prostaglandin inhibition is not a topical effect but a systemic effect.[3,11-15]

Renal vasoconstriction is a side effect that is more likely to occur in patients who are dehydrated, elderly, on angiotensin converting enzyme inhibitors or

Table 1. Treatment of Mild to Moderate Pain: Acetaminophen and Traditional NSAIDs – Dosing[6,9,11-13]

Product	Acetaminophen OTC	Ibuprofen OTC	Ibuprofen Rx	Naproxen sodium ** (OTC: Aleve; RX: Naprelan; Anaprox DS) OTC and Rx	Naproxen base** (Naprosyn) Rx Only
Product availability	325mg, 500mg, 650mg tablets.	200mg tablets, gel caps.	400mg, 600mg, 800mg tablets.	OTC: 220mg tablets, capsules. Rx: 275mg, 550mg immediate-release tablets 375mg extended-release tablets.	250mg, 375mg, 500mg immediate- and extended-release tablets 250mg, 500mg and extended-release tablets.
Analgesic dosing	650mg every six hours* or 1,000mg every six to eight hours.* Max daily dose: Do not exceed 3,000mg to 4,000mg in 24 hours.	1 to 2 tablets/ gel caps every four to six hours.* Max daily dose: Do not exceed six tablets/ gel caps in 24 hours.	200mg to 400mg every four to six hours* or 200mg to 800mg every six to eight hours.* Max daily dose: 3,200mg per day	OTC: 220mg to 440mg every eight to 12 hours* or 660mg every 24 hours. Max OTC daily dose: 660mg. Rx immediate-release: 275mg every six to eight hours* or 550mg every 12 hours.* Rx extended release: 375mg twice daily.* Max daily dose: Immediate-Release • Day 1: 1,375mg • After day 1: 1,100mg Extended-release • Days 1-3: 1,500mg • After 1-3 days: 1,000mg	Rx immediate-release: 250mg every six to eight hours.* or 500mg twice a day.* Rx extended-release: 750mg to 1,000mg once daily.* Max daily dose: Immediate-release • Day 1: 1,250mg • After day 1: 1,000mg Extended-release • Day 1-3: 1,500mg • After 1-3 days: 1,000mg
Anti-inflammatory dosing (Higher end of dosing range is generally required)	Not applicable; no anti-inflammatory potential.	No dosing available.	400mg to 800mg every six to eight hours as needed Max dose: 3,200mg per day.	OTC: Not labeled for inflammation. Rx immediate-release: 275mg to 550mg every eight to 12 hours.* Rx extended release: 375mg twice daily.*	Rx immediate release: 500 mg every 12 hours.* Rx extended-release: 1,000mg to 1,500mg once daily.*
Notes* **	Avoid if patient consumes more than three alcoholic beverages per day.	Take with food.	Take with food.	Take with food. Naproxen sodium has a faster onset (30 minutes) than naproxen base (60 minutes).	Take with food. Naproxen base has a slower onset (60 minutes) than naproxen sodium (30 minutes).

* May be dosed "prn" (as needed) or "ATC" (around-the-clock) at the discretion of the optometrist. If inflammation is present, ATC dosing is generally recommended for part or all of the treatment duration.
** 200mg naproxen base = 220 mg naproxen sodium
*** Generally consider using lower doses in elderly patients and in those with renal dysfunction.

angiotensin receptor blockers, or in those with heart failure.[30] More than one risk factor increases the risk of vasoconstriction and acute renal failure. Additionally, NSAIDs can increase blood pressure, as these agents increase sodium and water retention in the blood stream in some patients. Optometrists should be aware of this blood pressure increase; however, it is not necessarily a predictable outcome in every patient who takes a NSAID. Lastly, hypersensitivity reactions may occur in patients taking NSAIDs, exacerbating asthma symptoms or causing a hypersensitivity reaction in patients without asthma. In general, if a patient is allergic to aspirin then there is a chance that they will also react to other NSAIDs. Due to the side effect potential of these agents, it is generally recommended that patients use NSAIDs in lower doses and for the shortest duration of time to limit adverse effects.[3,10,11-15]

NSAIDs are generally contraindicated in pregnancy but may be acceptable in breastfeeding women, depending upon the dose of the drug, the age of the infant and the number of times the infant is nursing. Optometrists are encouraged to consult the patient's pharmacist or pediatrician with questions regarding the safety of drugs in breastfeeding.[11] In patients currently taking Warfarin (coumadin, Bristol-Myers Squibb) or other anti-platelets or anti-coagulants, care must be taken to avoid bleeding Warfarin interacts with NSAIDs by more than one mechanism, so ODs and patients must be in contact with the practitioners that prescribed Warfarin.[3,10-15]

A cyclo-oxygenase 2 (COX-2) specific agent such as Celebrex (celecoxib, Pfizer) or a COX-2 selective agent such as Mobic (meloxicam, Boehringer Ingelheim Pharmaceuticals) may also be reasonable choices in patients with mild-to-moderate pain. Traditional NSAIDs inhibit both COX-1 and COX-2 enzymes in the arachidonic acid cascade. Researchers have discovered that inhibition of COX-2 enzymes is primarily responsible for two things: anti-inflammatory potential and cardiovascular risk. This means that COX-2 agents are better for chronic, inflammatory pain; however, their risk for cardiovascular complications is generally considered to be more likely to occur than with a traditional NSAID.

Regarding the gastrointestinal tract, the COX-2 agents are thought to be marginally less toxic to the gastric mucosa when compared to the traditional NSAIDs.[3,12,17] Additionally, it is thought that COX-2 specific and selective agents are less effective in pain management (particularly acute pain), as COX-1 enzyme is also involved in the transmission of pain. Celebrex and meloxicam are therefore not generally

Table 2: Treatment of Moderate Pain[6,9,11,13,18,19,21,24,25,28]

Product	Acetaminophen + Ibuprofen	Tramadol (Ultram)	Acetaminophen with Codeine
Product Availability	Ibuprofen: OTC: 200mg. Rx: 400mg, 600mg, 800mg. Acetaminophen: OTC: 325mg, 500mg, 650mg.	Immediate-Release: 50mg tablets. Extended-release: 100mg and 200mg.	Acetaminophen 300mg with 15mg codeine=Tylenol #2 30mg codeine=Tylenol #3 60mg codeine=Tylenol #4
Analgesic Dosing	Two 200mg ibuprofen every four hours while awake.* Two 325mg acetaminophen every four hours while awake. Maximum Daily Doses: Ibuprofen: 3,200mg. Acetaminophen: 4,000mg.	50mg to 100mg every six to eight hours.* Maximum Daily Dose: 400mg.	Tylenol #3: One to two tablets every four to six hours.* Maximum daily dose: Codeine: 360mg. Acetaminophen: 4,000mg.
Notes	Take with food. Avoid in patients who drink three or more alcoholic beverages per day. See previous section regarding precautions with NSAIDs. Alternate ibuprofen and acetaminophen every two hours (e.g., Ibuprofen at 8am, acetaminophen at 10am, ibuprofen at 12pm, acetaminophen at 2pm, etc...).	Extended-Release not indicated in acute pain. Maximum daily dose in elderly patients is 300mg daily. Use caution in patients taking selective-serotonin reuptake inhibitors (SSRIs) or triptans as it may result in serotonin syndrome. Avoid or use with caution in patients taking other CNS depressants.	Recommended to use Tylenol #3 due to availability. Avoid in patients who drink three or more alcoholic beverages per day. Avoid or use with caution in patients taking other CNS depressants..

* May be dosed "prn" (as needed) or "ATC" (around-the-clock) at the discretion of the optometrist.

recommended in acute pain scenarios like those seen in optometric patients.[3,12,15-17]

Moderate Pain

Acute pain that is a four to six on the numeric scale may be appropriately treated with higher doses of acetaminophen, NSAIDs or acetaminophen plus NSAID.[31,32] If those drug options are insufficient, practitioners with a DEA license may consider a Schedule IV controlled substance such as Ultram (tramadol, Johnson & Johnson), or a Schedule III controlled substance such as Tylenol-Codine No. 3 (acetaminophen with codeine, Johnson & Johnson). Acetaminophen plus ibuprofen dosed alternately have proven to control pain better than acetaminophen or ibuprofen alone.[6,9,13,18,19] The typical dosing regimen can be referred to as "two and two every four hours," which means that two acetaminophen are given every four hours alternating with two ibuprofen every four hours while awake (*Table 2*).[6,9,13,18,19]

Ultram is another option in the management of moderate pain. It has a dual mechanism of action, as it plugs into the mu opioid receptor as an agonist and it also inhibits the reuptake of serotonin and norepinephrine, which results in a relative increase in both of these neurotransmitters.[15,21-23] Ultram became a Schedule IV controlled substance in 2014, because at higher doses it can cause euphoria. Additionally, it is sometimes used by opioid abusers to mitigate withdrawal symptoms when other opioids are not available. In general, Ultram is fairly well tolerated and is less likely to cause sedation when compared with other opioid analgesics, due to the dual mechanism of action.[15,21-23] Typical side effects include mild sedation, mild constipation and dizziness. The FDA requires Ultram to have a "black box warning" cautioning patients and prescribers about potential respiratory depression and drug interactions. This drug is contraindicated in pregnancy and breastfeeding and should be avoided in patients with allergic reactions to other opioid analgesics and in patients with a substance abuse history.[20,21-23]

Tylenol-Codeine No. 3 is a commonly used combination opioid in the management of moderate pain. It is a relatively weak opioid, and it is generally understood that 200mg of oral codeine is equivalent to 30mg of oral morphine. Mechanistically, opioids such as codeine plug into mu, kappa and delta receptors in

Severe Pain Meds

- **Non-opioids:** Acetaminophen plus ibuprofen
- **Opioids:** Tramadol or Acetaminophen with Codeine

the central nervous system (CNS), producing inhibition of ascending pain pathways.[24,25]

Tylenol-Codeine No. 3 is available in 15mg, 30mg and 60mg tablets; however, it is generally recommended to use the product that contains 30mg of codeine with 300mg of Tylenol-Codeine No. 3, as it is the most widely available at pharmacies. The most common side effects include nausea, vomiting, constipation, sedation, miosis, euphoria, respiratory depression and dizziness.

When an opioid is used in the management of ocular pain, ODs may recommend that the patient take measures to manage the constipation, such as Peri Colace (senna + docusate sodium, Avrio health), which contains both a stimulant laxative and a stool softener, but with a typical course of only three to five days, this may not be necessary.[26] Acetaminophen with codeine is generally contraindicated in pregnancy and breastfeeding, and it should be avoided in combination with other CNS depressants due to additive CNS and respiratory depression. Codeine products should not be used in patients with a Type-1 hypersensitivity reaction to codeine or morphine derivatives, nor in patients with a substance abuse history.[3,4,7,16,20,24,25]

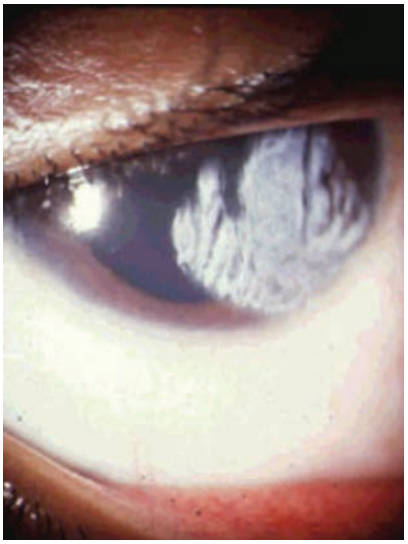
Severe Pain

The highest degree of ocular pain—a seven to 10 on the numeric scale—may be treated with a stronger opioid analgesic, such as hydrocodone with acetaminophen. These options include Vicodin (hydrocodone bitartrate and acetaminophen, Abbott) and Norco (hydrocodone bitartrate and acetaminophen, Allergan). This requires a DEA license that allows optometrists to write for this Schedule II narcotic. Any hydrocodone with acetaminophen is generally contraindicated in pregnancy and breastfeeding, and it should be avoided in combination with other CNS depressants due to additive CNS and respiratory depression. These products should not be used in patients with a Type 1 hypersensitivity reaction to codeine or morphine derivatives, nor in patients with a substance abuse history.[1,7,20,24,27,28]

With or without a DEA license, optometrists still ought to familiarize themselves with the pharmacologic agents available for pain treatment. No patient should needlessly suffer, so appropriate choices need to be carefully evaluated on an individual basis. With the

recent emphasis on the "opioid crisis," it is required of many optometrists with prescribing privileges for systemic medications to enroll in the prescription drug monitoring program (PDMP).[29] This free website covers several dozen states, and it lists every controlled substance, every practitioner that has written a prescription for a controlled substance and every pharmacy that has filled a prescription for a controlled substance. This valuable resource helps every medical practitioner determine what controlled substances a patient is taking, which is helpful in lessening the chance of drug interactions, as well as helps determine if a patient has a prescription drug use habit.

A practitioner's best plan is the old sports metaphor that says "the best offense is a good defense," where the offensive plan is to arm oneself with knowledge. In particular, this should include education regarding medication choices, legislative changes to controlled substances, and tools available to monitor an individual patient's use of controlled substances. Most patients do not present with drug-seeking behaviors; however, consistent education and vigilance by the optometric practitioner will ensure that appropriate choices are made for individual diagnoses and patients. In doing so, no patient suffers with acute ocular pain. ■



This patient's corneal burn developed following an incident with a curling iron. They were treated with a bandage contact lens and antibiotic steroids as well as proper oral threapy based on their reported pain severity.

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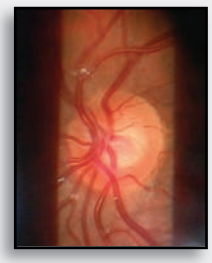

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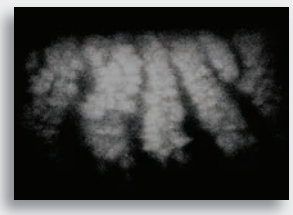

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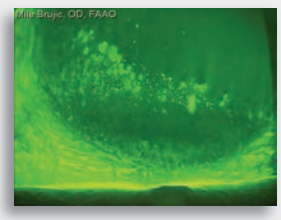

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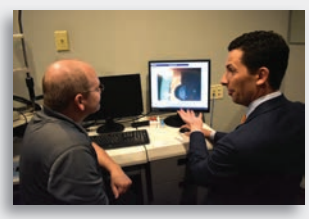

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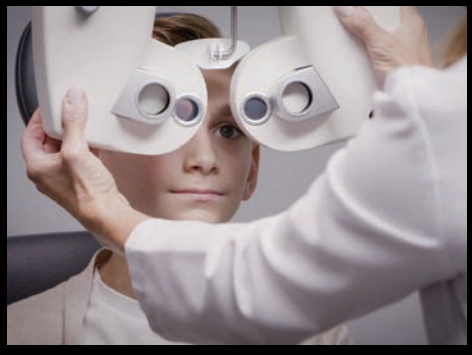

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Infiltrative Keratitis:

Fight the Battle for Corneal Clarity

When infections invade the cornea and leukocytes gain ground, you need skillful strategy and carefully chosen weaponry. **By Jeffrey R. Urness, OD, and Chad E. Gosnell, OD**

The human cornea is unique among our body's structures. It is designed for clarity, and its purpose is image formation, protection and beauty. Maintaining this corneal structure and function for a lifetime involves some impressive genetics, anatomy and physiology.

About 3,800,000 children are born in the United States each year, which means approximately 7,600,000 clear corneas begin their role to image a lifetime of vision.[1] Though nearly all US infants begin life with a clear "window" to the world, some will, along life's journey, be among the 30,000 to 70,000 US residents seen annually for microbial keratitis.[2-4] Though a precise number is not known, more people seek care for non-infectious keratitis (sterile keratitis).[3]

The healthy cornea consists of a tear-coated, five- to six-layer epithelial covering, an approximately 500-layer "cross-woven" collagen core, and a single layer of endothelium. Apart from the sparse population of resident keratocytes, dendritic cells and leukocytes, the stroma is collagen and water.

The ocular surface has a multimodal defense design. First and most used is the innate biomechanical protection provided by the orbit,

Release Date: September 15, 2019
Expiration Date: September 15, 2022
Estimated Time to Complete Activity: 2 hours

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Educational Objectives: After completing this activity, the participant should be better able to:

- Describe the underlying difference in origin between sterile and infectious infiltrates (and ulcers).
- Explain the importance and process of history-taking, as well as the signs and symptoms, to help characterize the two different corneal presentations.
- Describe the "usual suspects" that may cause sterile and infectious infiltrates (e.g. CLPU, CLARE, EKC, microbial keratitis, etc.).
- Determine when to pursue further diagnostic procedures, such as culturing, and/or appropriate referral.
- Recognize which therapeutic strategy to employ, and when to employ it.

Target Audience: This activity is intended for optometrists engaged in the care of patients with corneal infiltrates.

Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by the Postgraduate Institute for Medicine and Review Education Group. Postgraduate Institute for Medicine is jointly accredited by the Accreditation Council for Continuing Medical Education, the Accreditation Council for Pharmacy Education, and the American Nurses Credentialing Center, to provide continuing education for the healthcare team. Postgraduate Institute for Medicine is accredited by COPE to provide continuing education to optometrists.

Faculty/Editorial Board: Chad Gosnell, OD, Mann-Grandstaff VA Medical Center in Spokane, WA and Jeffrey Urness, OD, Mann-Granstaff VA Medical Center.

Credit Statement: This course is COPE approved for 2 hours of CE credit. Course ID is **64410-AS**. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

Disclosure Statements:

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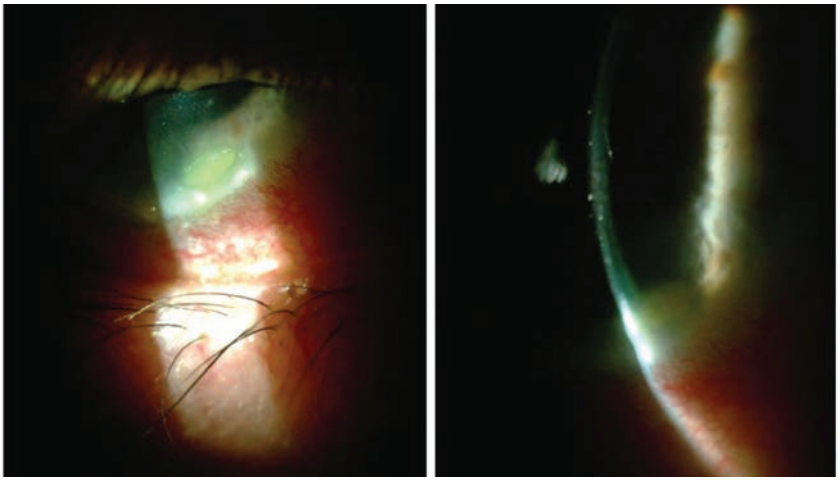


A 70-year-old male with Staphylococcal hypersensitivity marginal keratitis presented with a peripheral infiltrate (with fluorescein staining) and adjacent conjunctival hyperemia. The optic section shows minimal overlying epitheliopathy.

adnexal skin, eyelids, lashes, tear glands and tear film and conjunctiva and corneal epithelium. When healthy, these structures provide both static and dynamic biomechanical protection to the cornea and ocular surface.[5] When this protection is insufficient or merely needs support, the innate immune system is mobilized to neutralize offending agents, remove injured tissue and restore any disrupted anatomy.[6,7] Apoptosis and acute inflammation are two mechanisms of innate immunity serving to protect and restore ocular structure and function.[7]

The "first responders" of ocular innate immunity are local natural killer cells, macrophages and neutrophils, followed closely by dendritic cells.[8] When corneal insult occurs, the complement system is activated and cellular cytokines and chemokines are released to recruit and guide immune cells in the immune-privileged cornea.[9] The battle for corneal clarity ensues. This is the stage when leukocyte infiltration of the otherwise clear cornea begins.

When innate biomechanical and immune defenses need additional support, the third arm of protection is called to action: adaptive immunity. The macrophage and dendritic cells of the memory-less innate system serve to activate adaptive immunity. They bind foreign antigen, liberated by early invader destruction, and present them to local eye-related lymphoid tissue (lacrimal gland, conjunctiva and nasolacrimal mucosa), regional lymph nodes and, in some conditions, the spleen and thymus for generation of antibody-producing B-lymphocytes and priming of regulating T-lymphocytes, bringing both arms of adaptive immunity to the battle.[7,10,11]

Immune Privilege

Surrounding the visual pathway is anatomy and physiology that provide privileged, or "specialty," immune regulation to protect the visual system from potentially self-destructive activity, while defending against "outsider" invasion. Cellular tight junctions, blood-brain barrier and immune downregulation are a few features providing this specialized protection to the eye and visual pathway.[9] The design and organization of this protective system is greatly responsible for the varied clinical presentation seen in infiltrative keratitis.

Infectious infiltrative keratitis is defined as inflammation of the cornea due to attachment/invasion by viable microorganisms, while sterile infiltrative keratitis presents in corneal tissue devoid of live microorganisms. At times this distinction can be difficult to determine.[10]

Clinical appearance. The appearance of the cornea and surrounding structures yield valuable clues about the etiology and stage of disease. Larger, deeper, more central corneal infiltration with overlying epithelial destruction suggests microbial infection. The privilege of the central cornea and visual pathway provide greater opportunity for microbial attachment and invasion before the immune system "recognizes" the invasion.

In contrast, this paucity of central corneal immune cells supports tolerance to non-infectious forms of corneal insult. In the peripheral cornea and limbus reside numerous and diverse resident immune cells, which can respond quickly to local threats. In the case of microbes, innate immunity can quickly neutralize and

Sterile vs. Infectious Infiltrates[13]

Sterile	Infectious
• Smaller lesion (<1mm) • <2 clock-hours extent • More peripheral • Minimal epithelial damage (defect size compared with underlying infiltrate) • No mucous discharge • Less pain and photophobia • Little or no anterior chamber reaction • No lid involvement	• Larger lesion (>2mm) • >2 clock-hours extent • More central • Significant epithelial defect (size of staining defect closely mirrors size of underlying stromal lesion) • Mucopurulent discharge • Pain and photophobia • Anterior chamber reaction • Lid edema, tear film debris, hypopyon

eliminate invaders before they can become established. Non-infectious insult can trigger a rapid immune response, often generating characteristic peripheral corneal infiltration. For this reason, peripheral corneal infiltrates that are small, shallow, with minimal overlying epitheliopathy are more often sterile in etiology.

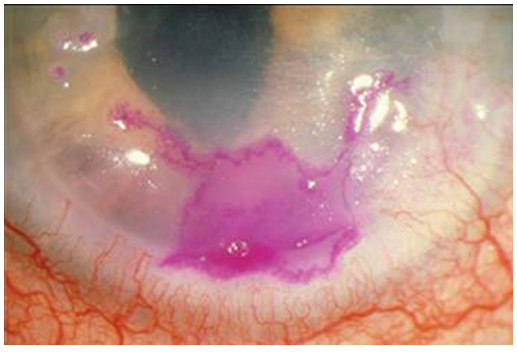
Empirical clinical exam guidelines, with slight variations, are well established to aid in determining whether infiltrative keratitis is sterile or infectious.[12]

Pertinent history. Sufficient history coupled with accurate observation is key to accurate assessment and effective treatment of infiltrative keratitis. The demographics and temporal descriptive history hold important clues to the absence or presence of infection.[12] For example, a red, painful eye that develops shortly after a week's vacation of hot tubbing supports suspicion for *Acanthamoeba* keratitis.

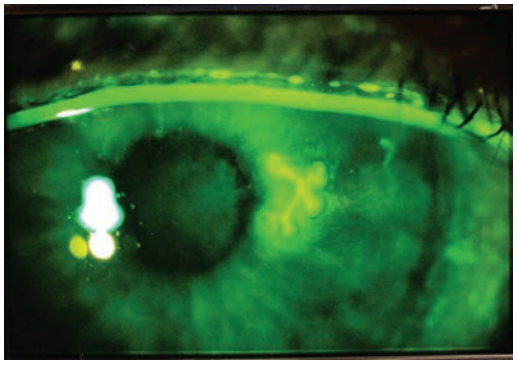
History of contact lens wear, pericorneal infections, recent eye trauma (including surgery), abnormal eyelid globe appositions, recent ocular exposure to contaminated water sources, concurrent head infection and relevant systemic conditions add to the risk and suspicion for infectious causes of infiltrative keratitis.

Detection of infection. Determining whether infection is likely is a high priority in the management of keratitis. The importance and nature of clinical workup and treatment differ based on any suspicion for infection, stage of disease and risk for visual compromise.

Historically and currently, culture and cytology are the most commonly employed ancillary tests used to improve detection of infectious etiologies; however, studies show that 38% of suspected corneal infections can be culture negative.[4,12,14,15] Though positive culture, cytology or both support the identification of a causal microbe, correlation with history, clinical findings and clinical course are needed to support a diagnosis of an infectious etiology.[12]



Herpes simplex geographic keratitis, with the corneal lesion highlighted by rose bengal dye. Note that the geographic lesion exhibits border dendriform branches.



Herpes simplex dendriform keratitis, with the corneal lesion highlighted with fluorescein dye.

Additional testing is sometimes employed to determine whether infection is present. This includes, but is not limited to, polymerase chain reaction (PCR), confocal microscopy and tissue biopsy.[12,16-18] Familiarity with techniques (e.g., corneal sampling and biopsies) and co-participating professionals (microbiologists processing culture samples) improve the clinical benefit of results (fewer false outcomes).

Sterile infiltrative keratitis develops from a variety of corneal insults. Three conditions commonly seen in the optometric practice include hypersensitivity marginal keratitis, contact lens-related peripheral ulceration (CLPU) and contact lens-associated red eye (CLARE)

Bacterial Hypersensitivity Marginal Keratitis

Also known as sterile corneal infiltrates, this is a common non-infectious unilateral or bilateral immune response to uncontrolled bacterial blepharitis.[19] Gram-positive *Staphylococcus aureus* is the most common offending species, although other bacteria have also been implicated.[20]

Signs and symptoms. Classically, this keratitis presents unilaterally or bilaterally with non-infectious infiltrates located at the 2, 4, 8 and 10 o'clock positions of the peripheral cornea.[10] These locations are fertile ground for this characteristic hypersensitivity immune response. First, the lid margins rest at these locations, promoting the highest concentration of bacterial exotoxins at these positions. Second, the close proximity of the limbal vasculature and eye-associated lymphoid tissue provides the resource for the immune response.

Eyelid-sourced bacterial exotoxins adhere to the corneal surface proteins, increasing corneal surface insult.[21] Host antibodies develop and bind bacterial antigen, producing a type III hypersensitivity immune reaction.[10] This signals the migration of polymorphonuclear leukocytes and fibroblasts to the site of the antigen-antibody complex. Once on site, these immune cells are positioned to protect the cornea from infection; however, their protective effort generates collagenase and proteoglycanase enzymes, producing collateral tissue damage and perpetuating cor-

neal infiltration that can lead to ulceration.[20]

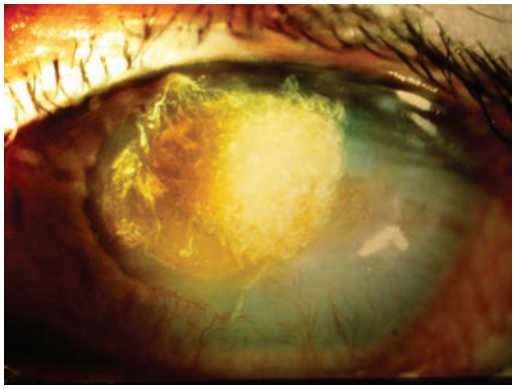
Typically, sterile marginal keratitis manifests with peripheral subepithelial infiltrates parallel the limbus and 1mm to 2mm from the corneoscleral junction. There is usually a corneal "clear zone" between the infiltration and the limbus. Infiltrates are characteristically circular and less than 2mm in diameter, but can coalesce into larger linear lesions.[10,21] Less often, the infiltrate becomes circumferential in form.[21,22]

Frequently, corneal infiltrates express variable fluorescein staining, usually smaller than the underlying infiltrate. Mild, fine keratic precipitates and anterior chamber cells can accompany this keratitis. Another corneal finding seen with Staphylococcal lid disease is punctate epithelial keratopathy.[23]

Correlated signs of bacterial lid disease usually accompany hypersensitivity marginal keratitis.[24] In some cases, facial signs of rosacea reveal the etiology, with blepharitis present in as many as 45% to 85% of rosacea patients.[10]

Symptoms from this sterile keratitis vary from mild foreign body sensation to mild pain. Patients often report light sensitivity, burning sensation, tearing, itch and excess mucin.[19,24] Vision reduction is not a common feature of this disease.

Treatment. In general, management of infiltrative keratitis consists of two goals. First, the population of causal pathogens must be reduced, kept in check and hopefully eliminated. Second, the inflammatory tissue response should be modified and guided to resolution, minimizing permanent tissue damage. Depending on the severity of blepharitis and related keratitis, treatment usually involves eyelid hygiene as well as antibiotic and anti-inflammatory agents.



A female patient in her late 20s developed *Candida* superinfection subsequent to culture-positive bacterial keratitis, which was initially responsive to a fortified topical antibiotic. Once the fungal infection was eliminated, the resulting scar required a corneal transplant, restoring acuity to 20/25.

During the acute phase, eyelid hygiene should be performed morning and evening. The application of warm, moist compresses prior to lid cleaning, with a commercially available cleanser, will soften tissue production and facilitate biofilm reduction with subsequent cleaning.

Antibiotic application should follow eyelid hygiene and be directed at the lid margins. A broad-spectrum antibiotic agent is ideal when empirically treating blepharitis-related marginal keratitis. Clinicians should note that the antibiotic vehicle—drop vs. ointment—is important. Ointments are preferred due to ease of application and increased duration of effective contact with the eyelids and surrounding tissue.

Commonly used broad-spectrum antibiotic ointments are bacitracin or erythromycin. Because bacitracin is available only in topical form, less bacterial resistance has developed against it—an advantage over erythromycin. Bacitracin has also been shown to be more effective against methicillin-resistant *Staphylococcus aureus*, as well as outperforming the only fluoroquinolone ophthalmic ointment, ciprofloxacin.[25-27] When you are confident the keratitis is sterile, the use of a topical corticosteroid will accelerate resolution of the keratitis and related symptoms.[28]

In severe and aggressive cases of marginal keratitis, clinicians should employ oral tetracyclines for their anti-inflammatory benefit, as well as their propensity to concentrate in the meibomian glands. Glandular concentration improves meibum quality while reducing pathogenic populations of offending bacteria.

Following antibiotic selection and dosing, anti-inflammatory treatment may be considered.[20] When corticosteroid treatment is anticipated, infiltrates with overlying epithelial defects should prompt consideration for diagnostic cytology and cultures.[10] If corticosteroids are instituted, their use should be preceded by effective broad-spectrum antibiotic therapy.[10] This treatment approach requires prescribing independent antibiotic and corticosteroid agents. A mild steroid eyedrop such as fluorometholone or even a more effective steroid, such as loteprednol or prednisolone, may be selected depending on the severity of the inflammatory response.

Until the corneal epithelial defects are resolved and the keratitis is determined to be sterile, corticosteroid use should be "covered" by ongoing antibiotic treatment. If iritis is present, cycloplegia can add to symptomatic relief.

Another medication strategy widely used employs combination ointments (antibiotic + steroid). It is our position that this choice should be reserved for keratitis with intact epithelium and anticipated treatment course of seven to 10 days.

Some patients with staphylococcal hypersensitivity marginal keratitis should be followed every two to seven days until resolution is complete.[19] With appropriate treatment

and compliance, prognosis is good and improvement usually swift. Recurrences can be common and, as much as possible, patients and caregivers should understand the important role of eyelid hygiene in reducing future episodes.

Once the acute keratitis is resolved, expressive meibomian gland treatments may be beneficial for optimal control of causative eyelid disease. Low-dose maintenance therapy of oral doxycycline may be helpful, and can be used for months to years to reduce recurrences.[10,19,20] If antibiotic ointments are chosen for long-term prophylaxis, consider monthly pulse treatments or cycling between different ointments to minimize resistance.

Contact Lens Peripheral Ulcer

CLPU is a common finding in hydrogel contact lens wearers. CLPU occurs at a rate of 7% per patient-year and is primarily associated with extended-wear contact lenses.[29,30]

CLPU is defined as an inflammatory reaction of the cornea to include corneal epithelial defect, infiltration, and necrosis of the anterior stroma.[31] The pathogenesis is not well understood; however, it is thought the corneal infiltration is an adverse response to the *Staphylococci* species in the presence of contact lenses and epithelial defects. The corneal tissue is not infected with an organism, but rather is undergoing a sterile inflammatory response similar to that seen in marginal keratitis. A study in rabbit models showed that it is the corneal epithelial break that may allow the entry of bacterial exotoxins to the stroma and induce the infiltrative response.[32]

Signs and symptoms. The conjunctiva is typically moderately hyperemic in the sector of the ulcer(s), though diffuse injection is common. Circular focal excavation with underlying infiltration and anterior stromal necrosis of the anterior stroma are located mid-peripheral to peripheral of cornea.[31] Bowman's layer is not breached, in contrast to microbial keratitis.[29] CPLUs consistently stain with fluorescein. Patients experience moderate to severe pain or can be asymptomatic.

Treatment. This includes discontinuation of contact lens wear and the initiation of antimicrobial therapy until the corneal epithelium recovers from the ulcer. Typically, a fourth-generation fluroquinolone is used until re-epithelialization occurs.

As with marginal keratitis, once positive response to treatment is established, consider the addition of topical corticosteroids to reduce the inflammation and minimize scarring. Reduced epithelial defect size with smooth regular epithelial borders is an early finding with effective treatment.

If the infiltrate worsens or does not respond to treatment, further testing should include culturing, a second opinion or both.

What We've Learned From the SCUT Study

Historically, the use of steroids in the treatment of infectious keratitis has been incorporated using "clinical wisdom." More recently, research has been exploring the risks, benefits and optimal guidelines for treatment with ophthalmic corticosteroids. The recent Steroids for Corneal Ulcers Trial (SCUT) has often been cited in support of the safety of topical steroids in the treatment of confirmed drug-sensitive bacterial keratitis.[12]

For this study, empirical antibacterial treatment was initiated but a subsequent topical steroid was not instituted until culturing confirmed the presence of infectious bacteria and 48 hours of microbe-sensitive antibiosis had been delivered. The SCUT study supported the safety of topical steroid use when independent, non-combination antibacterial and corticosteroids were sequentially deployed in culture-positive drug-sensitive bacterial keratitis.[12]

If infection is believed to be present, yet the organism and its sensitivity have not been discovered, delay the use of steroids until empirical treatment appears effective and will eliminate the offending bacteria. Loading doses plus 24 hours of antibacterial medication will provide valuable information for the decision of corticosteroid use.

One published study indicates up to 30% of infectious keratitis cases are polymicrobial.[43] Polymicrobial infections are not exclusively bacterial. Clinical experience as well as research implicate greater challenge to treatment of polymicrobial infectious keratitis.[43] Such cases often require poly-pharmaceutical intervention using fortified antibiotics. Because the SCUT study enrolled only cases of monomicrobial infection, it cannot be generalized to polymicrobial cases.[12]

Contact Lens-Induced Acute Red Eye

This is a common non-infectious infiltration of the cornea and inflammation of the conjunctiva, occurring at a rate of 11% per patient-year.[29]

CLARE is defined as an inflammatory reaction of the cornea and conjunctiva following eye closure.[31] Typically, this occurs acutely following sleeping or napping in contact lenses. Risk factors for CLARE include extended wear contact lenses, high water contact lenses, tight fitting contact lenses, and recent upper respiratory infection.[30]

Signs and symptoms. Patients experience irritation to moderate pain, tearing and photophobia. Small, diffuse, mid-peripheral to peripheral corneal infiltrates will appear. No corneal fluorescein staining of the infiltrates is seen. There is moderate to severe circumferential redness.[31]

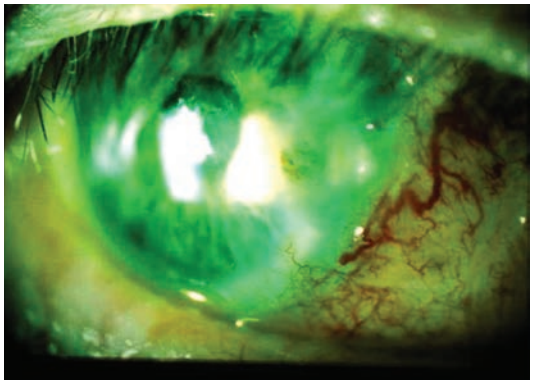
The pathogenesis of CLARE is similar to CLPU with no infected corneal tissue, but an inflammatory response to bacterial toxins. Additionally, there has been association between gram-negative bacteria and

CLARE, including *Haemophilus influenzae* and *Haemophilus parainfluenzae*.[30]

Treatment. CLARE will self-resolve with discontinuation of contact lenses. In most cases, treatment includes not only discontinuation of contact lens wear but also copious lavage with artificial tears. In more severe cases, consider topical steroids four to five times per day with taper according to clinical course.[31]

The prognosis of CLARE is excellent. Scarring is rare. The infiltrates typically resolve in seven to 14 days without affecting visual acuity.[31]

When history and clinical features fail to convince the clinician that infiltrative keratitis is sterile, the search for an infectious etiology is necessary. There are four microbe classes that cause infectious keratitis: bacteria, virus, fungus and parasite.



After a pterygiectomy with graft, a male patient in his 70s failed to use his medications and ocular lubricants. By one week post-op, he had developed descemetocele with adjacent infiltrates and positive Seidel sign. Corneal culture was positive for *Staphylococcus*. Fortified antibiotics cleared the infection while a full-thickness button graft repaired the descemetocele.

Viral Keratitis

Viral infection is an intracellular invasion. The target tissue of attack and defense is cellular, producing infiltration of epithelium and/or endothelium along with adjacent stroma. The influence of immune privilege predisposes the central cornea to viral infectious infiltration (e.g., adenovirus and herpes virus).[9] Though viral keratitis more often manifests in the central cornea, exception to such infection is seen in limbal keratoconjunctivitis.

Herpes simplex virus (HSV) is the leading cause of infectious monocular blindness.[33] Currently, there is an upward trend of recurrent and new cases with approximately 60,000 active cases annually in the United States, resulting in nearly 1,000 corneal transplants each year.[33]

Dendriform herpetic keratitis is often readily diagnosed from appearance of epitheliopathy and infiltration; however, some hosts manifest non-dendriform disease. Geographic and limbal HSV keratitis are less common forms of disease that can be uncovered with corneal sensitivity and vital dye testing.

Correlating reports of pain with vital dye staining and corneal sensitivity testing can be helpful in suspected HSV keratitis. We have found off-label use of dental floss to be a good material for testing corneal and conjunctival sensation. If sterility is a concern, suture material is equally effective. Initial onset and early recurrence of HSV keratitis more often produces acute distressful pain.[34] Damage to the sensory nerves with successive episodes tends to change and reduce the report of pain.

If HSV is suspected, but not evident based on history and appearance, ancillary studies (including culture, PCR and confocal imaging) can be effective.[18]

Current treatment of active corneal herpes simplex infection involves antiviral medications either in topical or oral form and, in some cases, cycloplegia and topical anti-inflammatory medications. Though still used today, topical trifluridine is less often the drug of choice, giving way to newer and less toxic topical ganciclovir or oral antivirals (acyclovir, famciclovir or valacyclovir).[23,34] When stromal disease presents, topical corticosteroids in addition to antiviral medication are indicated.[33,36]

In select cases of central corneal disease, epithelial debridement of viral-laden cells is indicated to accelerate removal of the immune stimulus. Following debridement, bandage contact lens and amniotic membranes in recalcitrant cases can be applied to facilitate re-epithelialization.[35]

The Herpetic Eye Disease Study (HEDS) found oral acyclovir treatment of epithelial keratitis to be equally effective as topical therapy, providing another option to minimize unintended effects and optimize compliance. (In patients with renal failure, topical therapy should be considered first.) The HEDS investigators found significant reduction in recurrence of both epithelial and stromal disease with oral antiviral prophylaxis; however, there was no reduction in the conversion from epithelial disease to the sight-threatening stromal form of the keratitis. The recurrence of stromal disease was reduced by about 50% over the year following prophylaxis with 400mg of oral acyclovir two times daily.[33]

Fungal Keratitis

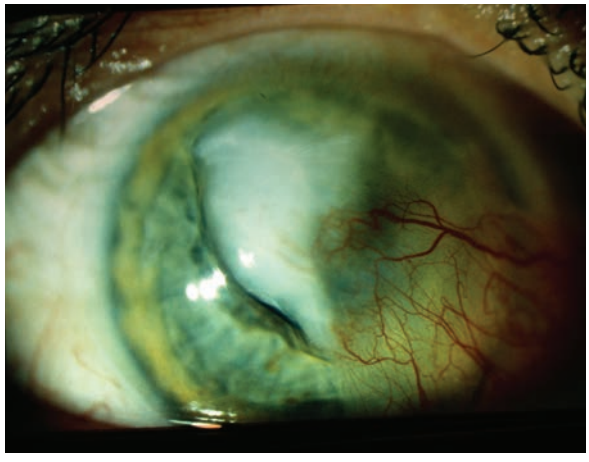
In the western world, fungal keratitis is an infrequent intercellular opportunistic invasion of the cornea associated with contact lens wear, immune suppression and vegetative trauma.[37] The incidence in the United States is uncertain, but is likely less than 2% of infectious keratitis cases, or about 30,000 cases/year.[37,38]

The most frequently detected fungi in keratitis are *Fusarium*, *Aspergillus* and *Candida* species.[37] The prognosis for fungal keratitis is generally poorer than for other causative microbes, with perforation as high 50%.[39]

Biomechanical defense and innate immunity are the primary defenses against fungal keratitis.[37] Macrophages and neutrophils are the early and predominant leukocyte responders to fungal invasion. Yield in fungal culture is low, so cytologic stains, confocal scanning and tissue biopsy can be valuable diagnostic adjuncts.[37]

In contrast to bacterial keratitis, fungal corneal infiltrates often have irregular feathery margins with satellite lesions. Early fungal infiltration often thickens the stroma and elevates compromised epithelium.[37]

Fungal keratitis frequently generates pain that is disproportionate to the acute inflammatory features, similar to many cases of *Acanthamoeba* infection.[37] Contact lens wear, organic tissue injury and immune suppression/modulation are predisposing factors for fungal keratitis.[37] Immune suppression not only adds risk for fungal infection, it facilitates bacterial and viral co-infection and complicates effective treatment.



A large central corneal infiltrate/ulcer with adjacent pre-existing pterygium, aggravated by culture-positive *Streptococcus* infection.

From our experience, fungal keratitis frequently exhibits a gradual buildup of acute inflammatory signs, with corneal infiltration being a predominant finding. Fibrinous iritis and hypopyon are less common in early fungal keratitis.[40]

Infiltration from fungal infection requires access to the inter and subepithelial space, yet can progress beneath re-established epithelium.[41] This re-epithelialization can impair drug penetration, thereby impeding sterilization of corneal tissues. Topical natamycin and amphotericin B are drugs often used in treatment of fungal keratitis. Natamycin remains the only commercially available ophthalmic drug FDA approved for treatment of fungal keratitis.[42] Neither medication readily penetrates intact epithelium, requiring periodic epithelial debridement to maintain therapeutic drug levels at the site of infection. The dualistic fungal life forms (spore and hyphae) add to the difficulty of *in vivo* eradication, requiring longer treatment periods and often epithelial debridement to clear infection.

The toxicity of topical and systemic antifungal medications, along with extended course of drug delivery and corneal debridement, has historically prompted clinicians to have "definitive" cultures, cytology and/or biopsy before committing patients to the extended treatment needed to eradicate fungal keratitis. Recent studies looking at best practices for fungal keratitis management show that topical natamycin remains the most effective ophthalmic antifungal drug with the fewest adverse effects.[37] Corneal crosslinking has yet to demonstrate added benefit to traditional pharmaceutical intervention.[37,42]

Bacterial Keratitis

Nearly all cases of infiltrative bacterial keratitis exhibit corneal epithelial compromise often exploited by contaminated contact lenses/solutions, eyelid disease, immunosuppression or prior ocular surgery, which can lead to infection.[36] *Staphylococcus*, *Pseudomonas* and *Streptococcus* species remain the most frequently isolated bacteria.[12]

Once again, structure and function of ocular defenses increase the likelihood that larger and more central infiltrates have infectious etiology. Immune privilege, having its greatest impact farthest from the limbal cornea, gives greater opportunity for bacteria to exploit more central surface trauma, attaching, invading and populating central corneal tissue.

Subsequent immune signaling must call in surrounding resident leukocytes from lacrimal glands and eye-associated lymphoid tissue. The more intense and widespread the inflammatory symptoms and signs, the more likely bacterial infection is at play in infiltrative keratitis. Features of acute bacterial keratitis often include fast onset infiltrates 2mm in size with equal or larger overlying epithelial defects, greater than two clock-hours of extent involving mid-to-deep stroma with thinning and stromal necrosis.[12] Hypopyon and fibrinous iritis should elevate suspicion for bacterial infection.

When faced with the possibility of bacterial keratitis, you must decide whether to pursue empirical treatment alone or ancillary test-guided intervention.

Empirical treatment. The advent of commercially available, broad-spectrum, highly effective topical fluroquinolones in the 1990s increased the practice of empirical treatment

by non-corneal specialists.[12] Patient history coupled with signs of sterile or mild infectious infiltrative keratitis often supports the decision for broad-spectrum topical fluoroquinolone treatment.

Test-guided treatment. When history and clinical presentation/course implicate moderate to high risk for sight loss, culture and cytology-guided management should be pursued.[12] Culturing can be obtained using transport media or direct plating to culture media. The common transport media are Amies agar gel or modified Stuart's medium.[12] When "direct plating," the most common media are blood, chocolate and Sabouraud agars.[12] The clinician should sample eyelids along with the ulcerative infiltrative lesions. When there is history of contact lens wear, then lenses, cases and solutions should also be cultured.

If the initial treatment was empirical and clinical course appears unresponsive after 48 to 72 hours, discontinue empirical antibiotics for 24 hours and culture or refer to a corneal specialist before resuming anti-infective treatment.[12]

Depending on the location, extent and stage of corneal infiltration, debulking all necrotic epithelium, stroma and infiltration can improve efforts to sterilize the cornea and re-establish its structure. When debulking, be careful to avoid perforation. Prior to and following corneal sampling, the size of the corneal infiltrate(s) and any epithelial defect should be documented by diagram or imaging.

Managing keratitis can be straightforward, but should not be considered simple. The better the optometrist's understanding of the various causations and impressively sophisticated immune responses, the greater the opportunity for rapid resolution of keratitis with minimal structural and functional damage.

Treatment of infiltrative keratitis often involves anti-infectives, anti-inflammatories and support therapies. The strength and dosing of needed medications should be employed with cautious confidence—confidence that comes from knowledge and experience. The use of anti-inflammatory and immune-suppressing corticosteroids is often beneficial, and frequently necessary, to achieve resolution of infiltrative keratitis. However, used indiscriminately or inappropriately, corticosteroid therapy can lead to poor outcomes with avoidable sight loss.

Knowing what medications to use at what strength for how long depends on the clinician's understanding of the cause of the keratitis, its stage and the risk for permanent damage and sight loss. With this understanding, there is no need to surrender care when infiltrative keratitis comes to do battle. ■

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OSC QUIZ

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You must achieve a score of 70 or higher to receive credit. Allow four weeks for processing. For each Optometric Study Center course you pass, you earn 2 hours of credit from Pennsylvania College of Optometry.

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

1. Annually, how many US residents seek care for microbial keratitis?
a. 10,000 to 20,000.
b. 30,000 to 70,000.
c. 70,000 to 100,000.
d. 100,000 to 120,000.

2. The "first responders" of ocular innate immunity include which of the following?
a. Natural killer cells.
b. Macrophages.
c. Neutrophils.
d. All the above.

3. Infectious infiltrative keratitis is defined as:
a. Inflammation of the cornea without invasion by a viable microorganism.
b. Inflammation of the cornea with or without invasion by a viable microorganism.
c. Inflammation of the cornea with invasion by a viable microorganism.
d. None of the above.

4. Sterile infiltrative keratitis is defined as:
a. Inflammation of the cornea without invasion by a viable microorganism.
b. Inflammation of the cornea with or without invasion by a viable microorganism.
c. Inflammation of the cornea with invasion by a viable microorganism.
d. None of the above.

5. Studies show ___ of suspected corneal infections can be culture negative.
a. 25%.
b. 38%.
c. 42%.
d. 50%.

6. What is the most common bacterial species involved in bacterial hypersensitivity marginal keratitis?
a. *Pseudomonas aeruginosa*.
b. *Acinetobacter baumannii*.
c. *Streptococcus pyogenes*.
d. *Staphylococcus aureus*.

7. Which type of hypersensitivity reaction is responsible for bacterial hypersensitivity marginal keratitis?
a. Type I (immediate hypersensitivity).
b. Type II (cytotoxic reaction).
c. Type III (immune complex reaction).
d. Type IV (cell-mediated).

8. Treatment for bacterial hypersensitivity marginal keratitis usually involves:
a. Eyelid hygiene.
b. Broad-spectrum antibiotic therapy.
c. Anti-inflammatory therapy.
d. All the above.

9. CLPUs occur at what rate per patient-year?
a. 5%.
b. 7%.
c. 9%.
d. 11%.

10. The anterior corneal appearance with a CLPU is most consistent with:
a. Bowman's layer is breached and consistently stains with fluorescein.
b. Bowman's layer is breached and does not consistently stain with fluorescein.
c. Bowman's layer is not breached and consistently stains with fluorescein.
d. Bowman's layer is not breached and does not consistently stain with fluorescein.

11. CLARE occurs at what rate per patient-year?
a. 5%.
b. 7%.
c. 9%.
d. 11%.

12. The risks for CLARE include all the following, *except*:
a. Low water contact lenses.
b. Tight fitting contact lenses.
c. Recent upper respiratory infection.
d. Extended wear contact lenses.

13. The leading cause of infectious monocular blindness is:
a. Bacterial keratitis.
b. Fungal keratitis.
c. *Acanthamoeba* keratitis.
d. Herpes simplex keratitis.

14. The HEDS showed that:
a. Oral acyclovir treatment of herpetic epithelial keratitis is less effective than topical therapy.
b. Oral acyclovir treatment of herpetic epithelial keratitis is equally effective as topical therapy.
c. Oral acyclovir treatment of herpetic epithelial keratitis is more effective than topical therapy.
d. Both oral acyclovir treatment and topical therapy should be used for the treatment of herpetic keratitis.

15. Fungal keratitis is associated with all the following, *except*:
a. Vegetative trauma.
b. Swimming pools.
c. Contact lens wear.
d. Immune suppression.

16. In fungal keratitis, the most frequently detected fungi include all of the following, *except*:
a. *Aspergillus*.
b. *Candida*.
c. *Cryptococcus*.
d. *Fusarium*.

17. Treatment for fungal keratitis includes:
a. Natamycin.
b. Amphotericin B.
c. Fluoroquinolones.
d. Both a and b.

18. The most frequently isolated bacterial species in infiltrative bacterial keratitis include all the following, *except:*
a. *Pseudomonas*.
b. *Staphylococcus*.
c. *Shigella*.
d. *Streptococcus*.

19. Clinical features of infiltrative bacterial keratitis include all the following, *except*:
a. Slow onset of infiltrate.
b. 2mm and larger infiltrate.
c. Hypopyon.
d. Stromal necrosis.

20. The SCUT showed:
a. Topical steroids should never be used in the treatment of bacterial keratitis.
b. Topical steroids should be used at the initial presentation of bacterial keratitis.
c. Topical steroids can be safely used in the treatment of confirmed drug-sensitive bacterial keratitis.
d. Topical steroids can be used without concurrent antimicrobial therapy.

Examination Answer Sheet

Infiltrative Keratitis: Fight the Battle for Corneal Clarity

Valid for credit through September 15, 2022

Online: This exam can be taken online at www.reviewsce.com. Upon passing the exam, you can view your results immediately and download a real-time CE certificate. You can also view your test history at any time from the website.

Directions: Select one answer for each question in the exam and completely darken the appropriate circle. A minimum score of 70% is required to earn credit.

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Payment: Remit $35 with this exam. Make check payable to Jobson Healthcare Information, LLC.

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Salus University has sponsored the review and approval of this activity.

Processing: There is a four-week processing time for this exam.

Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ
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18. Ⓐ Ⓑ Ⓒ Ⓓ
19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

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

21. Describe the underlying difference in origin between sterile and infectious infiltrates (and ulcers). ① ② ③ ④ ⑤

22. Explain the importance and process of history-taking, as well as the signs and symptoms, to help characterize the two different corneal presentations. ① ② ③ ④ ⑤

23. Describe the "usual suspects" that may cause sterile and infectious infiltrates (e.g. CLPU, CLARE, EKC, microbial keratitis, etc.). ① ② ③ ④ ⑤

24. Determine when to pursue further diagnostic procedures, such as culturing, and/or appropriate referral. ① ② ③ ④ ⑤

25. Recognize which therapeutic strategy to employ, and when to employ it. ① ② ③ ④ ⑤

26. Based upon your participation in this activity, do you intend to change your practice behavior? (choose only one of the following options)
Ⓐ I do plan to implement changes in my practice based on the information presented.
Ⓑ My current practice has been reinforced by the information presented.
Ⓒ I need more information before I will change my practice.

27. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ☐

28. If you plan to change your practice behavior, what type of changes do you plan to implement? (check all that apply)

ⓐ Apply latest guidelines ⓑ Change in pharmaceutical therapy ⓒ Choice of treatment/management approach ⓓ Change in current practice for referral ⓔ Change in non-pharmaceutical therapy ⓕ Change in differential diagnosis ⓖ Change in diagnostic testing ⓗ Other, please specify: _____

29. How confident are you that you will be able to make your intended changes?

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

30. Which of the following do you anticipate will be the primary barrier to implementing these changes?
ⓐ Formulary restrictions
ⓑ Time constraints
ⓒ System constraints
ⓓ Insurance/financial issues
ⓔ Lack of interprofessional team support
ⓕ Treatment related adverse events
ⓖ Patient adherence/compliance
ⓗ Other, please specify:

31. Additional comments on this course:

Please retain a copy for your records. Please print clearly.

First Name
Last Name
E-Mail

The following is your: ☐ Home Address ☐ Business Address

Business Name
Address
City State
ZIP
Telephone # - -
Fax # - -

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

32. The content was evidence-based. ① ② ③ ④ ⑤

33. The content was balanced and free of bias. ① ② ③ ④ ⑤

34. The presentation was clear and effective. ① ② ③ ④ ⑤

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

Signature _____ Date _____

Lesson 118657 RO-OSC-0919

The Risk-return Tradeoff

The pros of vaccination usually always outweigh the cons.

Edited by Joseph P. Shovlin, OD

Q I have a patient who had shingles 12 months ago. She still requires topical steroids and oral antivirals for persistent keratouveitis and is currently managed with one drop of topical steroid daily. She has inquired about receiving the new vaccine for shingles, Shingrix (GlaxoSmithKline). Is there any concern about reinvigorating her immune-mediated herpes zoster ophthalmicus (HZO)?

A "Before getting any vaccine, one must understand the reason to get it," says Paymaun Asnaashari, OD, who practices in northern California. He notes that the purpose of vaccines is to reduce or prevent the risk of acquiring an illness, such as shingles. Nearly one out of three people in the United States develops shingles in their lifetime, and roughly one million Americans are affected by it each year alone.[1] Dr. Asnaashari adds that the infection is serious and can be very painful and debilitating, leaving some house-ridden.

Two Forms of Protection

Shingles vaccines are the only way to protect yourself from the infection.[1] To prevent shingles and its complications, the CDC recommends adults older than 50 receive two doses of Shingrix two to six months apart.[1] These two doses are effective nearly 90% of the time throughout the first four years after they're administered.[2]

Zostavax (Merck) is still used to ward off shingles in adults older than 60, but Shingrix is the preferred method of vaccination.[1] Compared with Zostavax, Shingrix reduces the risk of shingles by almost double, is more efficacious and remains effective for longer.[2] While Zostavax contains a live but weakened virus, Shingrix contains an inactivate virus and combines a non-live antigen with a specifically designed adjuvant to stimulate an enhanced response by the body's immune system.[3] Caution is advised with Zostavax in certain immunocompromised patients, especially those on high-dose corticosteroids, as the vaccine can be contraindicated.[4] It contains a higher concentration of the live attenuated varicella vaccine, and the adult version is up to 14 times stronger than the version for children.[4]

The Risk of Reinvigorating

Despite the promise of reduced risk of sickness, patients may still face obstacles when considering vaccination.[5] The same applies to shingles and its sequelae. According to Dr. Asnaashari, misconceptions about the infection still prevail, its seriousness is not understood and there are mixed messages about age. He notes, however, that the general consensus about shingles vaccination is clear—it is highly encouraged.

Dr. Asnaashari says patients who have a history of shingles and require chronic topical steroids and oral antivirals are encouraged to get vaccinated as long as they are good candidates. He explains that Shingrix provides better immunity and protection against future shingles outbreaks by stimulating the production of specific immune memory cells. Because it is not a live virus, he notes that the risk of reinvigorating someone with a history of shingles is slim to none and encourages vaccination with Shingrix in this patient. He adds that Shingrix may also be a good option for high-risk immunocompromised patients who are not good candidates for Zostavax.

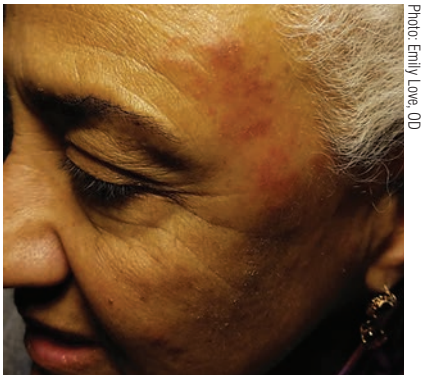

Photo: Emily Love, OD

As seen by her red skin irritation, this 71-year-old patient has HZO.

Nonetheless, when it comes to the unrelenting threats that today's environment poses to our health, getting vaccinated is important, especially for those who have a history of the illness in question. ■

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Beyond The Pale

The ophthalmic manifestations of Waardenburg syndrome may be striking—making the diagnosis easy. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

It's not often that you see heterochromia iridis, but when you do, you should also be on the lookout for depigmented hair, a displaced medial canthus and even congenital hearing loss. That's because heterochromia iridis—along with these other findings—is a classic sign of a rare systemic condition, Waardenburg syndrome (WS).

This hereditary group of conditions, first described in 1951, is rare, affecting an estimated one in 40,000 people, and accounts for 2% to 5% of all cases of congenital hearing loss.[1-3] It may be inherited in an autosomal dominant (most commonly) or autosomal recessive manner, although signs and symptoms often vary between and even within families. Common features include congenital sensorineural deafness; pale blue eyes, different colored eyes, or two colors within one eye; a white forelock (hair just above the forehead); or early graying of scalp hair before age 30. Various other systemic signs may also be present, from chronic constipation to slightly decreased intellectual functioning.[1,2]

While WS requires a thorough case history to confirm the diagnosis, the ocular features are easily recognizable to the OD, providing a clue to the underlying diagnosis.

The Choroid

Research shows mutations in at least six different genes cause WS, including EDN3, EDNRB, MITF, PAX3, SNAI2 and SOX10.[2-4] All of these genes are involved in the development of several cell types, including pigment-producing melanocytes.[2-4]

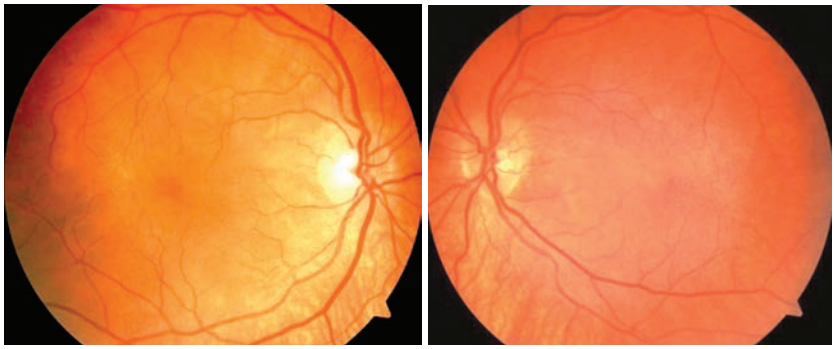

Fig. 1. The patient's dilated fundus exam revealed a post-equatorial hypopigmented choroid as well as choroidal folds in the right and left eyes.

In the eye, melanocytes are responsible for the pigment in the choroid and the stroma of the iris and ciliary body. Uveal melanocytes are developed from the neural crest, the same origin as the melanocytes in skin and hair.[5]

In addition to iris pigmentary abnormalities, careful inspection can reveal posterior uveal pigmentary anomalies. One study observed broad areas of choroidal hypopigmentation with WS, and foveal OCT revealed that the hypopigmented choroid was slightly (19%) thinner compared with the normal opposite subfoveal counterpart.[5]

Diagnosis and Management

WS is classified into four subtypes based on the presence of certain features and the genetic cause, with types I and II being the most common.[2-4] In 1992, the Waardenburg Consortium proposed diagnostic standards, which include both major and minor criteria. A diagnosis of type I must meet two major, or one major and two minor criteria:[2,4,6]

Major criteria:

- Congenital sensorineural hearing loss
- Iris pigmentary abnormality, such as heterochromia iridis (complete, partial or segmental); pale blue eyes (isohypochromia iridis); or pigmentary abnormalities of the fundus (choroid)
- Abnormalities of hair pigmentation, such as white forelock, or loss of hair color
- Dystopia canthorum—lateral displacement of inner canthi (in types I and III only)
- A first degree relative with WS

Minor criteria:

- Leukoderma (white patches of skin) present from birth
- Synophrys (connected eyebrows) or medial eyebrow flare
- Broad or high nasal bridge
- Hypoplasia of the nostrils
- Premature gray hair (younger than age 30)

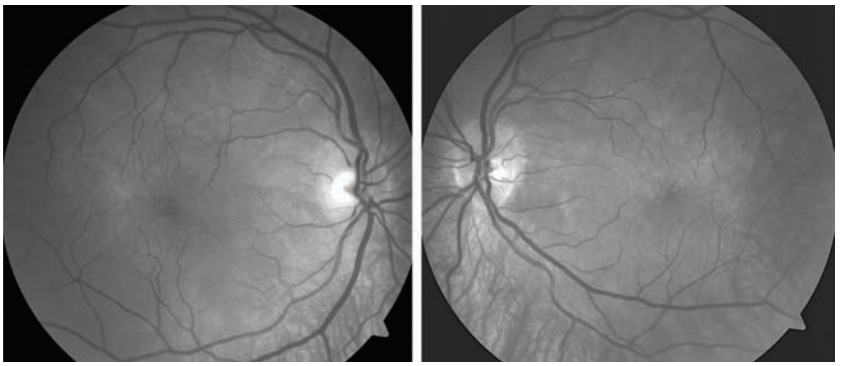

Fig. 2. Red-free images show broad areas of choroidal hypopigmentation, most prominent in the area inferior to each optic disc.

WS type II has features similar to type I, but with no signs of dystopia canthorum. Type III is additionally characterized by musculoskeletal abnormalities such as muscle hypoplasia; flexion contractures (inability to straighten joints); or syndactyly (webbed or fused fingers or toes). Type IV has similar features to type II, but with Hirschsprung disease (a condition resulting from missing nerve cells in the muscles of part or all of the large intestine).[2,4,6]

Because no specific treatment exists for this condition, management is directed toward specific symptoms and signs. Optimal care often requires the coordinated efforts of a team of medical professionals, such as optometrists, dermatologists, hearing specialists, orthopedists and gastroenterologists. Patients with chronic constipation are often prescribed special diets and medications to promote gastrointestinal health. All patients with WS require close monitoring for hearing loss and an annual comprehensive ophthalmic workup.[2,4,6,7]

Case Report

By Jeanne I. Ruff, OD

A 43-year-old Caucasian female presented with complaints of near blur and a history of WS type II. Her entering corrected distance acuities measured 20/20-2 OD and OS. Manifest refraction was +2.50 -1.50 003 OD and +2.50 -0.75 060 OS. She had a plus build-up of +1.50D OU, and her near visual acuities were 20/20 OU.

Biomicroscopy revealed iris herterochromia OD and bilateral iris transillumination defects, greater in the OS. No pigment cells on the corneal endothelium were observed in either eye.

Dilated funduscopy was remarkable for a subtle, wave-like appearance of the posterior pole OS > OD, consistent with choroidal folds.

Fundus photography documented a post-equatorial hypopigmented choroid in the right and left eyes (*Figures 1 and 2*). OCT demonstrated normal retinas and slight thinning of choroidal tissue in both eyes, along with subtle choroidal folds (*Figure 3*). Frequency doubling technology perimetry was normal OD, but revealed central deficits with some foveal sparing OS.

We relayed these findings to the patient and her primary care provider and prescribed a reading addition of +1.50 D OU to alleviate the near blur. She was scheduled for ocular echography to rule out other causes of choroidal folds, which are most likely due to the hyperopia. She was also educated on the need for semi-annual follow-up. ■

Dr. Ruff earned her Doctor of Optometry from Nova Southeastern University. She practices in the Williamsburg, VA area, with an emphasis on full-scope care and advanced diagnostic technology.

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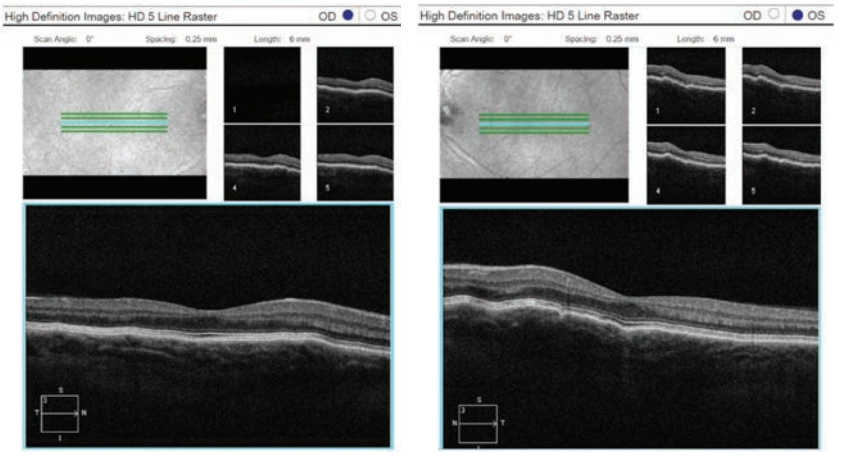

Fig. 3. OCT 5-line raster scans of both eyes demonstrated a normal retina, but slight thinning of choroidal tissue and subtle choroidal folds.

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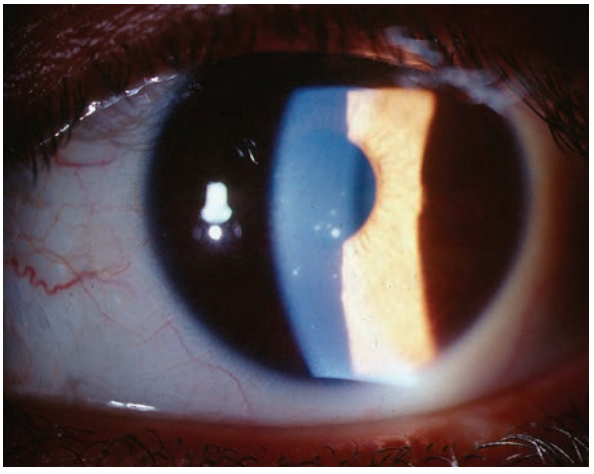
Pressure By Any Other Name

Know when elevated IOP is due to a glaucomatocyclitic crisis.

By Joseph W. Sowka, OD

A 66-year-old male presented urgently with mildly blurred vision and a "pressure feeling" in his right eye. It began on Saturday and he waited until Monday to come in as, he said, it "wasn't that bad." He wore no visual correction, but pinhole acuity was 20/30 in the right eye. He had longstanding vision decrease in his left eye from an old macular scar.



This patient demonstrates centrally located keratic precipitates in glaucomatocyclitic crisis.

He had a mild injection of his right bulbar conjunctiva. There was, perhaps, a mild bit of corneal edema in his right eye with a few centrally located endothelial keratic precipitates and a rare anterior chamber cell floating freely. His intraocular pressures (IOPs) were 50mm Hg OD and 18mm Hg OS. Years earlier, he had undergone laser iridotomy in each eye for an anatomically narrow angle, which had subsequently opened. Gonioscopy was deferred on this visit.

While the symptomatically elevated IOP might be a diagnostic dilemma, in this case the etiology was apparent, especially in light of the fact that this was his third episode within the past year. Even the patient himself understood the diagnosis before coming in: he was having a recurrence of glaucomatocyclitic crisis (GCC).

Defining GCC

The condition, also known as Posner-Schlossman syndrome, is characterized by these features:

1. Elevated IOP appearing nearly simultaneously with inflammatory cells in the anterior chamber followed by between one and 20 unpigmented keratic precipitates on the corneal endothelium. Posterior synechiae are never formed.
2. An eye that is white or that has minimal dilation of the conjunctival vessels without ciliary flush.
3. The anterior chamber angles are open gonioscopically.
4. The condition is unilateral (although, rarely bilateral cases are possible).
5. The presenting symptoms include slight discomfort, colored halos, visual blurring and an absence of frank pain. The patient may also be completely asymptomatic.
6. The attacks last from hours to a month, but typically occur spontaneously and resolve within two weeks.
7. The episodes may recur with varying frequency and are unpredictable and unprovoked.

Diagnosis

GCC typically occurs between ages 20 and 50.[1-3] It is considered uncommon after age 60 and before age 20, though research shows instances of patients exceeding these ranges.[4] GCC is typically recurrent and unilateral.[5-7] When it recurs, it afflicts the same eye. Bilateral, simultaneous involvement is uncommon.[8,9]

Classic uveitic symptoms of pain, photophobia and lacrimation are absent in GCC. Also, a markedly elevated IOP—ranging between 30mm Hg to 40mm Hg—is often seen, although some cases show IOP exceeding 70mm Hg.[1,2,6,7,10] The elevated IOP serves to disrupt the sodium-potassium pump on the corneal endothelium with resultant corneal edema and associated symptoms of blurred vision and halos.

The patient may complain of mildly blurred vision, ocular and periorbital discomfort, or may report no symptoms at all.[5-7,10] There may be a slightly injected eye with a mild anterior chamber inflammatory reaction with centrally located keratic precipitates.[1,2] Flare, common in uveitis, is not common in GCC and synechiae typically do not form.

GCC is an idiopathic inflammatory disease preferentially affecting the trabecular meshwork.[11-13] Both herpes zoster and cytomegalovirus (CMV) is associated with GCC.[11-13] Additionally, aqueous aspirations of patients in GCC have been positive for genomic fragments of herpes simplex virus (though no live virus has been sampled).[14] Currently, ample evidence exists for viral involvement in GCC, particularly CMV.[15-17] However, CMV is not invariably present in the aqueous humor of all eyes with GCC; thus the understanding of the pathophysiology of this syndrome remains incomplete.

Researchers have postulated that viral-induced inflammation of the trabecular meshwork (acute trabeculitis) impedes the tissue's aqueous processing ability, though this does remain speculative. The support for inflammation being concentrated in the trabecular meshwork is the fact that GCC is a non-granulomatous uveitic syndrome with no pronounced inflammatory cellular response causing mechanical obstruction of the trabecular meshwork, nor synechiae development leading to angle closure.

Management

As GCC is an inflammatory condition, the most effective treatment of both the inflammation as well as the secondary IOP elevation is a topical corticosteroid.[1,5-8] In that the inflammation is mild, doses of a steroid such as prednisolone acetate 1% greater than hourly is not necessary. As synechiae are unlikely to occur and patients are only minimally uncomfortable, cycloplegia is usually unnecessary. However, the use of a mild cycloplegic agent may be employed on a case-by-case basis.

Research documents successful reduction of IOP in GCC with carbonic anhydrase inhibitors, topical beta-blockers and alpha-2 adrenergic agonists.[18-20] However, all medications employed to reduce IOP should only be used adjunctively with primary anti-inflammatory therapy. Since GCC is an inflammatory condition, avoid miotics and prostaglandin analogs, as they can potentially exacerbate inflammation. In extremely recalcitrant cases, trabeculectomy remains a viable option in IOP management.[21,22] Recent research shows *ab interno* procedures with trabectome can assist in managing uncontrolled IOP in GCC.[23]

While permanent glaucomatous damage is not typical, it can occur, especially in patients with frequent recurrences and prolonged attacks.[1,2,5,24,25] Since a significant number of eyes with GCC likely have a viral etiology, such as herpes or CMV, antiviral therapy remains a option.[26-29]

Valcyte (valganciclovir, Genentech), an oral anti-CMV agent, can both manage the acute form of the syndrome and suppress recurrent outbreaks.[26,27] The dosage is 900mg BID for two weeks, followed by 450mg BID as long-term suppression therapy.[26,27] Cessation of therapy usually results in recurrence.[26,27] It is unlikely that all cases of GCC are due to CMV (or any virus), so a blanket recommendation to use oral antiviral medications to manage or suppress GCC has little evidence-based support.[26,27]

For this patient, we reviewed the clinical situation, prescribed topical prednisolone acetate 1% QID and dorzolamide/timolol BID. He felt back to normal within two days and the steroid was tapered after a week. At two weeks, he had no signs of inflammation and IOP, untreated, was now at 19mm Hg. ■

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NEW TECHNOLOGIES & TREATMENTS IN 2019 EYE CARE REVIEW
EDUCATION GROUP

CHARLESTON, SOUTH CAROLINA

Date: November 1-3, 2019

Location:
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170 Lockwood Blvd,
Charleston, SC 29403
Reservations: 1-800-228-9290

Limited discounted room rate: **$229/night**. Please book with the hotel directly by calling the number above. Mention "*Review*'s New Technologies & Treatments" for group rate.

Program Chair:
Paul M. Karpecki, OD, FAAO

Registration Cost: $495

Faculty:
Marc Bloomenstein, OD, FAAO
Douglas K. Devries, OD
Jack Schaeffer, OD, FAAO
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SAVE THE DATE!

The Optometric Retina Society & Review Education Group Present:

RETINAUPDATE2019

December 6 - 7 • Scottsdale, AZ

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ORS MISSION STATEMENT

The mission of the Optometric Retina Society (ORS) is to promote the advancement of vitreoretinal knowledge for clinicians, ophthalmic educators, residents, and students. The ORS is dedicated to posterior segment disease prevention, diagnosis, management and co-management.

THREE WAYS TO REGISTER

Email: reviewmeetings@jhihealth.com or Call: 800-999-0975

Online: www.reviewsce.com/ORSRetUpdate2019

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*This activity, COPE Activity Number 117855, is accredited by COPE for continuing education for optometrists.Approved by the Florida Board of Optometry.

Contact Lenses

New Monthly Toric

Astigmatic contact lens patients now have a new option for monthly replacement: the Air Optix Plus Hydraglyde for Astigmatism from Alcon. Like other products in its line, this one includes Alcon-specific technologies to increase comfort and lens stability. Alcon says unique attributes of the lens's design support long-lasting surface moisture, hold the lens in place with every blink and guard against deposit build-up. Alcon says the lens "performs as well on the last day of wear as the first."

Made of Alcon's lotrafilcon B silicone hydrogel material, the lens has a water content of 33% and dk/t of 108. Sphere power range is -10.00D to +6.00D, and cyl powers are -0.75, -1.25, -1.75 and -2.25 at axes 10 to 180 in 10-degree steps.

Visit www.alcon.com.

CL Aims to Reduce Dropout

By early next year, optometrists will be able to prescribe a new daily disposable contact lens called Precision1 that its manufacturer, Alcon, says is ideally suited to the new contact lens wearer. Alcon developed Precision1 to negate what it identifies as the top three motivators of dropout: poor vision, poor comfort and even the frustrations that arise from poor lens handling.

Precision1 uses a new silicone hydrogel material, verofilcon A, and includes a permanently adhered 'microthin' (2-3µm) layer of moisture. Alcon says this feature, which it calls SmartSurface, improves comfort and supports a stable tear film to reduce visual fluctuation. The lens has a water content of 51% at the core and greater than 80% at the anterior surface.

The company is positioning the new lens as a mid-tier option between its value-priced Dailies Aqua Comfort Plus and premium Dailies Total1 lines, with a recommended price of $78.75 per 90-pack. That's about $630 for an annual supply, though the company says pricing will ultimately be at the discretion of the doctor.

The lens will be available in a power range of -12.00D to +8.00D, with a 14.2mm diameter and an 8.3 base curve. Precision1 will begin rolling out to select US doctors this month, with widespread access anticipated for early 2020.

Visit www.alcon.com.

Vision Therapy

At-home Amblyopia Treatment

Clinicians looking for a vision therapy option for their amblyopia patients can now consider AmblyoPlay, a home-based platform designed by Smart Optometry. Patients wear red and blue glasses while using interactive gaming software designed to stimulate the ocular muscles for two 15-minute sessions per day, according to the company. Users are rewarded with tokens they can exchange for physical awards that are sent to their home.

The platform—designed with patients ages four to 14 in mind—provides different durations that address the unique needs of the user, according to the company. Parents can monitor their child's progress through the system's automated progression tracker and share the results at subsequent eye examinations.

AmblyoPlay is available with a three-month, six-month or one-year subscription and starts at $110.

Visit www.amblyoplay.com.

Supplementation

New Vitamin Combats Light Hazards

With our eyes increasingly exposed to blue light from digital devices, many people worry about potential long-term consequences. To help, Bausch + Lomb recently released a vitamin supplement called Ocuvite Eye Performance to help strengthen the macula. The goal, B+L says, is to help protect the eye from the stress of both natural sun light and artificial blue light.

The company says Ocuvite Eye Performance contains:

- lutein and zeaxanthin
- omega-3s
- zinc
- vitamins C, D and E

Ocuvite Eye Performance vitamins are available at major retailers nationwide and have a suggested retail price of $19.99 for a 50-count bottle.

Visit www.ocuvite.com. ■

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Faculty

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Candidates must be willing to actively participate in curricular assessment, professional development, student counseling and service activities within the college, university and the scientific community. Successful candidates are also expected to be involved in research and scholarly activities, and have a sincere commitment to optometric education, community service and patient care. Primary duties include, but are not limited to:

*a) **Teaching***
- *Developing and delivering lectures and/or laboratories for related areas, as assigned;*
- *Embracing and enhancing the didactic philosophies in the O.D. program;*
- *Maintaining and expanding the high quality clinical practice environment for optometry students on rotation;*
- *Precepting students on clinical rotation at the Midwestern University Eye Institute wher applicable;*

*b) **Service***
- *Helping to maintain and grow the state of the art optometry program with a strong interdisc plinary focus that meets the needs of patients in the surrounding community; is efficient, patient friendly, and cost-effective;*
- *Working closely together with all optometry and ophthalmology faculty to provide a complete range of eye and vision care services;*
- *Participating in leadership roles in state, regional, and national optometry organizations;*

*c) **Scholarly activity***
Engaging in research and scholarly activity, including presentations at scientific meetings, research, and publication in peer reviewed journals sufficient to qualify for academic advancement in a non-tenure or tenure track position.

***Qualifications:** Candidates must possess a Doctor of Optometry degree from an ACOE-accredited institution, must have completed an ACOE-accredited residency, and must be eligible for an illinois optometric state license. Primary eye care clinical expertise is also required.*

Salary will be commensurate with qualifications and experience
Review of applications will begin immediately and continue until the position is filled

Contact information: Interested applicants should apply online at www.midwestern.edu and include curriculum vitae and letter of interest specifying the position and college that he/she wishes to be considered for. Application packet should include curriculum vitae and letter of interest. Inquiries may be directed to Dr. Melissa Suckow, Dean; Midwestern University: msucko@midwestern.edu.

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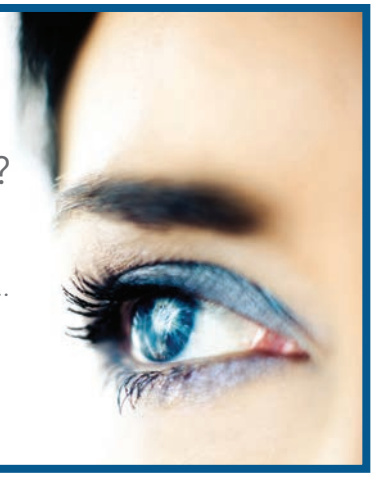

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3. Practice in a medical setting for a minimum of two years.#

www.abcmo.org

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^ At this time, 127 JC accredited hospitals, clinics and teaching institutions recognize ABCMO specialist certification.
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October 2019

■ **3-5.** *Idaho Optometric Physicians Annual Congress.* Boise Centre, Boise, ID. Hosted by: Idaho Optometric Physicians. Key faculty: Ryan P. Ames, Steven G. Ferrucci, Danica J. Marrelli, Mark W. Roark. CE hours: 19. For more information, email Randy Andregg at execdir@iopinc.org, call 208-461-0001 or go to idaho.aoa.org.

■ **3-6.** *SCO's Fall Homecoming/Fall CE Weekend.* Southern College of Optometry, Memphis, TN. Hosted by: Southern College of Optometry. CE hours: 24. For more information, email Jeanie Snider at jsnider@sco.edu, call 901-722-3397 or go to www.sco.edu/homecoming.

■ **3-7.** *The Art & Science of Optometric Care: A Behavioral Perspective.* OEP Education Center, Timonium, MD. Hosted by: The Optometric Extension Program. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call 410-561-3791 or go to www.oepf.org.

■ **4-5.** *East Coast Optometric Glaucoma Symposium.* Renaissance Baltimore Harborplace Hotel, Baltimore, MD. Hosted by: RGVCE. Key faculty: Murry Fingeret, Robert Weinreb. CE hours: 12. For more information, go to www.reviewsce.com/ecogs2019.

■ **4-6.** *Envision NY.* SUNY College of Optometry, New York City. Hosted by: SUNY College of Optometry. CE hours: 21. For more information, email Betsy Torres at btorres@sunyopt.edu, call 212-938-5830 or go to www.sunyopt.edu/cpe.

■ **5-6.** *Envision Conference West.* Western University of Health Sciences College of Optometry. Hosted by: Pennsylvania Optometric Association. CE hours: 24. For more information, email Michael Epp at michael.epp@envisionus.com, call 316-440-1515 or go to university.envisionus.com/conference.

■ **5-6.** *CE in Austin.* DoubleTree by Hilton Ausin, Austin, TX. Hosted by: University of Houston College of Optometry. Key faculty: Pat Segu. CE hours: 16. For more information, email UNCHO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live/2019/ce-in-austin/.

■ **5-7.** *CAO Annual Education Conference.* Mystic Marriott Hotel & Spa, Groton, CT. Hosted by: Connecticut Association of Optometrists. Key faculty: Ben Gaddie, Brad Sutton, Denise Valenti, Chris Wroten. CE hours: 18. For more information, email Lynn Sedlak at lsedlak@cteyes.org, call 860-529-1900 or go to www.cteyes.org/conference.

■ **10-13.** *GWCO Congress.* Oregon Convention Center, Portland, OR. Hosted by: Great Western Council of Optometry. Key faculty: Melissa Barnett, Mile Brujic, David Hicks, Aaron McNulty, Chris Wolfe, Sharon Carter. CE hours: 75 total; 26 per OD. For more information, email Patty Anderson at info@gwco.org, call 206-209-5273 or go to gwco.org.

■ **12-13.** *Forum on Ocular Disease.* Sheraton Lake Buena Vista Resort, Orlando, FL. Hosted by: PSS Eyecare. Key faculty: Damon Dierker, Ron Melton, Randall Thomas, Nathan Lighthizer, Deepak Gupta. CE hours: 18. For more information, email Sonia Kumari at education@psseyecare.com, call 203-415-3087 or go to psseyecare.com/orlando-fl.

■ **17-19.** *EastWest Eye Conference.* Huntington Convention Center of Cleveland, Cleveland, OH. Hosted by: Ohio Optometric Association. Key faculty: Melanie J. Frogozo, Alan Kabat, Blair Lonsberry, Sherrol Reynolds, Ron Melton, Randall Thomas CE hours: 78 total; 25 per OD. For more information, email Jordan Quickel at jquickel@ooa.org, call 800-999-4939 or go to www.eastwesteye.org.

■ **21-22.** *AFOS at Academy 2019.* Rosel Plaza Hotel and Orange County Convention Center, Orlando, FL. Hosted by: The Armed Forces Optometric Society. Key faculty: Federal Service Chiefs (Army, Navy, Air Force, VA and IHS) plus nationally recognized optometric educators. For more information, email Lindsay Wright at lwright@afos2020.org, call 720-442-8209 or go to www.afos2020.org.

■ **22.** *OGS Annual Meeting.* Hyatt Regency Orlando, Orlando, FL. Hosted by: Optometric Glaucoma Socety. For more information, go to optometricglaucomasociety.org/.

■ **23-27.** *Academy 2019 and World Congress of Optometry.* Orange County Convention Center, Orlando, FL. Hosted by: the American Academy of Optometry and the World Council of Optometry. CE hours: 400+. For more information, email the AAO at aaoptom@aaoptom.org, call 321-319-4860 or go to aaopt.org/annual-meeting/orlando-2019.

■ **26-27.** *GOA Fall Education Conference.* University of Georgia Center for Continuing Education & Hotel, Athens, GA. Hosted by: Georgia Optometric Association. CE hours: 18. For more information, email Vanessa Grosso at vanessa@goaeyes.com, call 770-961-9866 ext. 1 or go to www.goaeyes.com.

November 2019

■ **1-3.** *Music City Fall Classic.* Nashville Marriott at Vanderbilt University, Nashville, TN. Hosted by: Optometric Education Consultants. Key faculty: Paul Karpecki. CE hours: 20. For more information, email Vanessa McDonald at optoec@gmail.com, call 954-262-4224 or go to www.optometricedu.com.

■ **1-3.** *New Technologies & Treatments in Eye Care.* Charleston Marriott, Charleston, SC. Hosted by: RGVCE. Key faculty: Paul Karpecki, Doug Devries, Marc Bloomenstein, Robert P. Woodridge, Jack Schaeffer. CE hours: 19. For more information go to www.reviewsce.com/charleston2019.

■ **2-3.** *Forum on Primary Eyecare.* Twelve Midtown by Marriott, Atlanta, GA. Hosted by: PSS Eyecare. Key faculty: Ron Melton, Randall Thomas, Jerome Sherman, Robert Rebello, Larry Brown. CE hours: 18. For more information, email Sonia Kumari at education@psseyecare.com, call 203-415-3087 or go to psseyecare.com/orlando-fl.

■ **2-3.** *Maryland Optometric Association Annual Convention.* The Gaylord National Resort and Convention Center, National

Harbor, LA. Hosted by: Maryland Optometric Association. For more information, email Linda Cohen at lindacohen@marylandoptometry.org, call 410-486-9662 or go to www.marylandoptometry.org.

■ **7-11.** *VT2/Learning-related Visual Problems.* Western University, Pomona, CA. Hosted by: Optometric Extension Program Foundation. Key faculty: John Abbondanza. CE hours: 35. For more information, email Karen Ruder at karen.ruder@oep.org, call (410) 561-3791 or go to www.oep.org.

■ **8-10**. *ALOA Annual Convention.* Hyatt Regency Ballroom, Birmingham, AL. Hosted by: Alabama Optometric Association. Key faculty: Mile Brujic, Ron Melton, Randall Thomas, Walt Whitley, Mohammad Rafieetary. CE hours: 17. For more information, email Teri Hatfield at teri@alaopt.com, call 334-273-7895 or go to alabama.aoa.org/events/aloa-annual-convention.

■ **8-10.** *NCOS Fall Congress.* The Westin Charlotte, Charlotte, NC. Hosted by: North Carolina Optometric Society. Key faculty: Bruce Onofrey, Jennifer Lyerly, Nathan Lighthizer, Ron Melton, Randall Thomas. CE hours: 18. For more information, email Christy Santacana at christy@nceyes.org, call 919-977-6964 or go to www.nceyes.org/fall-congress.

■ **9-10.** *CE in Fort Worth.* Dallas Fort Worth Marriott Hotel & Golf Club, Fort Worth, TX. Hosted by: Univeristy of Houston College of Optometry. Key faculty: Marcus Gonzales. CE hours: 16. For more information, email UNCHO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live/2019/ce-in-fort-worth/.

■ **15-17.** *Monterey Symposium.* Monterey Conference Center, Monterey, CA. Hosted by: California Optometric Association. Key Faculty: Alan Kabat, Leonard Messner, Mohammand Rafieetary, James Thimons, Mark Wright. CE hours: 35. For more information, email Brenda Stewart at brends@coavision.org, call 916-266-5035 or go to www.coavision.org/i4a/pages/index.cfm?pageid=3288.

■ **15-17.** *AZOA Fall Congress.* Hilton Sedona Resort, Sedona, AZ. Hosted by: Arizona Optometric Association. Key faculty: Bryan M. Rogoff, Greg Caldwell, Joshua Duncan, Jay Haynie. CE hours: 14. For more information, email Kate Diedrickson at kate@azoa.org, call 602-279-0055 or go to www.azoa.org/connect.

■ **23-24.** *Everything Therapeutic: San Antonio.* Westin Riverwalk Hotel, San Antonio, TX. Hosted by: University of Houston College of Optometry. Key faculty: William Townsend, Joseph Sowka, David Sendrowski, Robert Prouty. CE hours: 16. For more information, email UNCHO Continuing Education at optce@central.uh.edu, call 713-743-1900 or go to ce.opt.uh.edu/live/2019/everything-therapeutic-san-antonio/.

To list your meeting, please send the details to:
Mark De Leon, Associate Editor
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Diagnostic **Quiz**

A Diagnosis in the Same Vein

By Andrew S. Gurwood, OD

History

A 67-year-old black female reported to the office with a chief complaint of vision changes OS of four weeks' duration. She reported noticing a cloudiness after waking and that the reduction in clarity continued to worsen over the next three weeks. Her systemic history was remarkable for hypertension, diabetes and dyslipidemia which was well controlled with appropriate oral medications.

Her ocular history was remarkable open angle glaucoma controlled with prostaglandin monotherapy and an uncomplicated non-ischemic central vein occlusion OS, which had occurred a year earlier and resolved without requiring ocular intervention. She denied allergies of any kind.

This 67-year-old patient has a history of open-angle glaucoma and an uncomplicated non-ischemic central vein occlusion. She also had hypertension, diabetes and dyslipidemia. Does this presentation plus her history help identify the cause of her reduced visual clarity?

Diagnostic Data

Her best-corrected entering visual acuities were 20/20 OD and 20/70 OS at distance and near with no improvement upon pinhole. Her external exam was normal with no evidence of afferent pupil defect. The biomicroscopic exam of the anterior segment was normal with no evidence of iris neovascularization. Goldmann applanation tonometry measured 15mm Hg OU. The pertinent findings are demonstrated in photographs. We also noted peripheral pathologies in both eyes.

Your Diagnosis

Does the case presented require any additional tests, history or information? Based on the information provided, what would be your diagnosis? What is the likely prognosis? To find out, visit us at www.reviewofoptometry.com. ■

Next Month in the Mag

Coming in October, *Review of Optometry* will present its Annual Diagnostic Skills & Techniques Issue.

Topics include:

- *Is It Glaucoma, Ocular Hypertension or Something Else?*
- *"If I'm 20/20, Why Can't I See Well?"*
- *Diagnosing Thyroid Eye Disease*
- *What to Do When You Suspect Neuro Disease*

Also in this issue:

- *Solving Tricky Accessibility Problems for Patients*
- *When Medications Worsen Dry Eye Disease* (Earn 2 CE Credits)

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Biofinity® toric is the **most prescribed** toric lens on the market[3] with over **20,000 SKUs** – more than **5x the range of other monthly toric contact lenses**

Biofinity® multifocal with 4 distinct ADDs up to +2.50 and **OptiExpert™**, a free multifocal fitting app from CooperVision® gives you a **fast, accurate fit to save valuable chair time**[4]

Innovative products to meet your patients' needs today. And a pulse on your future needs as **CooperVision® continues to invest in the Biofinity® product portfolio**

Prescribe Biofinity® and stay tuned to see what's on the horizon at OnlyBiofinity.com

FROM SPHERE TO SPECIALTY
Only Biofinity®

CooperVision®
SPHERE TORIC MULTIFOCAL

1. Data on file. **2.** US monthly single vision lens. **3.** US industry data on file. **4.** CVI data on file 2019. Retrospective analysis; N=55 subjects (110 eyes); DV Rx +1.25D to -3.25D, ADD powers +1.25 to +2.50DS .

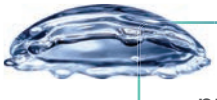
INTRODUCING PRECISION 1®

A LENS DESIGNED WITH NEW WEARERS IN MIND

FEATURING SMARTSURFACE® TECHNOLOGY FOR

PRECISE VISION AND DEPENDABLE COMFORT[1]

Contact lens wearers rated PRECISION1® as **SUPERIOR** to 1-DAY ACUVUE^ MOIST for end of day vision, end of day comfort and overall handling in a clinical study[2]



New SMARTSURFACE® Technology provides a microthin, high-performance layer of moisture on the lens surface that **EXCEEDS 80% WATER.**[3]

THE LENS FOR YOUR NEW WEARERS TO

START IN AND **STAY IN**

^Trademarks are the property of their respective owners.

References: 1. Alcon data on file, 2018. **2.** Alcon data on file, 2019. Based on mean subjective ratings from a prospective, randomized, bilateral crossover, double-masked, controlled clinical trial of PRECISION1® and 1-DAY ACUVUE^ MOIST contact lenses; $p \leq 0.0001$. **3.** Alcon data on file, 2018.

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

Alcon