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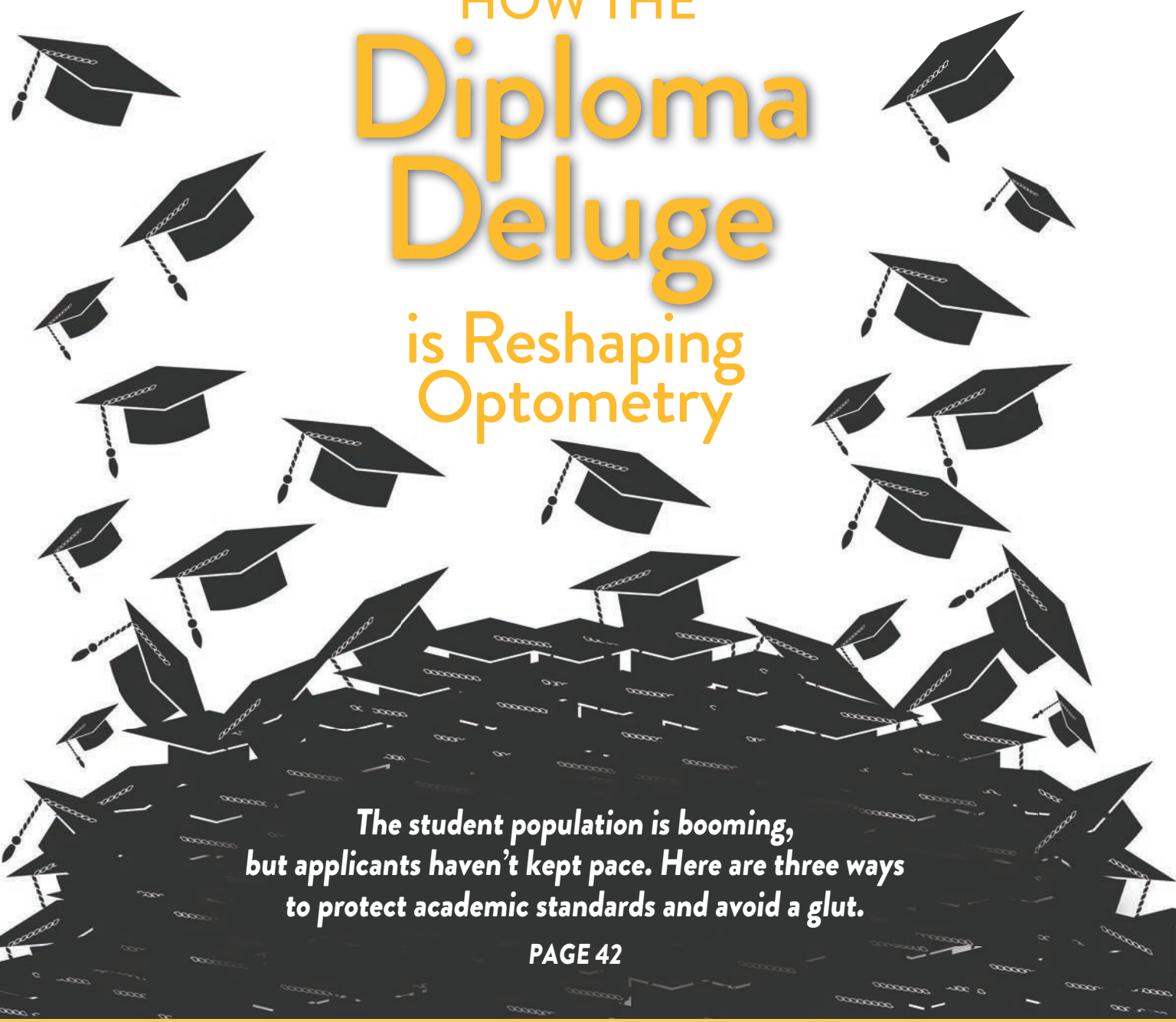
REVIEW® OF OPTOMETRY

February 15, 2018 www.reviewofoptometry.com

HOW THE Diploma Deluge is Reshaping Optometry



The student population is booming, but applicants haven't kept pace. Here are three ways to protect academic standards and avoid a glut.

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• **Prospects for Neuroprotection in Glaucoma,** *Page 66* • **AI for DR: "The Digital Doctor Will See You Now,"** *Page 74*

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

REFERENCE: 1. Data on file. Bausch & Lomb Incorporated. 3rd Party Industry Report. 2016-2017.
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VOL. 155 NO. 2 ■ FEBRUARY 15, 2018

IN THE NEWS

Researchers have found an **improved gene therapy delivery mechanism** that might aid in **future glaucoma treatments**. By blocking proteasomes when using the feline immunodeficiency virus to deliver marker genes, they found it **roughly doubled the transfer of genes** entering the trabecular meshwork cells. Such improved delivery methods could boost the efficacy of future gene therapy for glaucoma, the researchers said.

Aktas Z, Rao H, Slauson SR, et al. Proteasome inhibition increases the efficiency of lentiviral vector-mediated transduction of trabecular meshwork. Invest Ophthalmol Vis Sci. 2018;59(1):298-310.

Eye injuries related to paintball or BB and pellet guns more than doubled from 1990 to 2012, a new study found. Although the injury rate has slightly declined for all sports in this time, these **gun injuries accounted for almost half of all hospitalizations**, data from the National Electronic Injury Surveillance System revealed—highlighting the need for better prevention efforts for patients participating in air-gun related recreation.

Miller KN, Collins CL, Chounthirath T, et al. Pediatric sports- and recreation-related eye injuries treated in US emergency departments. Pediatrics. 2018;141(2):e20173083. [Epub].

After undergoing an extensive, voluntary review process, the Council on Optometric Practitioner Education (COPE) **CE accreditation has been deemed** "**substantially equivalent**" to the Accreditation Council for Continuing Medical Education's **CME accreditation requirements**, criteria, policies and decision-making process for medical practitioners. Such distinction creates a path for shared inter-professional education, leading the way forward for optometry to become a substantial and accepted part of a larger healthcare system, according to COPE.

Rare Condition, Not-so-rare Therapies

Precision eye care is in the pipeline—and may suggest surprising treatment strategies.

By Rebecca Hepp, Managing Editor

Patients with rare eye conditions such as neovascular inflammatory vitreoretinopathy (NIV) often have limited therapeutic options, especially when conventional treatments fail. But researchers at the Stanford University School of Medicine are looking to change that with precision medicine. They turned to proteomics to better identify molecular pathways in NIV pathologies—and available potential targets for therapy.

Using liquid biopsies, the investigators tested for expression of 200 cytokine-signaling proteins and found 64 that were different, 61 of which were upregulated.

"By performing a proteomic evaluation of an ocular sample in a disease state, and determining up-or down-regulation of specific molecules, like cytokines, ideal treatable targets can be identified," says Jessica Steen OD, an assistant professor at Nova Southeastern University. Several from this study show promise:

Anti-vascular endothelial growth factor (VEGF). After noting elevated VEGF, the researchers tried intravitreal Avastin (bevacizumab, Genentech) in seven eyes. In each case, the vitreous hemorrhage grade resolved without surgery and the patients' vision returned to baseline.

Methotrexate. The proteomic data suggested the significant role played by the mTOR and class I PI3K signaling pathways, which are critical in the development of T-cells. Thus, the researchers used intravitreal injections of methotrexate, and the number of cells in the anterior chamber dropped dramatically in five eyes with Stage II and Stage III NIV.

Corticosteroids. Because the personalized proteome revealed upregulated cytokines that should be quelled by corticosteroids, the researchers used a fluocinolone acetonide (Retisert, Bausch + Lomb) implant in three eyes to mitigate the adverse effects of oral therapy such as weight gain and osteoporosis. They were able to reverse several clinical features such as retinal neovascularization for three years.

Anti–IL-6. Some study participants continued to have elevated IL-6, possibly responsible for their severe intraocular fibrosis, according to the researchers. For one patient with retinal fibrosis following retinal detachment repair surgery (causing re-detachment), the researchers tried monthly intravenous infusions of tocilizumab (anti–IL-6) over six months post-reattachment surgery. The new treatment allowed her retina to remain attached—a first for reattachment surgery in an

(continued on page 6)

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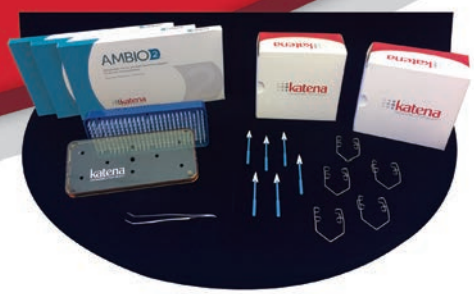
[1] Koob TJ, Lim JJ, Zabek N, Massee M. 2014. Cytokines in single layer amnion allografts compared to multilayer amnion/chorion allografts for wound healing. J Biomed Mater Res Part B 2014:00B:000-000



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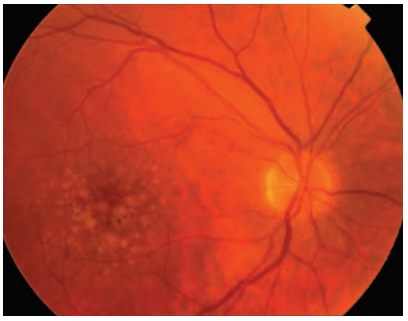
AMD Assessment Made Easy

Clinicians looking for a simple way to assess a patient's risk for age-related macular degeneration (AMD) can consider adding a new tool to their practice. Researchers from Italy and France have created the simplified Théa AMD risk-assessment scale (STARS), a 13-item questionnaire that includes risk factors such as age, ethnicity, family history, body mass index, smoking habits, hypercholesterolemia, hypertension and myocardial infarction.

"Strengths of this questionnaire are that it can be administered quickly, is validated by an external population and includes many of the risk factors widely considered to be associated with, if not causally linked to, the development of AMD," says Andrew J. Rixon, OD, of the Memphis VA Medical Center. "While this questionnaire does not contain novel information, it streamlines the risk assessment process in a meaningful way."



A new questionnaire may help clinicians catch early forms of dry AMD, as seen here.

Based on data from 12,639 patients in Italy and 6,897 patients in France, the questionnaire provides an opportunity to increase awareness through AMD public health initiatives, which "ideally results in patients at all risk levels seeking appropriate comprehensive eye care," Dr. Rixon says. "The earlier the patient and practitioner are aware of risk, the greater the chance for reduction in loss of function."

While useful, the questionnaire should help inform the broader assessment of the patient, not replace it, Dr. Rixon stresses. "Funduscopic evaluation, cross sectional macular analysis by OCT, fundus autofluorescence and knowledge of the patient's nutritional status, among others, still supplant strict interpretation of a risk questionnaire in the decision making process."

Proteomics Tailor NIV Therapy, Shows Promise

(continued from page 4)

NIV patient, the researchers wrote.

They also found TNF-α was not elevated, which explains why infliximab (anti-TNF-α) infusions hadn't alleviated patient symptoms.

These new findings hold promise for future strategies for NIV and, one day, other ocular conditions.

"Looking ahead, the goal of ophthalmic precision medicine will be to use proteomics and genetic evaluation to predict disease course and prognosis and to determine ideal therapies for patients with a wide range of conditions, including glaucoma, diabetic retinopathy and age-related macular degeneration," according to Dr. Steen.

Of course, research has a long way to go before this technique achieves a wider application, considering the variability and genetic complexity in most ocular diseases.

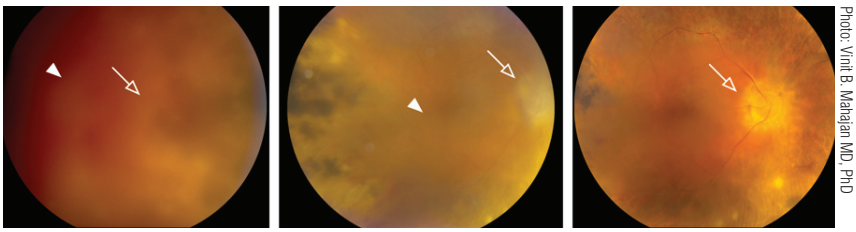


Photo: Vinit B. Mahajan MD, PhD

This patient with Stage III NIV has significant vitreous hemorrhage (arrow heads) and vision reduced to count fingers. At left is initial presentation, while the middle and left images are two and four weeks after Avastin injection, respectively. At two weeks, the vitreous hemorrhage was resolved and vision improved to 20/70. By four weeks, vision was 20/50 and the optic nerve head (open arrow) is visible.

"The authors had the advantage of reducing the variation of gene expression between individuals, as the family of patients studied all had the identical genetic variant resulting in NIV," says Dr. Steen. "This is not the case in multifactorial retinal conditions such as diabetic retinopathy and macular degeneration, which are a result of multiple genes, many gene products and their interaction with environmental and metabolic factors."

But one thing is for sure: the potential of precision medicine is closer than ever before.

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SECO, VEE Kick Off 2018 Conference Season

The first quarter of 2018 is packed with educational opportunities for optometrists. Two of the biggest shows interrupt the late-winter blues with CE sessions, learning labs, workshops, specialty tracks, business programs and more: SECO in late February into early March and Vision Expo East just two weeks later. Here's a look at what each one offers so you can show up prepared:

Atlanta-bound

At the SECO Annual Congress in Atlanta Feb. 28 to March 4, you can expect to hear and learn from some of the most respected eye care practitioners in the profession.

"SECO has changed the game when it comes to value for the dollar and invaluable information that can be put to immediate use," says Paul C. Ajamian, OD, SECO optometric education program committee chair. "With all-new tracks like 'Squawk Box' and 'Snap Chat' plus attention to specialized topics like MRSA, GCA and TBI, SECO provides custom-crafted courses, special sessions and learning labs available nowhere else in eye care."

Here are some conference highlights you won't want to miss:

MedPro360. Learn proven business management strategies from pros like management consultants Jenn Lim of Delivering Happiness and Donald Miller of Storybrand, plus HGTV's Vern Yip.

Gear Up for Glaucoma! James Thimons, OD, and Robert Noecker, MD, will provide an in-depth look at the new diagnostic and management technologies and pharmaceutical agents revolutionizing glaucoma care, now and in the future.

Eye Cancers: From Melanoma to Retinoblastoma. J. William Harbour, MD, will discuss diagnostic techniques, differentials, treatment, outcomes and breakthroughs for patients with uveal melanoma and retinoblastoma.

Anterior Segment Solutions. Zaina Al-Mohtaseb, MD, will discuss diagnosing infectious and non-infectious keratitis through cases and videos. He will also touch on cataract surgery in patients with corneal disease and secondary and presbyopia-correcting IOLs.

Retina 2018. Jay Haynie, OD, and Ali Zaidi, MD, will highlight OCT angiography and its role in primary eye care in 2018. Attendees will also hear about the latest clinical trials regarding AMD and diabetic retinopathy.

Cornea Update. Joe Shovlin, OD, FAAO, and Andrew Bartlett, MD, will explore the epidemiology and pathogenesis of ulcerative keratitis.

Secrets of a Vascular Surgeon. Brad R. Grimsley, MD, FACS, will provide an in-depth look at extracranial cerebrovascular occlusive disease as it relates to vision.

For more information or to register, go to attendseco.com.

The Big Apple

New York City's Jacob Javits Center will welcome thousands of optometrists, opticians and industry executives on March 15 for the International Vision Expo & Conference East (VEE), which runs until the 18th.

Hosted by Reed Exhibitions and the Vision Council, VEE boasts a line-up of heavy hitters on the CE lecture circuit and all-new learning opportunities.

"For the first time, at Vision Expos East and West 2018, we'll be providing our education attendees with hands-on training in some of today's most important topics," says Dr. Gaddie, co-chairman of the conference advisory board. "Our new optical coherence tomography workshop provides the opportunity to work with the latest diagnostic equipment and hear best practices direct from the experts. We'll also have a new scleral lens workshop, allowing first-hand experience with new techniques as specialty contact lenses gain more traction with optometry."

Here are some of the must-see sessions at VEE 2018:

Aesthetics Track. Patients are always searching for ways to look younger. Learn how to implement an ocular aesthetics model into your practice and help make their dreams a reality.

Specialty Lens Track. Attendees will hear a comprehensive discussion of specialty lens fitting and evaluation.

Global Contact Lens Forum. This "meeting within a meeting" will deliver the latest insights and solutions used by today's contact lens practitioners, with a focus on business and growth strategies.

OptiCon. In 2018, OptiCon officially joins the VEE line-up, offering educational programming that will cover four key areas: spectacles, contact lenses, business and exam review. Attendees will also be able to take ABO and NCLE review courses and sit for the exams.

Additional courses throughout the conference will cover all the skills and responsibilities an optometrist is tasked with today—and tomorrow.

For more information or to register, go to east.visionexpo.com. ■

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THE FORMULATION OF LOTEMAX® GEL

- **ENGINEERED TO ADHERE TO THE OCULAR SURFACE**[1,2]
  - Adaptive viscosity: Gel at rest, viscous liquid on the eye
  - Drug-related blurred vision was rarely reported (0.25%, 2/813)
- **~70% LESS PRESERVATIVE than LOTEMAX® SUSPENSION (loteprednol etabonate ophthalmic suspension) 0.5%**[2,3,5]
- **DOSE UNIFORMITY–EVERY DROP, EVERY TIME**
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- **pH OF 6.5 CLOSE TO THAT OF HUMAN TEARS**[2]
- **CONTAINS 2 KNOWN MOISTURIZERS**[3]
  - Glycerin and propylene glycol

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Indication

LOTEMAX® GEL (loteprednol etabonate ophthalmic gel) 0.5% is indicated for the treatment of post-operative inflammation and pain following ocular surgery.

Important Safety Information about LOTEMAX® GEL

- LOTEMAX® GEL is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.
- Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. If this product is used for 10 days or longer, IOP should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.
- Use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation and occurrence of perforations in those with diseases causing corneal and scleral thinning. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, and where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infection. In acute purulent conditions, steroids may mask infection or enhance existing infection.
- Use of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.
- Patients should not wear contact lenses when using LOTEMAX® GEL.
- The most common ocular adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%) and foreign body sensation (2%).

Please see brief summary of Prescribing Information on adjacent page.

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

* Fingertip Formulary data 2017

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loteprednol etabonate ophthalmic gel 0.5%

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to prescribe Lotemax Gel safely and effectively. See full prescribing information for Lotemax Gel.

Lotemax (loteprednol etabonate ophthalmic gel) 0.5%

Rx only
Initial Rx Approval: 1998

INDICATIONS AND USAGE

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

DOSAGE AND ADMINISTRATION

Invert closed bottle and shake once to fill tip before instilling drops.

Apply one to two drops of LOTEMAX into the conjunctival sac of the affected eye four times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

CONTRAINDICATIONS

LOTEMAX, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

Intraocular Pressure (IOP) Increase

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

Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing

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

Bacterial Infections

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

Viral Infections

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

Fungal Infections

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

Contact Lens Wear

Patients should not wear contact lenses during their course of therapy with LOTEMAX.

ADVERSE REACTIONS

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

The most common adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%), and foreign body sensation (2%).

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects

Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (35 times the maximum daily clinical dose), a dose which caused no maternal toxicity. The no-observed-effect-level (NOEL) for these effects was 0.5 mg/kg/day (6 times the maximum daily clinical dose). Oral treatment of rats during organogenesis resulted in teratogenicity (absent innominate artery at ≥5 mg/kg/day doses, and cleft palate and umbilical hernia at ≥50 mg/kg/day) and embryotoxicity (increased post-implantation losses at 100 mg/kg/day and decreased fetal body weight and skeletal ossification with ≥50 mg/kg/day). Treatment of rats with 0.5 mg/kg/day (6 times the maximum clinical dose) during organogenesis did not result in any reproductive toxicity. Loteprednol etabonate was maternally toxic (significantly reduced body weight gain during treatment) when administered to pregnant rats during organogenesis at doses of ≥5 mg/kg/day.

Oral exposure of female rats to 50 mg/kg/day of loteprednol etabonate from the start of the fetal period through the end of lactation, a maternally toxic treatment regimen (significantly decreased body weight gain), gave rise to decreased growth and survival, and retarded development in the offspring during lactation; the NOEL for these effects was 5 mg/kg/day. Loteprednol etabonate had no effect on the duration of gestation or parturition when administered orally to pregnant rats at doses up to 50 mg/kg/day during the fetal period.

There are no adequate and well controlled studies in pregnant women. LOTEMAX should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether topical ophthalmic administration of corticosteroids could result in sufficient systemic absorption to produce detectable quantities in human milk. Systemic steroids appear in human milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. Caution should be exercised when LOTEMAX is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment Of Fertility

Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with up to 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (600 and 300 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender.

PATIENT COUNSELING INFORMATION

Administration

Invert closed bottle and shake once to fill tip before instilling drops.

Risk of Contamination

Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the gel.

Contact Lens Wear

Patients should be advised not to wear contact lenses when using LOTEMAX.

Risk of Secondary Infection

If pain develops, redness, itching or inflammation becomes aggravated, the patient should be advised to consult a physician.

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

US Patent No. 5,800,807

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Based on 9269101/9269201 Revised: 08/2016

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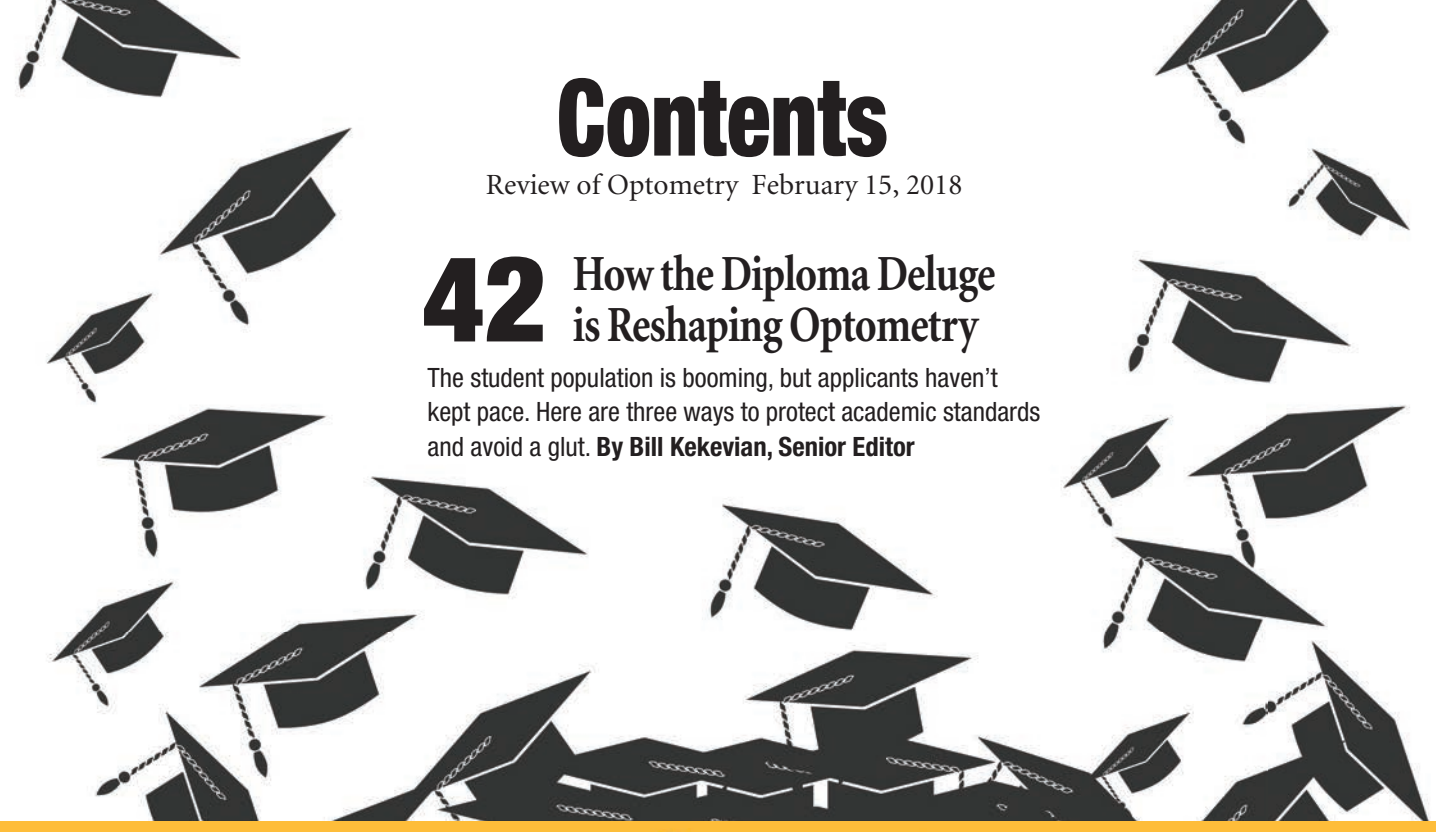

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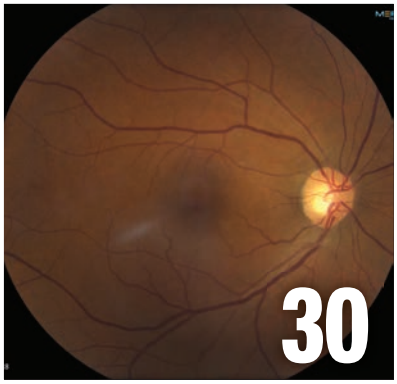
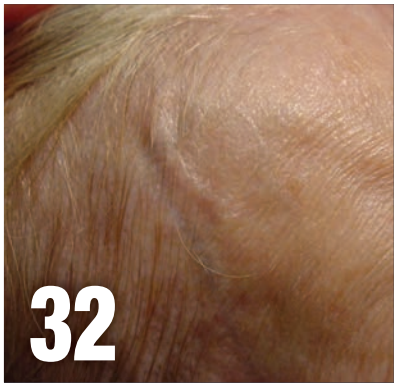
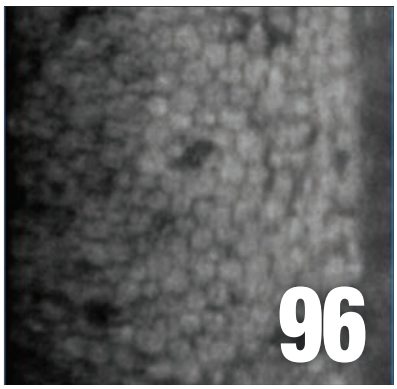
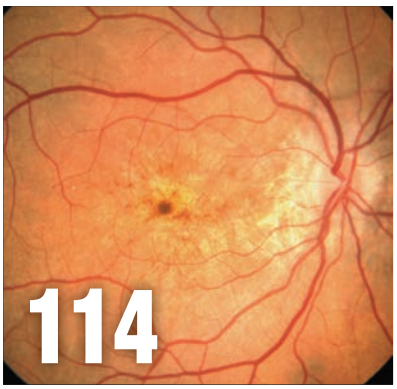
She's on a Losing Streak
ANDREW S. GURWOOD, OD









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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.

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BRIEF SUMMARY:
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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

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For more information, go to www.Xiidra.com or call 1-800-828-2088.
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Letters to the **Editor**

> *We must, as a profession, start speaking about low vision to all AMD patients. The only way for that to happen is for our leaders to speak it in the journals, supplemental publications and CE course.*

Low Vision Advocacy for AMD

In the October 2017 article "Flex Your Core Muscles," Paul Karpecki, OD, describes a patient with a condition and symptoms that had been managed medically without improvement. He states the most important step in solving this person's issues was "remembering the core strengths that set optometrists apart from other professions; in this case, looking for and measuring subtle eye misalignments," which changed the patient's life. He concludes, "never lose sight of the core strengths we have in this profession—that's what truly differentiates us."

Yet, in a supplement in the same issue ("Practical Guidelines for the Treatment of AMD"), seven highly respected optometrists have done exactly what Dr. Karpecki warns against. It ignores why people visit optometrists: they want to *see*.

One line stands out: "Currently, there is no cure for AMD." Medically that may be true, but not visually, and optometry is the vision care profession. So it confounds me why the most important practical recommendation—referral to a low vision optometrist—is not mentioned.

The recommendations that are included are correct:

- Prescribe smoking cessation.
- Prescribe nutritional supplementation.
- Discuss lifestyle modifications such as diet and exercise.
- Manage systemic disease.
- Prescribe blue light protection.
- Prescribe UVA and UVB sunglasses protection for outdoors.

But not one of these will improve the vision, function or independence of the person seeking help for their vision loss. Not one of these recommendations will alleviate the anxiety and possible depression that frequently accompanies the AMD diagnosis.[1] None can, as Dr. Karpecki states, "change the patient's life."

The introduction states the sponsor assembled a clinical advisory board to develop practical guidelines that can be implemented in a medically oriented practice. While this makes sense to optometrists, I question whether people who visit optometrists know the difference between an optometrist and a medically oriented optometrist, given that optometry has no "specialties."

We must, as a profession, start speaking about low vision to all AMD patients. The only way for that to happen is for our leaders to speak it in the journals, supplemental publications and CE courses.

People visit us because they want to see better. Let's remember our core strengths, regardless of how we orient our optometric practices.

Richard J. Shuldiner, OD,
Low Vision Diplomate,
American Academy of Optometry;
President, International Academy of Low Vision Specialists;
Clinical Director, Low Vision Optometry of Southern California

1. Cimarolli VR, Casten RJ, Rovner BW, et al. Anxiety and depression in patients with advanced macular degeneration: current perspectives. Clin Ophthalmol. 2016;10:55-63.

Editor's note: There's no doubt that patients with visual disabilities can be helped through low vision interventions. However, the goal of the AMD piece in question was to describe how an optometrist can influence the disease course itself. Managing the visual consequences, while certainly important, fell outside the scope of that particular project, just as an article on treating glaucoma might be limited to medical options and exclude surgical methods (or vice-versa). Other articles *Review* publishes are narrowly focused on low vision specifically and, by design, don't devote space to diagnostic or medical issues.

We at *Review* strive to cover all important topics in the profession in due course, while limiting the scope of any given article to a specific segment of the chosen topic for brevity and simplicity of message.

Dr. Shuldiner is to be commended for raising awareness of low vision services and the ways an optometrist can use these interventions on their patient's behalf.

Praise for Planner

Just wanted to express my gratitude for your *2018 Conference Planner* in the December issue. I'm involved in planning for various groups, and it's important to know the dates and focus of events in the field. No one person can keep up with it all, so your supplement is really handy, not just for doctors but exhibitors, too. People can pick and choose meetings that interest them educationally, strategically and even socially.

During my long career, I've watched many of these meetings grow into real powerhouses. They advance our profession as a whole and each of us as individuals. What a wonderful way for us to help each other to learn and improve.

Frank D. Fontana, OD
St. Louis, Mo.

Editor's note: Dr. Fontana entered practice in 1950 and was instrumental in founding the Heart of America conference in 1962. ■

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Outlook

By Jack Persico, Editor-in-Chief

The Stars of Tomorrow

We shine a light on several new ideas that might change clinical practice. Will they flourish—or flop?

In 2005, three young men—none over age 30—started a video-sharing website in a small office over a pizzeria. They called the site YouTube. Less than two years later, they sold it to Google for $1.6 billion, at an age when most people are still paying off student loans. YouTube's founders had noticed three interesting trends: internet connection speeds were increasing, computer storage costs were dropping and a new phenomenon called social networking was on the rise. At the nexus of those they found a billion-dollar idea.

Even though medical research lacks the dazzle (and frivolity) of YouTube, the site's story is akin to the ingenuity and impact we look for when choosing topics for our annual Innovations in Eye Care issue. Is there a product, concept or scientific breakthrough that stands a chance of radically changing and improving the day-to-day lives of doctors and their patients? Did it come about in an unusual, and possibly unanticipated, way? Is it close enough to launch to reliably expect it to come to fruition?

This month we highlight four we think qualify: an eye drop that might help regrow corneal endothelial cells, an implant that could radically reduce the number of eye injections performed, a wholly different way of thinking about glaucoma therapy and a technology-driven solution to the diabetes epidemic.

Maybe one of them will be the next YouTube, or maybe not. But they're all interesting stories about the sweet spot where science, business and inspiration converge.

Health care has the same mix of big ideas and deep pockets as Silicon Valley, often with success stories to match. One product in this month's series, the Port Delivery System (PDS) for sustained-release anti-VEGF therapy, comes from a start-up 'factory' of sorts called ForSight Labs. Founded by retina specialist Eugene de Juan, MD, the company has already sold off three products that landed at behemoths: the PDS at Roche, the CyPass shunt at Alcon and a glaucoma drug sustained-release implant at Allergan.

Of course, not every promising idea makes it. ReVision Optics, manufacturer of the Raindrop corneal inlay for presbyopia, abruptly announced in late January that the company was going out of business. So far, there's been no announcement about what might become of the Raindrop. Maybe another company will acquire it at fire-sale prices and try again. Or maybe it's just gone. ReVision had a tough row to hoe—only a single product to sell, and in a very crowded market. Presbyopia correction already runs the gamut from dollar-store reading glasses to pricey premium IOLs, with options to suit every budget and personality.

Consider the Raindrop a cautionary tale as you read about what we (or anyone) speculates might be the Next Big Thing. Everyone wants to believe in and root for a new product at first. But after the honeymoon phase is over, it needs to offer more than just incremental improvement over other options, especially when going up against established players and entrenched habits. ■

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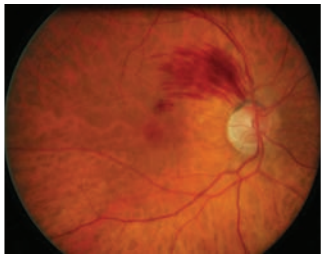

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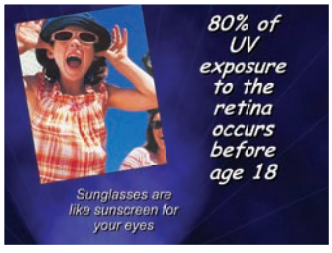

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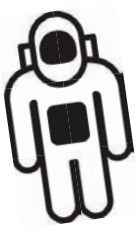

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Reinventing Optometry Every Day

One thing we can count on is continuous, rapid change—and our profession is no exception. **By Paul M. Karpecki, OD, Chief Clinical Editor**

Extraordinary—dare I say paradigm-shifting—changes are streaming by us almost daily. New drug delivery systems, treatments for previously untreatable eye conditions, advanced diagnostics and ground-breaking research are making it harder to know the limitations of patient care. This month's innovations special issue tackles some of the largest eye disease categories, giving you a sneak peek of your future.

Glaucoma

It's been two decades since we've had a new glaucoma drug treatment class or mechanism of action, and we have two approved in one month! Vyzulta (latanoprostene bunod 0.024%, Bausch + Lomb), includes a nitric oxide donor to increase aqueous outflow and works directly on the trabecular meshwork (TM). Rhopressa (netarsudil 0.02%, Aerie Pharmaceuticals), also works directly on the TM to increase aqueous outflow while decreasing aqueous production and inhibiting episcleral venous pressure.

It's still too early to recommend off-label use, given what little we know about rho-kinase effects on human corneal endothelium, but further investigation may also lead to a future treatment for corneal edema in endothelial diseases.

AMD

One of the fastest growing diseases in the United States is macular degeneration, in part due to the aging population and unavoidable genetic influences, but unfortunately also perhaps due to our diets and lifestyle. Macular degeneration is already the leading cause of vision loss in Americans 60 years of age and older, and the incidence is expected to double by 2050.[1] In addition, advanced AMD (wet and geographic atrophy) are the leading causes of irreversible blindness and visual impairment worldwide.[1] Optometry must play a greater role in patient care, including earlier diagnosis (e.g., dark adaptometry), better monitoring (e.g., home-based Amsler testing), better use of technology (e.g., autofluorescence, OCT-angiography, swept-source OCT) and active treatment. This last group may include new anti-VEGF delivery systems, implantable telescopic IOLs or referral to a low vision specialist within our profession.

Diabetes

The CDC expects the number of people with diabetes in the United States, already more than 30 million, to triple by the year 2050.[2] As the quarterback for diabetic retinopathy and the physician/specialist communication, optometrists must prepare for this. Advanced digital imaging—from higher resolution and ultra-widefield testing to OCT—in conjunction with a dilated fundus exam, will all be crucial in early diagnosis and treatment.

Thyroid Disease

According to the American Thyroid Association, more than 12% of Americans (more than 20 million people) will develop a thyroid condition during their lifetime—and 60% of those with thyroid disease are not yet diagnosed.[3] You would be surprised by how many non-responsive ocular surface conditions turn out to later be associated with thyroid disease. This month's Optometric Study Center comes from a true expert in the field with both clinical knowledge and personal experience.

Myopia

Finally, we can't overstate the growing incidence of myopia in the United States and around the world. Because of changing environmental and lifestyle factors, such as digital device use and less time outdoors, its prevalence will only increase. While the issues surrounding vision with high myopia are cause enough for concern, the ocular disease risks such as glaucoma, myopic degeneration, retinal detachment and myopic cystoid macular edema only make this condition more alarming. All clinicians must be prepared to care for this patient population, and evolving standards of care are geared toward controlling myopia in each and every patient.

Innovation is booming—and essential to our future. Heed what's written here and use it to help boost your knowledge, your practice and, most importantly, your patients. ■

1. Rein DB, Wittenborn JS, Zhang X, et al. Forecasting age-related macular degeneration through the year 2050: the potential impact of new treatments. Arch Ophthalmol. 2009;127(4):533-40.
2. Centers for Disease Control and Prevention. Number of Americans with diabetes projected to double or triple by 2050. CDC newsroom. www.cdc.gov/media/pressrel/2010/r101022.html. Accessed January 4, 2018.
3. American Thyroid Association. General Information/Press Room. www.thyroid.org/media-main/about-hypothyroidism. Accessed January 4, 2018.

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25 Ways to Win the Temp Wars

Private practice optometry runs hot and cold—and I'm not talking about the business side of things. **By Montgomery Vickers, OD**

The way most offices are laid out, with little rooms spread about, ensuring a comfortable temperature everywhere is like trying to get pachymetry on a four-year-old or a wild boar (no difference), which is roughly equal to getting a 50-year-old to understand why he needs reading glasses after LASIK. This room is blazing; that one is freezing. The staff is miserable, and the thermostat is a war zone.

I always told my kids that you can solve almost any problem if you throw enough money at it, but that's not true for HVAC problems. After practicing in all kinds of weather since 1979, here's some advice:

1. If you get the chance to design your office (not just inherit the space that destroyed the previous owner's spirit), accept that you will freeze or burn up, no matter what you do.

2. Dress in layers. Start with Speedos and end with polar bear fur, no matter the season. Use plenty of Velcro for more efficient changes in the hallway.

3. When the staff complains about the temperature, pretend you care.

4. Avoid the rooms that are always too hot or too cold. The best way to do this is to stay on vacation.

5. If your hands are freezing, take advantage of it by making piecrusts.

6. If your hands are sweating, refer foreign body removals to a cooler colleague or duct tape the spud to your fingers. Otherwise, just remember that two corneal abrasions should heal the same as one.

7. Attach a fan to your indirect.

8. In summer, wear scrubs and keep a fresh set in the freezer. In winter, wear a woolen Santa hat. Patients think that's cute.

9. Save money on shredding by burning old records in a HIPAA-compliant fireplace.

10. Keep a cooler full of water bottles (vodka in winter) nearby.

11. Get a machine that blows snow around the office. Don't tell anyone it's not fake.

12. Tell patients your HVAC system is permanently being serviced.

13. Make something up about saving the environment for your grandchildren.

14. Open windows. Nevermind, I forgot eye docs don't have windows.

15. Tell patients you give a 10% discount to those who don't gripe about being too hot or too cold.

16. If a patient starts sweating, say you are diagnosing them with swine flu and they need a second pair of glasses.

17. If a patient is chilly, talk about something they are passionate about, like their kids or their kid's most recent arrest.

18. Before calling the HVAC guys, remember they charge $600 an hour. That's more than you make. That's more than they made as ODs, too.

19. If a patient can't see through his new glasses, scrape the frost off.

20. Put a can of compressed electronic cleaner in your pocket and give yourself a spritz now and then.

21. Change the air filter at least once every career.

22. Install a large refrigerator in your reception area and stick a couple of chairs in there.

23. Allow patients to sit outside where it's a more comfortable 90°.

24. Have your mother sit in reception to chatter about the good ol' days before air conditioning.

25. When patients complain about their comfort, complain back about their vision plan.

You can overcome these heating and cooling challenges. Don't let them distract you from your primary mission: retiring. ■

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The Coding is in the Details

Using new technologies to screen for early disease can be great for patient care—as long as you use them wisely. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

Today's advances in posterior segment imaging are mind-boggling; just think of all the technologies we have at our fingertips to assist in the early diagnosis of potentially debilitating diseases.

While integrating imaging technology for screening purposes is fairly common in ophthalmic practices, the associated coding and billing can sometimes create practice management mayhem—communication gaps with patients, coverage issues, insufficient documentation, economic issues and, of course, significant risk exposure for audit.

Screening 101

By definition, screening is based upon the assumption that the person is not yet diagnosed with a disease and a broad, indiscriminate protocol will identify those who either have the disease, are at risk but do not have the disease or are not at risk and do not have the disease. For coding and third-party billing purposes, screening does not meet the requirements of medical necessity; therefore, the code generally used is not a CPT code, but a Level II HCPCS code: S9986 – Not Medically Necessary Service.

A few ground rules are important to keep in mind when using screening tests and the S9986 code:

1. You must communicate to the patient that the screening test is not medically necessary.

2. You must explain to the patient that they are financially responsible for the cost of the screening, even if you find pathology.*

3. The test should be done prior to the patient seeing the physician to avoid any implied medical necessity.

The S9986 code is quite broad in its application because it doesn't refer to a specific procedure, but rather a category of testing. For example, clinicians can use it to code a screening retinal image or a screening OCT, provided that in either case the screening image captured is different than the image that would be used to bill your fundus photograph (92250) or your OCT (92132, 92133, 92134). And here is where individuals create exposure.

Details Matter

Technological advances have created single instruments that can perform many types and levels of tests, and clinicians must be careful to choose the proper testing type and level.

For example, say a patient presents for a general exam as part of a managed care vision plan (MVCP). When given the option to have a pre-exam screening retinal image taken for a specific fee, the patient agrees. For these images, the instrument must be in the designated "screening mode."

During the subsequent exam, the physician notes an area of concern in the retina that deserves further photo documentation. The physician writes the order for the fundus photo based upon the medical necessity established during the exam.

This second test, performed using the same instrument, must provide additional information (i.e., stereo images) for the physician—information not present in the screening image—that aids in the patient's diagnosis, treatment and outcomes. This test can be performed on the same day as the screening or later.

The key is in the information provided. If the ordered images are the same as the screening or do not provide additional information, they should not be performed at all, as the primary tenet of medical necessity has not been met.

Assuming the burden of medical necessity was met and the test provided the additional information to qualify, the billing would be:

S9986 – payable by the patient
920X4 – copay as per MVCP
92015 – copay as per MVCP
92250 – billable to medical carrier, subject to specific co-pay/deductible guidelines

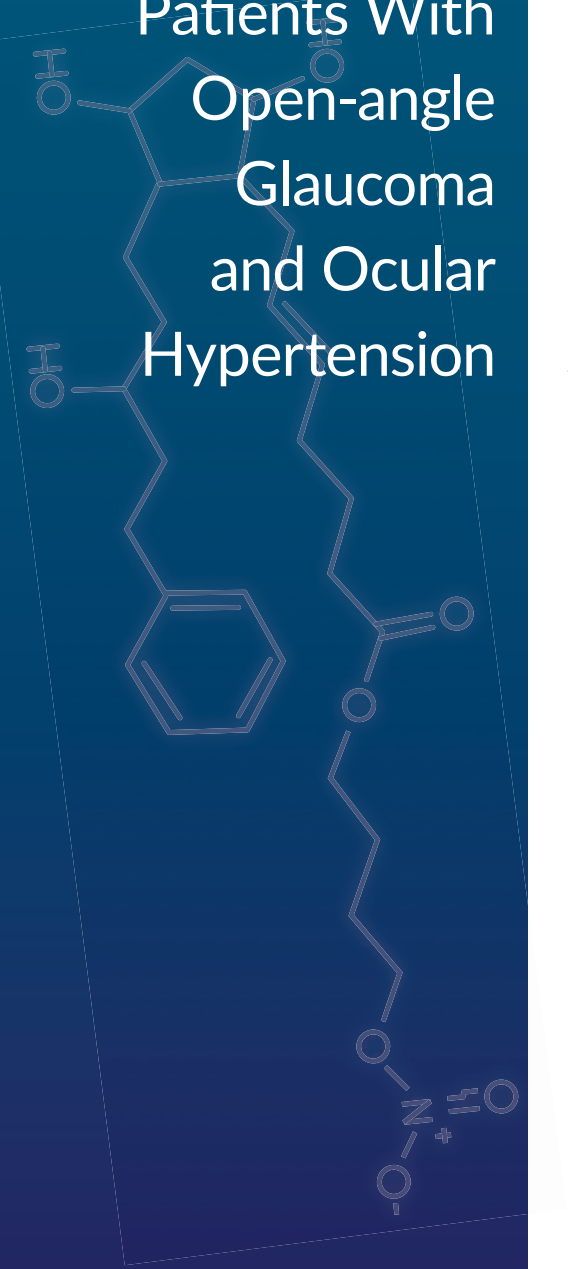
Just because the physician found pathology with the screening image doesn't mean the patient fee is waived, nor does it mean an additional photo is required; also, patients identified as having disease are not eligible for yearly screening.

Advanced screening technology provides a tremendous advantage when identifying early disease. Proper application of the guidelines surrounding screening and medical necessity can provide your patients with the care they deserve and success for your practice. ■

Send questions and comments to rocodingconnection@gmail.com.

* Some carriers are now including retinal screening photos as part of their structured benefit package. Please follow the specific carrier's policies in these situations.

Vyzulta™ (latanoprostene bunod ophthalmic solution), 0.024%: A New First-line Treatment Option for Patients With Open-angle Glaucoma and Ocular Hypertension



BEN GADDIE, OD, FAAO

The initial standard treatment for patients with open-angle glaucoma (OAG) is medical by tradition. Currently, topical prostaglandin analogs (PGAs) are the most widely prescribed IOP-lowering agents,[1] thanks to their tolerability and proven efficacy in lowering intraocular pressure (IOP). Other classes of topical IOP-lowering medications, including beta blockers, alpha agonists, and carbonic anhydrase inhibitors (CAIs), are commonly used as second-line therapy. Despite the wide spectrum of IOP-lowering medications, medical therapy of glaucoma often fails at lowering IOP to within target levels. The approval of Vyzulta™ (latanoprostene bunod ophthalmic solution), 0.024%, a PGA that releases nitric oxide (NO), provides a new first-line treatment option for patients with OAG or ocular hypertension. Upon topical ocular administration, Vyzulta is metabolized into two moieties: the first, latanoprost acid, is a prostaglandin F2α analog, while the second, butanediol mononitrate, releases NO. Vyzulta is thought to lower IOP by increasing aqueous humor outflow through both the trabecular and uveoscleral routes. Having demonstrated potent and durable IOP-lowering efficacy and an acceptable safety profile in clinical studies, Vyzulta holds great promise for the medical management of glaucoma patients.

INDICATION

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% is a prostaglandin F2α analog indicated for the reduction of intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

- Increased pigmentation of the iris and periorbital tissue (eyelid) can occur. Iris pigmentation is likely to be permanent
- Gradual changes to eyelashes, including increased length, increased thickness, and number of eyelashes, may occur. These changes are usually reversible upon treatment discontinuation
- Use with caution in patients with a history of intraocular inflammation (iritis/uveitis). VYZULTA should generally not be used in patients with active intraocular inflammation
- Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. Use with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema
- There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products that were inadvertently contaminated by patients
- Contact lenses should be removed prior to the administration of VYZULTA and may be reinserted 15 minutes after administration
- Most common ocular adverse reactions with incidence ≥2% are conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%)

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Elevated intraocular pressure (IOP) is a major modifiable risk factor for glaucoma progression.[2-7] In patients with primary open-angle glaucoma (POAG) or ocular hypertension, chronic cellular contraction and increased extracellular matrix deposition within the trabecular (ie, conventional) outflow pathway result in reduced aqueous humor outflow, which causes IOP elevation.[8-10] It is believed that high IOP levels cause progressive damage to the optic nerve, resulting in vision loss.[8] While glaucoma therapy has evolved to include various treatment modalities, reduction of IOP has remained its cornerstone. Large multicenter studies have shown that the reduction of IOP reduces the risk of glaucomatous visual field loss.[2-7]

Today, the armamentarium of glaucoma medications comprises four major classes of IOP-lowering agents: prostaglandin analogs (PGAs), beta blockers, alpha-agonists, and carbonic anhydrase inhibitors (CAIs).[11] Despite multiple drug choices, however, many glaucoma patients do not reach target IOP with a single-agent regimen.[11-12] Even if pressure is maintained within target levels, some patients may continue to develop progressive glaucoma damage and field loss. These treatment challenges highlight a continued need for additional effective therapies.

STANDARD FIRST-LINE THERAPY

In my practice, the preferred choice for initial glaucoma therapy is a PGA. One major advantage of this class of drugs is that they produce an IOP reduction of about 30%,[13-16] by increasing uveoscleral (ie, nonconventional) aqueous outflow. In the uveoscleral pathway, aqueous humor outflow occurs by diffusion through the interstitial spaces of the ciliary muscle.[8,17] PGAs are believed to work primarily within this pathway by upregulating matrix metalloproteinase activity. This increases the interstitial spaces between the ciliary muscle bundles to allow for greater aqueous humor outflow.[8,18]

What supports the use of PGAs as first-line monotherapy for glaucoma is not just their IOP-lowering efficacy but also their safety profile.[8,9,11] Furthermore, once-a-day administration of the PGAs can provide round-the-clock IOP control. At the time they first became available, this once-daily dosing regimen was revolutionary in the treatment of glaucoma.

THE TARGET PRESSURE

While it is difficult to accurately predict what IOP level is low enough for the individual patient, most practitioners use an estimated "target" pressure as a general guideline for treatment.

The target pressure should not be a single number or range set in stone; rather, it is a clinical estimate that needs to be constantly adjusted in accordance with state of the disease.

For the most part, I determine how low I should aim to reduce IOP by disease severity. My treatment goal typically is to lower the pressure by 30% for patients that have ocular hypertension or mild glaucoma and by 40 or 50% for those with moderate to severe glaucoma. Given that the PGAs produce a 28% to 35% reduction of pressure,[14-15,19] I would expect at least a 30% reduction in IOP with PGA monotherapy—regardless of disease severity in patients with glaucoma.

A DUAL-ACTION MOLECULE

Vyzulta™ (latanoprostene bunod ophthalmic solution), 0.024%, a nitric oxide (NO)-releasing prostaglandin F2α receptor agonist indicated for the reduction of IOP in patients with OAG or ocular hypertension, is the first novel IOP-lowering drug the US FDA has approved since the introduction of PGAs nearly two decades ago. Vyzulta is thought to lower IOP by increasing aqueous humor outflow through both the trabecular and uveoscleral routes,[20-22] achieved by chemically fusing two moieties—latanoprost and butanediol mononitrate, which releases NO—into one molecule, creating a dual-action drug. (Figure 1).

When instilled in the eye, Vyzulta™ is rapidly metabolized to latanoprost acid, a PGA, and butanediol mononitrate, which releases NO; both moieties are active and responsible for the molecule's pharmacological activities.[21] While latanoprost acid increases uveoscleral outflow, the release of NO is believed to contribute to IOP lowering by increasing aqueous outflow through the trabecular meshwork (TM).[22] The trabecular pathway is the primary route of aqueous outflow in the human eye and the site of extra resistance that results in elevated pressure in POAG.[23]

Preclinical studies provide support for the hypothesis that NO increases outflow through the trabecular pathway.[24-28] To increase trabecular outflow, NO induces cell relaxation in the TM by activating the NO-soluble guanylate cyclase–cyclic guanosine-3',5'-monophosphate (NO-sGC-cGMP) signaling pathway. This leads to a widening of intercellular spaces in the TM, thus increasing aqueous humor outflow.[24,25,29,30] Because the majority of aqueous humor outflow occurs through the trabecular pathway, this process plays an increasingly recognized role in regulating IOP.[8-9]

Studies have found reduced levels of NO markers in the anterior chambers of the eyes of patients with POAG, providing further evidence for the potential therapeutic value of NO-releasing molecules for patients with this disease.[31-33]

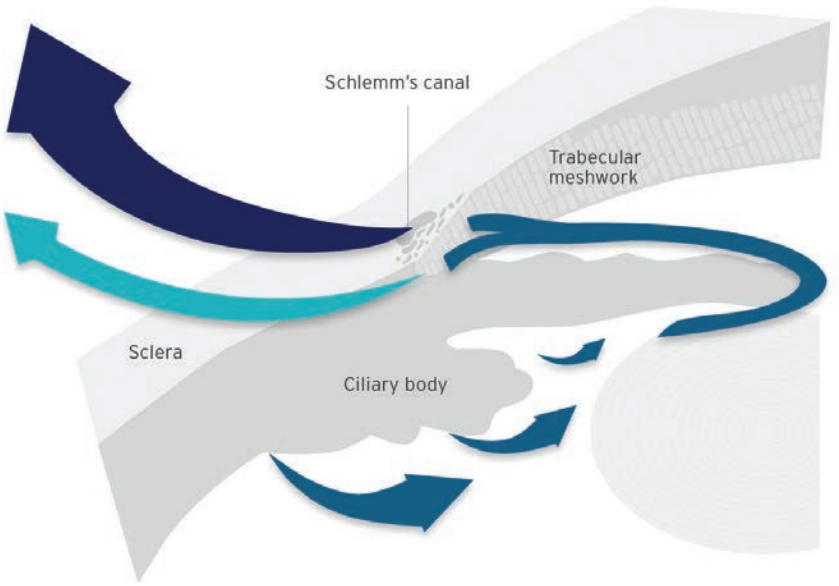

FIGURE 1 Two pathways for aqueous outflow in the eye. Vyzulta™ increases uveoscleral outflow through latanoprost acid and trabecular outflow through NO.

A NEW OPTION

Vyzulta™ should have a definite place in the initial treatment of a patient, where the general goal is to achieve successful IOP control with a single agent.

In clinical studies of up to 12 months' duration in patients with OAG or ocular hypertension, the IOP-lowering effect of Vyzulta™ once daily ranged from 7.5 to 9.1 mm Hg.[20,35,36] The two pivotal phase 3 clinical trials—APOLLO and LUNAR—involved a total of 831 patients with OAG or ocular hypertension. IOP reduction with Vyzulta dosed once daily at night was non-inferior to that with timolol 0.5% dosed twice daily and was superior to timolol 0.5% for IOP lowering dosed twice daily in the combined intent-to-treat population (Table 1).[35,36] The most common ocular adverse reactions observed in patients treated with Vyzulta were conjunctival hyperemia (5.8%), eye irritation (4.3%), eye pain (3.1%), and instillation site pain (2.1%).[20]

In the JUPITER study, a phase 3 single-arm, multicenter, open-label clinical trial, Vyzulta™ produced robust, stable IOP reduction over a 1-year treatment period in 130 Japanese patients with a predominance of normal-tension glaucoma.[37] The study and treated fellow eyes achieved mean IOP reductions from baseline of 22.0% and 19.5% by week 4, respectively, and maintained those reductions through week 52.

In VOYAGER, a phase 2, 28-day dose-ranging comparison study of subjects with OAG or ocular hypertension, Vyzulta™ led to a 9 mm Hg decrease from baseline—an additional IOP reduction of 1.23 mm Hg over latanoprost 0.005% ($P = 0.005$).[34] Among the latanoprostene bunod groups, IOP

TABLE I Vyzulta™: Non-inferior to Timolol at All Tested Timepoints (Primary Endpoint, Data Not Shown) and Superior to Timolol at Month 3

APOLLO (Study 769)					
	Vyzulta™		Timolol 0.5%		
Month 3	mm Hg IOP at timepoints	Reduction from baseline mm Hg	mm Hg IOP at timepoints	Reduction from baseline mm Hg	*P* value
Baseline	**26.7**	—	**26.5**	—	—
8AM	**18.7**	–8	**19.7**	–6.8	0.002
12 PM	**17.9**	–8.8	**19.2**	–7.3	< 0.001
4 PM	**17.8**	–8.9	**19.2**	–7.3	< 0.001
LUNAR (Study 770)					
	Vyzulta™		Timolol 0.5%		
Month 3	mm Hg IOP at timepoints	Reduction from baseline mm Hg	mm Hg IOP at timepoints	Reduction from baseline mm Hg	*P* value
Baseline	**26.6**	—	**26.4**	—	—
8AM	**18.7**	–7.9	**19.6**	–6.8	0.006
12 PM	**17.9**	–8.7	**19.2**	–7.4	< 0.001
4 PM	**17.7**	–8.9	**19.1**	–7.3	< 0.001

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reductions were dose-dependent, with maximal response at 0.024%-0.040%. Ocular tolerability (ocular discomfort, burning sensation, pain, tearing and blurring of vision) was similar between the Vyzulta and latanoprost groups.[34]

Vyzulta™ has been shown to be effective at reducing IOP, and it has the potential to replace the current PGAs as the first go-to drug for patients with OAG or ocular hypertension. We can now choose to substitute Vyzulta, which is one molecule with two mechanisms of action, for the primary PGA.

When having discussions with patients on Vyzulta™, I would explain that patients with glaucoma have been shown to have evidence of less endogenous NO than patients without glaucoma. Vyzulta lowers IOP through latanoprost acid, which increases uveoscleral outflow, and the release of NO, which is thought to increase conventional outflow. Given its strong IOP-lowering effects, Vyzulta should hold strategic importance to medical management of glaucoma.

Ben Gaddie, OD, FAAO, practices at Gaddie Eye Centers in Louisville, KY. He is executive vice president of the Optometric Glaucoma Society and co-chairman of the Vision Expo meetings.

REFERENCES

1. Symphony National. Total Glaucoma Market. July 2016-June 2017.
2. Garway-Heath DF, Crabb DP, Bunce C, et al. Latanoprost for open-angle glaucoma (UKGTS): a randomised, multicentre, placebo-controlled trial. *Lancet*. 2015;385(9975):1295-304.
3. Leske MC, Heijl A, Hussein M, et al; Early Manifest Glaucoma Trial Group. Factors for glaucoma progression and the effect of treatment: the early manifest glaucoma trial. *Arch Ophthalmol*. 2003;121(1):48-56.
4. Kass MA, Heuer DK, Higginbotham EJ, et al. The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. *Arch Ophthalmol*. 2002;120(6):701-13.
5. Heijl A, Leske C, Bengtsson B, et al. Reduction of intraocular pressure and glaucoma progression: Results from the Early Manifest Glaucoma Trial. *Arch Ophthalmol*. 2002;120(10):1268-79.
6. The AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. *Am J Ophthalmol*. 2000;130(4):429-40.
7. Collaborative Normal-Tension Glaucoma Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. *Am J Ophthalmol*. 1998;126(4):487-97.
8. Braunger BM, Fuchshofer R, Tamm ER. The aqueous humor outflow pathways in glaucoma: A unifying concept of disease mechanisms and causative treatment. *Eur J Pharm Biopharm*. 2015;95(Pt B):173-81.
9. Tamm ER, Braunger BM, Fuchshofer R. Intraocular pressure and the mechanisms involved in resistance of the aqueous humor flow in the trabecular meshwork outflow pathways. *Prog Mol Biol Transl Sci*. 2015;134:301-14.
10. Stamer WD. The cell and molecular biology of glaucoma: mechanisms in the conventional outflow pathway. *Invest Ophthalmol Vis Sci*. 2012;53:2470-2.
11. Aptel F, Chiquet C, Romanet JP. Intraocular pressure-lowering combination therapies with prostaglandin analogues. *Drugs*. 2012;72(10):1355-71.
12. Schmier JK, Hulme-Lowe CK, Covert DW. Adjunctive therapy patterns in glaucoma patients using prostaglandin analogs. *Clin Ophthalmol*. 2014;8:1097-104.
13. Alm A, Stjernschantz J. Effects on intraocular pressure and side effects of 0.005% latanoprost applied once daily, evening or morning. A comparison with timolol. Scandinavian Latanoprost Study Group. *Ophthalmology*. 1995;102(12):1743-52.
14. Higginbotham EJ, Schuman JS, Goldberg I, et al; Bimatoprost Study Groups 1 and 2. One-year, randomized study comparing bimatoprost and timolol in glaucoma and ocular hypertension. *Arch Ophthalmol*. 2002;120(10):1286-93.
15. Netland PA, Landry T, Sullivan EK, et al; Travoprost Study Group. Travoprost compared with latanoprost and timolol in patients with open-angle glaucoma or ocular hypertension. *Am J Ophthalmol*. 2001;132(4):472-84.
16. Schachtschabel U, Lindsey JD, Weinreb RN. The mechanism of action of prostaglandins in uveoscleral outflow. *Curr Opin Ophthalmol*. 2000;11:112-5.
17. Winkler NS, Fautsch MP. Effects of prostaglandin analogues on aqueous humor outflow pathways. *J Ocul Pharmacol Ther*. 2014;30(2-3):102-9.
18. Weinreb RN, Khaw PT. Primary open-angle glaucoma. *Lancet*. 2004;363:1711-20.
19. Camras CB. Comparison of latanoprost and timolol in patients with ocular hypertension and glaucoma: a six-month masked, multicenter trial in the United States. The United States Latanoprost Study Group. *Ophthalmology*. 1996;103(1):138-47.
20. VYZULTA Prescribing Information. Bausch + Lomb, Inc. MONTH 2017.
21. Krauss AH, Impagnatiello F, Toris CB, et al. Ocular hypotensive activity of BOL-303259-X, a nitric oxide donating prostaglandin F2a agonist, in preclinical models. *Exp Eye Res*. 2011;93:250-5.
22. Cavet ME, Vollmer TR, Harrington KL, et al. Regulation of Endothelin-1-Induced Trabecular Meshwork Cell Contractility by Latanoprostene Bunod. *Invest Ophthalmol Vis Sci*. 2015;56(6):4108-16.
23. Stamer WD, Acott TS. Current understanding of conventional outflow dysfunction in glaucoma. *Curr Opin Ophthalmol*. 2012;23:135-43.
24. Schneemann A, Dijkstra BG, van den Berg TJ, et al. Nitric oxide/guanylate cyclase pathways and flow in anterior segment perfusion. *Graefes Arch Clin Exp Ophthalmol*. 2002;240:936-41.
25. Wiederholt M, Sturm A, Lepple-Wienhues A. Relaxation of trabecular meshwork and ciliary muscle by release of nitric oxide. *Invest Ophthalmol Vis Sci*. 1994;35:2515-20.
26. Nathanson JA. Nitrovasodilators as a new class of ocular hypotensive agents. *J Pharmacol Exp Ther*. 1992;260(3):956-65.
27. Schuman JS, Erickson K, Nathanson JA. Nitrovasodilator effects on intraocular pressure and outflow facility in monkeys. *Exp Eye Res*. 1994;58:99-105.
28. Heyne GW, Kiland JA, Kaufman PL, Gabelt BT. Effect of nitric oxide on anterior segment physiology in monkeys. *Invest Ophthalmol Vis Sci*. 2013;54(7):5103-10.
29. Cavet ME, Vittitow JL, Impagnatiello F, et al. Nitric oxide (NO): an emerging target for the treatment of glaucoma. *Invest Ophthalmol Vis Sci*. 2014;55:5005-15.
30. Buys ES, Potter LR, Pasquale LR, et al. Regulation of intraocular pressure by soluble and membrane guanylate cyclases and their role in glaucoma. *Front Mol Neurosci*. 2014;7:38.
31. Galassi F, Renieri G, Sodi A, et al. Nitric oxide proxies and ocular perfusion pressure in primary open angle glaucoma. *Br J Ophthalmol*. 2004;88:757-60.
32. Doganay S, Evereklioglu C, Turkoz Y, et al. Decreased nitric oxide production in primary open-angle glaucoma. *Eur J Ophthalmol*. 2002;12:44-8.
33. Nathanson JA, McKee M. Alterations of ocular nitric oxide synthase in human glaucoma. *Invest Ophthalmol Vis Sci*. 1995;36:1774-84.
34. Weinreb RN, Ong T, Scassellati Sforzolini B, et al; VOYAGER study group. A randomised, controlled comparison of latanoprostene bunod and latanoprost 0.005% in the treatment of ocular hypertension and open angle glaucoma: the VOYAGER study. *Br J Ophthalmol*. 2015;99(6):738-45.
35. Weinreb RN, Scassellati Sforzolini B, Vittitow J, et al. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: The APOLLO study. *Ophthalmology*. 2016;123(5):965-73.
36. Medeiros FA, Martin KR, Peace J, et al. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. *Am J Ophthalmol*. 2016;168:250-9.
37. Kawase K, Vittitow JL, Weinreb RN, Araie M; JUPITER Study Group. Long-term Safety and Efficacy of Latanoprostene Bunod 0.024% in Japanese Subjects with Open-Angle Glaucoma or Ocular Hypertension: The JUPITER Study. *Adv Ther*. 2016;33(9):1612-27.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use VYZULTA safely and effectively. See full Prescribing Information for VYZULTA.

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024%, for topical ophthalmic use.

Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE

VYZULTA™ (latanoprostene bunod ophthalmic solution) 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% may cause changes to pigmented tissues. The most frequently reported changes with prostaglandin analogs have been increased pigmentation of the iris and periorbital tissue (eyelid).

Pigmentation is expected to increase as long as latanoprostene bunod ophthalmic solution is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of VYZULTA, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes are likely to be reversible in most patients. Patients who receive prostaglandin analogs, including VYZULTA, should be informed of the possibility of increased pigmentation, including permanent changes. The long-term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly *[see Patient Counseling Information (17) in full Prescribing Information]*.

5.2 Eyelash Changes

VYZULTA may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and the number of lashes or hairs. Eyelash changes are usually reversible upon discontinuation of treatment.

5.3 Intraocular Inflammation

VYZULTA should be used with caution in patients with a history of intraocular inflammation (iritis/uveitis) and should generally not be used in patients with active intraocular inflammation as it may exacerbate this condition.

5.4 Macular Edema

Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. VYZULTA should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

5.5 Bacterial Keratitis

There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

5.6 Use with Contact Lens

Contact lenses should be removed prior to the administration of VYZULTA because this product contains benzalkonium chloride. Lenses may be reinserted 15 minutes after administration.

6 ADVERSE REACTIONS

The following adverse reactions are described in the Warnings and Precautions section: pigmentation (5.1), eyelash changes (5.2), intraocular inflammation (5.3), macular edema (5.4), bacterial keratitis (5.5), use with contact lens (5.6).

6.1 Clinical Trials Experience

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

VYZULTA was evaluated in 811 patients in 2 controlled clinical trials of up to 12 months duration. The most common ocular adverse reactions observed in patients treated with latanoprostene bunod were: conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%). Approximately 0.6% of patients discontinued therapy due to ocular adverse reactions including ocular hyperemia, conjunctival irritation, eye irritation, eye pain, conjunctival edema, vision blurred, punctate keratitis and foreign body sensation.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available human data for the use of VYZULTA during pregnancy to inform any drug associated risks.

Latanoprostene bunod has caused miscarriages, abortion, and fetal harm in rabbits. Latanoprostene bunod was shown to be abortifacient and teratogenic when administered intravenously (IV) to pregnant rabbits at exposures ≥ 0.28 times the clinical dose. Doses ≥ 20 µg/kg/day (23 times the clinical dose) produced 100% embryofetal lethality. Structural abnormalities observed in rabbit fetuses included anomalies of the great vessels and aortic arch vessels, domed head, sternebral and vertebral skeletal anomalies, limb hyperextension and malrotation, abdominal distension and edema. Latanoprostene bunod was not teratogenic in the rat when administered IV at 150 mcg/kg/day (87 times the clinical dose) *[see Data]*.

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 latanoprostene bunod daily by intravenous injection on gestation days 7 through 19, to target the period of organogenesis. The doses administered ranged from 0.24 to 80 mcg/kg/day. Abortion occurred at doses ≥ 0.24 mcg/kg/day latanoprostene bunod (0.28 times the clinical dose, on a body surface area basis, assuming 100% absorption). Embryofetal lethality (resorption) was increased in latanoprostene bunod treatment groups, as evidenced by increases in early resorptions at doses ≥ 0.24 mcg/kg/day and late resorptions at doses ≥ 6 mcg/kg/day (approximately 7 times the clinical dose). No fetuses survived in any rabbit pregnancy at doses of 20 mcg/kg/day (23 times the clinical dose) or greater. Latanoprostene bunod produced structural abnormalities at doses ≥ 0.24 mcg/kg/day (0.28 times the clinical dose). Malformations included anomalies of sternum, coarctation of the aorta with pulmonary trunk dilation, retroesophageal subclavian artery with absent brachiocephalic artery, domed head, forepaw hyperextension and hindlimb malrotation, abdominal distention/edema, and missing/fused caudal vertebrae.

An embryofetal study was conducted in pregnant rats administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 17, to target the period of organogenesis. The doses administered ranged from 150 to 1500 mcg/kg/day. Maternal toxicity was produced at 1500 mcg/kg/day (870 times the clinical dose, on a body surface area basis, assuming 100% absorption), as evidenced by reduced maternal weight gain. Embryofetal lethality (resorption and fetal death) and structural anomalies were produced at doses ≥ 300 mcg/kg/day (174 times the clinical dose). Malformations included anomalies of the sternum, domed head, forepaw hyperextension and hindlimb malrotation, vertebral anomalies and delayed ossification of distal limb bones. A no observed adverse effect level (NOAEL) was established at 150 mcg/kg/day (87 times the clinical dose) in this study.

8.2 Lactation

Risk Summary

There are no data on the presence of VYZULTA 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 VYZULTA, and any potential adverse effects on the breastfed infant from VYZULTA.

8.4 Pediatric Use

Use in pediatric patients aged 16 years and younger is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

8.5 Geriatric Use

No overall clinical differences in safety or effectiveness have been observed between elderly and other adult patients.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Latanoprostene bunod was not mutagenic in bacteria and did not induce micronuclei formation in the *in vivo* rat bone marrow micronucleus assay. Chromosomal aberrations were observed *in vitro* with human lymphocytes in the absence of metabolic activation.

Latanoprostene bunod has not been tested for carcinogenic activity in long-term animal studies. Latanoprost acid is a main metabolite of latanoprostene bunod. Exposure of rats and mice to latanoprost acid, resulting from oral dosing with latanoprost in lifetime rodent bioassays, was not carcinogenic.

Fertility studies have not been conducted with latanoprostene bunod. The potential to impact fertility can be partially characterized by exposure to latanoprost acid, a common metabolite of both latanoprostene bunod and latanoprost. Latanoprost acid has not been found to have any effect on male or female fertility in animal studies.

13.2 Animal Toxicology and/or Pharmacology

A 9-month toxicology study administered topical ocular doses of latanoprostene bunod to one eye of cynomolgus monkeys: control (vehicle only), one drop of 0.024% bid, one drop of 0.04% bid and two drops of 0.04% per dose, bid. The systemic exposures are equivalent to 4.2-fold, 7.9-fold, and 13.5-fold the clinical dose, respectively, on a body surface area basis (assuming 100% absorption). Microscopic evaluation of the lungs after 9 months observed pleural/subpleural chronic fibrosis/inflammation in the 0.04% dose male groups, with increasing incidence and severity compared to controls. Lung toxicity was not observed at the 0.024% dose.

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U.S. Patent Numbers: 6,211,233; 7,273,946; 7,629,345; 7,910,767; 8,058,467.

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Where's the Degeneration?

A significant case of subretinal hemorrhaging leads one OD to a rare disease.

Edited by Paul C. Ajamian, OD

Q I have a patient with a subretinal neovascular membrane with a bleed. However, the other eye shows no signs of macular degeneration. What else should I consider?

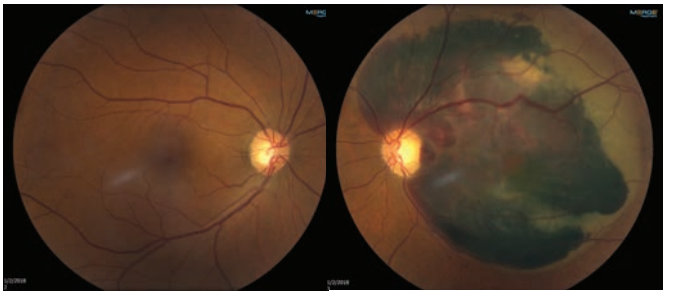
A A 73-year-old male presented to Nash Ditmetaroj, OD, of Kaiser Permanente in Atlanta, with a "spot" in his central vision OS, which had persisted for two weeks. Its onset was sudden and had not really changed since it first appeared. His right eye was unaffected.

History included hypertension (HTN), with well-controlled blood pressure. At the time of exam, blood pressure was 124/70, which he stated was typical for him. His meds included Coumadin (warfarin, Bristol-Myers Squibb) 5mg daily, losartan 50mg BID for HTN and low-dose aspirin (81mg) QID. Vision was 20/25-2 best corrected OD and hand motion OS with no improvement with refraction.

Dilation revealed a normal anterior segment aside from some early nuclear sclerosis OU. The right fundus was normal. The left eye had a significant subretinal hemorrhage extending throughout the posterior pole with a slightly irregular, elevated area just temporal to the optic nerve.

"My initial consideration was age-related macular degeneration (AMD) due to the location of the hemorrhage," says Dr. Ditmetaroj. "AMD made sense given the patient's age, but the unilateral presentation didn't. I was looking for other entities in my differential diagnosis."

Another consideration was a choroidal melanoma with choroidal neovascularization (CNV). Although rare, these cases can cause subretinal fluid or hemorrhaging, says Dr. Dimetaroj. "Typically, melanomas will present with elevated lesions, and that was not seen in my patient."



The patient's left eye (at right) shows subretinal hemorrhaging, but the right eye shows no signs of macular degeneration.

The Diagnosis

Despite the many considerations, closer evaluation led Dr. Ditmetaroj to diagnose polypoidal choroidal vasculopathy (PCV) due to polyp-shaped irregularities just temporal to the optic nerve. "The extensive hemorrhaging and significant elevation is more common in PCV than in AMD," says Dr. Ditmetaroj.

PCV is a disease of the choroidal vessels that can result in vascularized retinal pigment epithelium detachments (PEDs), CNV and subretinal fibrosis, says Mohammad Rafieetary, OD, of Charles Retina Institute in Germantown, Tenn. "Although PCV can be present in both men and woman of different ethnicity in the United States, the typical patient is a middle-aged African American female."

According to Dr. Rafieetary, the most common misdiagnosis of PCV is AMD, but it is possible for both conditions to be present in the same patient. Indocyanine green angiography is the decisive diagnostic test; it will highlight the polyp-like branching of choroidal vasculature, he says.

PCV's pathophysiology is unclear, but Dr. Rafieetary says systemic hypertension may play a role in PCV complications such as subretinal bleeding. Additionally, many PCV patients may be asymptomatic and will only be diagnosed on routine examination. "Patients presenting with symptoms complain of blurred and distorted vision as well as central or paracentral scotomas associated with the presence of subretinal fluid or hemorrhage," notes Dr. Rafieetary.

Anti-VEGF injections and photodynamic therapy are "the most common and effective therapies to minimize permanent central vision loss," Dr. Rafieetary says.

In this case, Dr. Ditmetaroj asked the patient to return the next day for evaluation by a retinal specialist. When he returned, the findings were unchanged, the diagnosis confirmed and the patient educated and monitored monthly. ■

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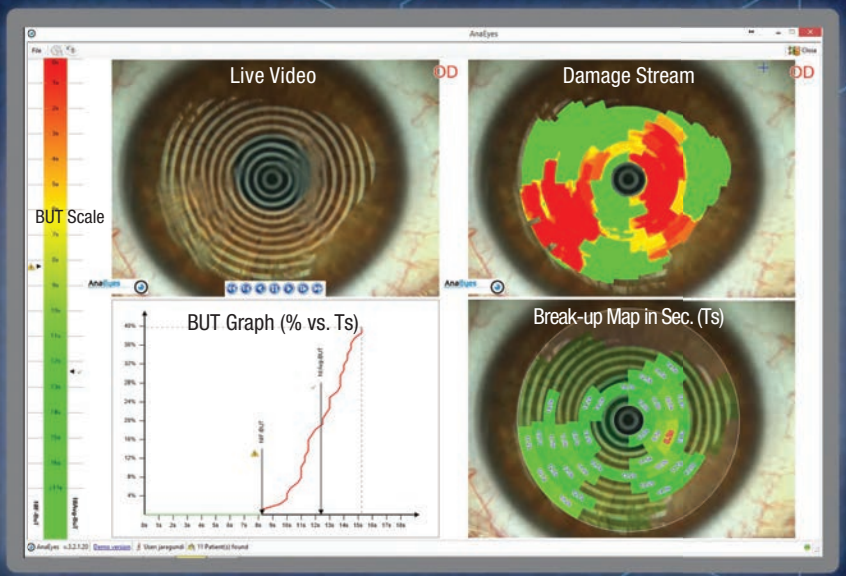

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A Giant Problem Overlooked

Don't always attribute new patient complaints to age—especially if it could be giant cell arteritis.

By Michael Trottini, OD, and Michael DelGiodice, OD

An 85-year-old Caucasian female presented with acute vision loss in both eyes. She stated that two days ago she developed pain on the left side of her head with vision loss in the left eye, which shortly spread to the right eye. She reported fatigue, weakness, jaw pain/claudication, scalp tenderness and pains around her head and the back of her neck for the past few months.

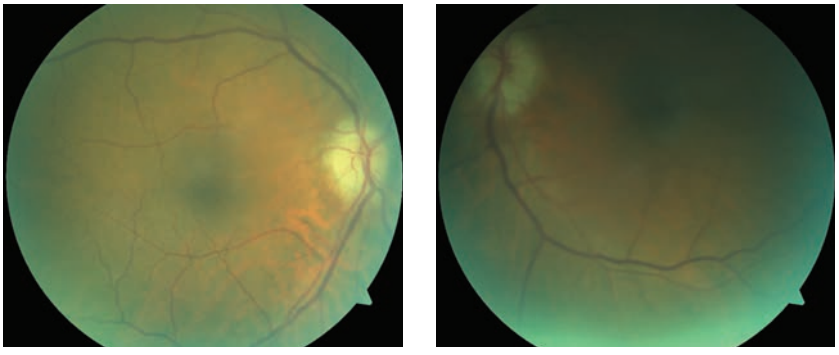
Her visual acuity was measured at hand motion in both eyes. Intraocular pressures were 10mm Hg in each eye. Anterior segment exam showed cataracts in both eyes; however, only three months prior she had best-corrected visual acuities of 20/30 OD and 20/40 OS. Posterior segment exam showed bilateral pallid disc edema.

Her retinal exam was otherwise unremarkable.

I (Dr. Trottini) called her internist—whom she had seen recently due to her general complaints of aches, pains and fatigue—and inquired about any recent erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) test results. There was no recent ESR, but a CRP from two weeks prior was 36.1mg/L (the normal range is <3mg/L); however, no additional testing or work up was performed based on this elevated lab study.

Diagnosis and Treatment

I consequently sent her to the emergency department and let the attending physician know that her likely diagnosis was bilateral arteritic ischemic optic neuropathy (AION) from giant cell arteritis (GCA). I recommended an ESR, CRP and temporal artery biopsy as well as rheumatology consultation. I also recommended neuroimaging to rule out any intracranial process, and results were normal.



Although our patient's optic nerve photos were compromised due to her cataracts and imperfect fixation, they show pallid disc edema, which is characteristic of GCA/AION.

Her ESR was >140mm/hr and CRP was 33mg/L in the emergency department. These lab values, along with the normal neuroimaging, exam findings and other patient symptoms, were all consistent with GCA, and the patient was admitted and started on intravenous Solu-medrol (methylprednisolone sodium succinate, Pfizer) 1g daily for the next three days.

A temporal artery biopsy was performed the next day, which was positive and confirmed GCA. The patient finished the three-day course of Solu-medrol and was started on 80mg of prednisone, which will be tapered accordingly by rheumatology.

One week later on follow-up exam, although she reported almost immediate relief of her aches, pains and fatigue since starting the steroids, her disc edema looked slightly worse compared with the previous visit, and her vision had worsened to no light perception (NLP) in both eyes. She continued her current dose of prednisone and saw rheumatology for GCA management.

On repeat examination two weeks later, she had moderate improvement in her optic nerve appearance. The discs were well perfused again, and the pallid edema was essentially resolved. Unfortunately, she remained NLP in both eyes and has not regained any of her sight over time.

Discussion

GCA is one of the few serious emergent disorders eye care providers encounter. It is a granulomatous inflammatory vasculopathy affecting medium and large size

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Indications and Usage

BromSite® (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

Recommended Dosing

One drop of BromSite® should be applied to the affected eye twice daily (morning and evening) 1 day prior to surgery, the day of surgery, and 14 days postsurgery.

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite®, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite®. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite®, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery. It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- **Keratitis and Corneal Effects:** Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite®, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.
- **Contact Lens Wear:** BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.
- **Adverse Reactions:** The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

Please see brief summary of Full Prescribing Information on the adjacent page.

NSAID=nonsteroidal anti-inflammatory drug.

Reference: 1. BromSite® [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016.

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SUN OPHTHALMICS

BromSite® (bromfenac ophthalmic solution) 0.075%
Brief Summary

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

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Slow or Delayed Healing
All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite® (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.

Potential for Cross-Sensitivity
There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.

Increased Bleeding Time of Ocular Tissue
With some NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.

Keratitis and Corneal Reactions
Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. These events may be sight threatening. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health.

Post-marketing experience with topical NSAIDs suggests that patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients.

Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

Contact Lens Wear
BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.

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

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

The most commonly reported adverse reactions in 1–8% of patients were: anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain and ocular hypertension.

USE IN SPECIFIC POPULATIONS
Pregnancy
Risk Summary
There are no adequate and well-controlled studies in pregnant women to inform any drug associated risks. Treatment of pregnant rats and rabbits with oral bromfenac did not produce teratogenic effects at clinically relevant doses.

Clinical Considerations
Because of the known effects of prostaglandin biosynthesis-inhibiting drugs on the fetal cardiovascular system (closure of ductus arteriosus), the use of BromSite® during late pregnancy should be avoided.

Data
Animal Data
Treatment of rats with bromfenac at oral doses up to 0.9 mg/kg/day (195 times a unilateral daily human ophthalmic dose on a mg/m^2 basis, assuming 100% absorbed) and rabbits at oral doses up to 7.5 mg/kg/day (3243 times a unilateral daily dose on a mg/m^2 basis) produced no structural teratogenicity in reproduction studies. However, embryo-fetal lethality, neonatal mortality and reduced postnatal growth were produced in rats at 0.9 mg/kg/day, and embryo-fetal lethality was produced in rabbits at 7.5 mg/kg/day. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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

Pediatric Use
Safety and efficacy in pediatric patients below the age of 18 years have not been established.

Geriatric Use
There is no evidence that the efficacy or safety profiles for BromSite® differ in patients 65 years of age and older compared to younger adult patients.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis and Impairment of Fertility
Long-term carcinogenicity studies in rats and mice given oral doses of bromfenac up to 0.6 mg/kg/day (129 times a unilateral daily dose assuming 100% absorbed, on a mg/m^2 basis) and 5 mg/kg/day (540 times a unilateral daily dose on a mg/m^2 basis), respectively revealed no significant increases in tumor incidence.

Bromfenac did not show mutagenic potential in various mutagenicity studies, including the bacterial reverse mutation, chromosomal aberration, and micronucleus tests.

Bromfenac did not impair fertility when administered orally to male and female rats at doses up to 0.9 mg/kg/day and 0.3 mg/kg/day, respectively (195 and 65 times a unilateral daily dose, respectively, on a mg/m^2 basis).

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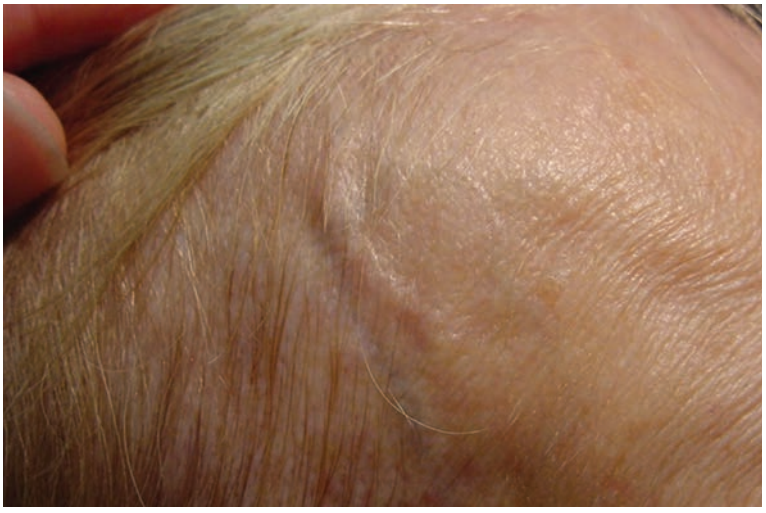
SUN OPHTHALMICS

arteries. The commonly affected vessels include the external carotid branches, ophthalmic, vertebral, distal subclavian and the thoracic aorta.

GCA is a disorder affecting an older population (usually patients >50) and females have a higher incidence compared with males.[1]

The main concern from an OD's standpoint with GCA is the development of arteritic ischemic optic neuropathy (AION). As the blood vessels supplying the optic nerve become inflamed, luminal occlusion occurs, which leads to ischemic complications.[1] The goal is to recognize and treat GCA before AION occurs.



The large, palpable, firm temporal artery in another patient is a good representation of what the temporal artery can sometimes look like in GCA. Not surprisingly, this patient's biopsy was positive.

Typical presenting symptoms of GCA include headache, temple pain, neck pain, scalp tenderness, jaw claudication, weakness and tiredness/fatigue. The temporal artery may be firm and tender on palpation and often nonpulsatile.

With AION, vision loss is usually severe and responds poorly to treatment, as seen in our patient. Presenting vision loss is generally much worse when compared with vision loss from nonarteritic ischemic optic neuropahty (NAION). For example, one study showed initial visual acuity of count fingers to no light perception in 54% of AION patients compared with only 14% of NAION patients.[2] Amaurosis fugax will be present in about one-third of patients and is a sign of impending AION.[2] Additional ocular findings from GCA can include sixth nerve palsy, cotton wool spots and retinal artery occlusion. The disc edema from GCA/AION generally has a characteristic pale edema or chalky white color.[2]

This case highlights just how visually devastating GCA can be and the importance of recognizing the symptoms and clinical findings as early as possible.

If steroid therapy is started early in the course of GCA, vision loss typically does not occur. If one eye develops vision loss, the goal is to start treatment to prevent further vision loss in that eye and prevent it in the fellow eye.

When suspected, ESR and CRP should be ordered, as these tests will be elevated due to the presence of inflammation within the body. Further verification of GCA is obtained either by temporal artery biopsy or temporal artery ultrasonography, which has a similar sensitivity to biopsy.

GCA is typically treated with chronic oral steroids under the management of rheumatology; however, if AION does occur, a pulse-dose of 1g intravenous Solumedrol is given for three days, then switched to oral steroids and generally continued for a lifetime.

Eye care providers should have a fairly low threshold of testing and evaluating for GCA, as ESR and CRP testing is inexpensive and easily obtainable. Temporal artery biopsy is also a routine procedure performed by vascular surgeons or even oculoplastic specialists, while temporal artery ultrasound is a good noninvasive alternative to biopsy.

Early diagnosis and treatment of GCA is imperative, given the severity of the disease and possibility of irreversible vision loss. As frontline eye care providers, ODs can help play a crucial role in identifying GCA as early as possible to mitigate negative outcomes.[3]

Clinicians should always maintain suspicion when presented with patient complaints of GCA and either initiate a work-up or promptly refer for a work-up.

Even after a patient has suffered vision loss due to GCA/AION, ODs can remain in contact with the rheumatologist, social workers, occupational therapists and other care providers to manage any vision loss affecting an individual's quality of life. ■

1. Weyand CM, Goronzy JJ. Giant-cell arteritis and polymyalgia rheumatica. N Engl J Med. 2014;371:50-7.
2. Heyreh SS. Management of ischemic optic neuropathies. Indian J Ophthalmol. 2011;59(2):123-36.
3. American Optometric Association. Doctors of optometry have big role in catching giant cell arteritis before blindness. Clinical Eye Care. www.aoa.org/news/clinical-eye-care/doctors-of-optometry-have-big-role-in-catching-giant-cell-arteritis-before-blindness. Accessed January 4, 2018.


A Bad Break-up

When the vitreous and retina don't separate fully, macular traction can be problematic—or benign. How, and when, to manage? **By Diana Schechtman, OD, and Jay M. Haynie, OD**

Even though vitreomacular adhesion (VMA) is a common retinal condition, with a prevalence of 14.7% in a 2016 retrospective review, it is often only diagnosed coincidently through the use of OCT.[1] When the adhesions alter retinal shape—in particular, the foveal contour—the patient is said to have vitreomacular traction (VMT). The condition has been associated with myriad other maculopathies, including but not limited to macular edema, epiretinal membrane and macular hole formation.

The natural history of the disease is unpredictable, adding to challenges in management. Some cases may remain both stable and asymptomatic for years, while others will show spontaneous resolution if the vitreoretinal interface ultimately achieves a complete posterior vitreous detachment. Yet some cases will progress, which may result in macular hole formation. Although VMT may be asymptomatic, patients may also present with metamorphopsia, relative scotoma and decreased vision.

Given the diversity of presentations, risk profiles and outcomes, optometrists often struggle to decide whether to refer the case to a retina specialist or simply monitor. Seeking consultation relies on numerous variables: Do you have the proper diagnostic modalities, such as OCT? What structural changes are associated with the condition? Is the patient symptomatic? What is your comfort level?

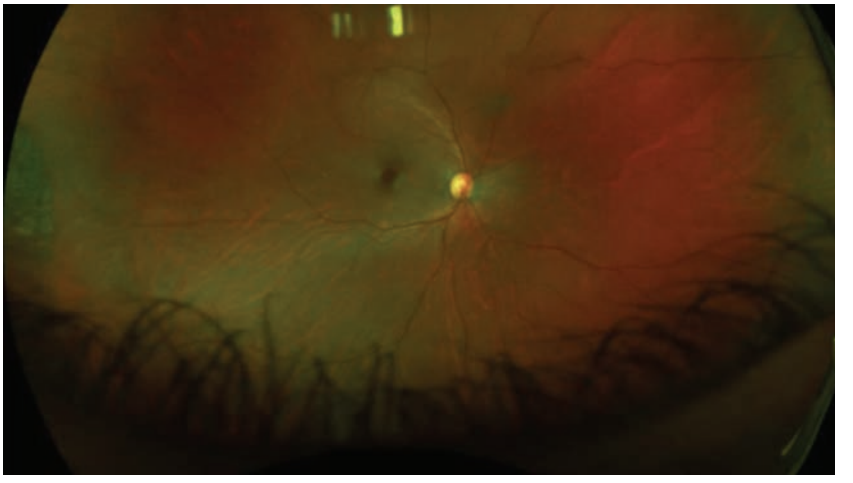


Fig. 1. The patient presented with 20/50 vision but no complaints. Fundus exam was suspicious for macular hole.

Adding another layer of complexity, treatment approaches also vary. These gray areas form the basis of this new bimonthly column. Each installment will describe a clinical dilemma common in retina care and will share the protocols and rationale of two optometrists who work in retina clinics. Dr. Shechtman practices in Miami and Dr. Haynie in Washington state. Readers will get the benefit of their two perspectives, with one author taking the lead and the other commenting on similarities and differences at the other practice.

Hole on the Horizon?

Case by Dr. Shechtman

A 54-year-old Asian female was referred by another optometrist following a fundus exam suspicious for possible macular hole OD *(Figure 1)*. OCT revealed VMT with partial hole. Although her best-corrected visual acuity was only 20/50, she had no visual complaints. Due to the absence of a full-thickness macular hole (FTMH) and symptoms, close observation was recommended. When the patient returned two weeks later, clinical exam showed no changes.

She was then asked to return in one month *(Figure 2)*. At that visit, the patient was advised that if she experienced any visual changes, she should return to clinic immediately. She was also educated on the possibility of pars plana vitrectomy and pneumatic vitreolysis as future treatment options. Close frequent monitoring remains ongoing.

In our practice, functional changes supersede structural ones in the decision-making process regarding whether and when to initiate treatment. The decision to

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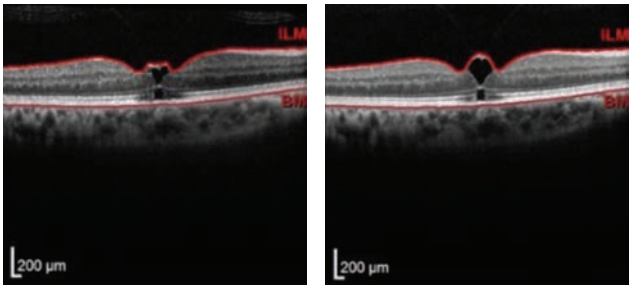

Fig. 2. Upon referral, the initial OCT scan (left) showed VMT and early macular hole formation, but vision was stable. Follow-up one month later (right) showed slight worsening. Patient was educated of recommendation for vitrectomy if FTMH develops.

treat typically depends on patients' symptomology: Is the patient complaining of decreased vision or metamorphopsia even in the presence of minimally affected vision? Does the patient have worsening symptoms, especially if the presentation initially was asymptomatic? Is the patient's corrected distance visual acuity in the range of 20/40 to 20/70? The answers to these queries also help us recognize a patient who requires consultation.

However, structural changes are not to be dismissed when deciding which patients need a consultation. For example, recalcitrant macular edema or an impeding macular hole (as in this case) should be referred for consultation, as well as progression of the condition or worsening traction, especially in the setting of new symptoms. Additionally, the status of the contralateral eye is significant. Surgeons have a lower threshold to treat VMT in the setting of a full-thickness macular hole of the contralateral eye.

Though some retina surgeons may have considered treatment in this case, our surgeon decided on frequent observation in the absence of patient complaints and in light of the relative small size of the defect. As some VMT cases may detach on their own, the risks and benefits of surgery need to be taken into account.

When considering a referral, keep these factors in mind: (1) the scope of practice laws in your state and the informal 'community standards' for optometric care in your area, (2) the diagnostic modalities available to you, (3) your background knowledge on the condition and 'gut check' on whether or not this case will keep you up at night, and (4) where the case currently is in the likely timeline of its natural progression, with more advanced status obviously arguing for prompt referral. VMT is not an emergency; referral should be considered within a few weeks.

How Our Practice Compares

By Dr. Haynie

My group here in the Pacific Northwest is for the most part quite conservative regarding VMT, given that a large percentage of patients will in fact spontaneously recover with a PVD over time. Still, chronic VMT can alter the photoreceptor layer and reduce the odds of symptomatic improvement even with treatment, so observation is not without risks itself. We consider intervention for those who have been symptomatic for a few months with persistent VMT as seen with OCT. Options include pharmacologic vitreolysis, pneumatic vitreolysis or vitrectomy surgery.

Pharmacologic vitreolysis is an in-office procedure done under local anesthesia using an injection of Jetrea (ocriplasmin, Thrombogenics) to induce posterior vitreous separation, with a reported success rate of 26.5%.[2] My group has treated a fair number of patients and achieved an estimated 50% to 60% success rate; however, we have recently reduced its use due to a reported concern of potential photoreceptor toxicity.

Pneumatic vitreolysis, also performed in-office with local anesthesia, induces a posterior vitreous detachment through intraocular injection of gas (either SF_6 or C_3F_8). Though reported success rates vary, it can be as high as 84%.[6] Pneumatic vitreolysis is cost effective and convenient, and may likely gain popularity as a result. My group has yet to embrace this modality, however.

Vitrectomy is of course the definitive treatment for VMT, but carries all the higher risks and costs of a surgical procedure. My group will consider vitrectomy for VMT that has been chronic or associated with a stage 2 macular hole on OCT imaging.

In summary, the management of symptomatic VMT demands case-by-case decisions. A detailed discussion with the patient regarding the treatment options is the most important, in my opinion. Knowing that a high percentage of patients will experience release of traction with a PVD over time, initial observation is the most popular management choice in my group currently. We largely concur with the management course outlined by Dr. Shechtman. ■

1. Reichel E, Jaffe GJ, Sadda SR, et al. Prevalence of vitreomacular adhesion: an optical coherence tomography analysis in the retina clinic setting. Clin Ophthalmol 2016;10:627-633.
2. Stalmans P, Benz MS, Gandorfer A, Kampik A, Girach A, et al. Enzymatic vitreolysis with ocriplasmin for vitreomacular traction and macular holes. N Engl J Med. 2012 Aug 16;367(7):606-15
3. Steinle N. Pneumatic Vitreolysis for VMT: a comparison of intravitreal injections of C3F8 versus SF6 versus air. Paper presented at: American Society of Retina Specialists Annual Meeting; August 11-15, 2017; Boston, MA.

For more information, visit the authors' practice websites.

Dr. Shechtman: www.retinamaculamiami.com

Dr. Haynie: www.retina-macula.com.

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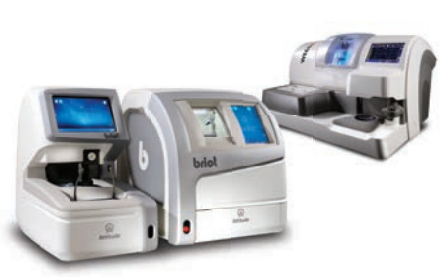

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FIRST IMPRESSIONS.

EASTWEST EYE CONFERENCE ATTENDEES get exclusive look at CooperVision's new MyDay® toric contact lens

The introduction of CooperVision's **MyDay® toric** daily disposable contact lenses has been met with enthusiasm, as eye care professionals across the U.S. realize the opportunity to significantly enhance their astigmatic patients' contact lens-wearing experience. The new addition to CooperVision's premium silicone hydrogel 1-day lens family integrates the features of **Biofinity® toric** – from a world leader in toric lens design – with the Smart Silicone™ chemistry of MyDay®, providing practitioners and their patients the best of both brands.

Before eye care professionals had the opportunity to fit MyDay® toric on their patients, however, many wanted to try it for themselves. Attendees at the 2017 EastWest Eye Conference in Cleveland, Ohio were given an exclusive sneak peek, with the ability to trial the lens on their own eyes.

"My visual acuity is great right away," said Jordan Claboine, optometric technician at Galloway Eye Care Professionals in Columbus, Ohio, after being fit with MyDay® toric. "Usually with toric lenses, it takes a while to settle. I feel like these settled quickly."

The exhibit hall was humming with practitioners and optometry students, many of whom have anxiously awaited the availability of MyDay® toric since MyDay® was first unveiled in 2015.

"I'm really excited that CooperVision is here with their new lens, MyDay® toric," said Jason Miller, OD, who practices at EyeCare Professionals of Powell in Powell, Ohio. "The buzz is out. People are excited to be part of the launch of a new lens they can leverage to improve patients' vision and comfort. It provides another opportunity for us to make our patients happy in contact lenses."

At the booth, CooperVision's Senior Manager of Technical Marketing, Steve Diamanti, Ph, D. spoke about the technology behind MyDay® toric. As a polymer chemist, Diamanti was part of the research and development team that designed the silicone hydrogel material for MyDay®.

"This is basically the Biofinity® toric design that you love and trust, now in a 1-day lens," said Diamanti. "MyDay® toric is the best toric design that CooperVision has ever made[1], and the best silicone hydrogel material that we have ever made—all in one package."

Practitioners trialing MyDay® toric at EastWest Eye had strikingly similar, positive reactions to the lens. Most commented on its comfort, saying that they could not feel the lenses in their eyes. Another common point of praise was its stability[2].

MyDay® toric features Optimized Toric Lens Geometry™, which provides uniform ISO thickness, an optimized ballast band design, large toric optic zone, and a smooth, continuous surface to make it an easy-to-fit[3], stable toric lens. This technology has long been revered in Biofinity® toric, the most prescribed toric lens in the United States[4].

Dr. Anita Chitluri of The Cleveland Clinic Foundation was one of the optometrists fitting the lens on her peers at the conference. Following a day of MyDay® toric fittings, she shared her thoughts about working with the lens.

"MyDay® toric performed really well. Wearers seem to love the initial comfort, and had great vision right off the bat[5]," said Dr. Chitluri. "It was really easy to fit, and didn't take much chair time[1]. It features great stability. The combination of fitting and performance with the health benefit and comfort—it's a no-brainer for practitioners to at least offer MyDay® toric to their patients."

Eye care professionals who have begun fitting MyDay® toric in their practices are seeing quick success. Mark Andre, Associate Professor of Optometry at Pacific University in Forest Grove, Oregon and a member of CooperVision's Professional Affairs team, said that after only one week with the lens, he already had five patients in it.

"The low modulus of MyDay® allows us to fit a much wider range of corneal shapes and sizes[3] than a stiffer modulus does," said Andre. "This is the best material with the best toric design[1]. It's a drop-the-mic moment."

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DR. CHITLURI

1. Comparison of fitting stability of the different soft toric contact lenses. Contact Lens & Anterior Eye 37 (2014) Hamed Momeni-Moghaddam et al. Optimised Toric Lens Geometry™ compared to available prism ballast, precision balance and accelerated stabilization toric lens designs. ACUVUE® Eyelid Stabilisation toric design, formerly called Accelerated Stabilisation Designs+C289. 2. CL & Anterior Eye37(2014) p349 & GFK toric fits Jan'16-Dec'17. 3. Results based on 144 participating eye care professionals in a multi-national online survey, 2016. 4. Q3 2017 US industry. Data on file. 5. During the Dispensing and 1-week visit. logMAR 0.00 = 20/20 Snellen acuity. Negative logMAR values indicate better vision). Footnote: Prospective, single center, double-masked, bilateral, randomized, 1-week cross-over, dispensing study. Inclusion criteria required 20/30 or better in each eye with habitual correction, or 20/20 best corrected vision (for binocular distance acuity). Individual results may vary. ©2018 CooperVision 4968 01/18

HOW THE Diploma Deluge is Reshaping Optometry

The student population is booming, but applicants haven't kept pace. Here are three ways to protect academic standards and avoid a glut. **By Bill Kekevian, Senior Editor**

You've heard the knock on optometric education: there are too many optometry colleges, pumping out too many new grads and coaxing them through the curriculum instead of holding them to appropriately rigorous standards. Though it may be a caricature, some elements ring true, say experts within and outside academia. "If you want to get into optometry school, you can," laments one educator.

Six new optometry colleges opened in the last decade, and more are on the way. Wingate University, a private institution in North Carolina, recently announced plans to break ground on a new school of optometry, and at least two more are exploring the option.[1] Established schools like SUNY College of Optometry have also expanded, adding 24 seats since 2008.

Growth itself isn't inherently bad. A bigger footprint for optometry gives the profession more clout with legislators and insurers. But while the number of seats has gone up, applicant volume hasn't, explains David Damari, OD, dean of Michigan College of Optometry at Ferris State University and president of the Association of Schools and Colleges of Optometry (ASCO). In fact, recent years have even seen declines. "That's going to make for some difficult choices," he says. Some schools "may have to fill classes with applicants who are seriously at risk of not completing the program or passing national boards."

Nathan Lighthizer, OD, assistant dean of clinical care services at Northeastern State University in Oklahoma, puts it this way: his institution offers seats to 28 students each year, the smallest class size in the country. With new options opening, some of the top students selected by Northeastern are likely to end up elsewhere. If, for instance, five students make that call, Northeastern has to offer spots to choices 29 through 33. It's a sort of domino theory of admissions standards, and educators are starting to worry that it's diluting the pool of qualified candidates.

Another concern: will new grads find productive roles in regions most in need of eye doctors, or merely bloat the ranks of well-served cities and towns? While more opportunities exist today—necessitating more optometrists—putting these new ODs where they can best serve the public remains a challenge.

Here, *Review of Optometry* considers recent data on the state of optometric education, what problems it presents and the safeguards being put into place to protect the discipline.

More Seats, Fewer Applicants

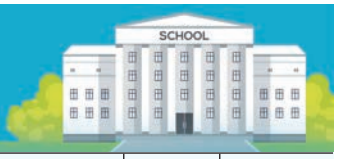
After a 20-year lull, optometry's current boom started in 2009 with the dual openings of schools at the University of the Incarnate Word and Western University of Health Sciences. Another four soon followed. Those six additions, plus incremental growth at established schools, expanded available seats by 31% from 2008 to 2017 (*Table 1*).[2,3]

Educators stress that while the increased number of seats may worry some, it's the number of applicants that worries *them*. The applicant-to-seat ratio is trending down and stands at roughly 1.4 applicants per seat.[4] ASCO reports a 4.4% year-over-year decline in applications from 2016 to 2017 but an increase of seats by 2.5% over the same period. Thus far in the 2018

Source: ASCO. See refs. 2,3

Table 1. Increase in Student Population Resulting from School Expansion

The growth that began in 2009 added 449, or 31%, more seats available for students in 2017. Three schools failed to fill all seats.



School	Year Founded	City, State	Seats in 2017	Change from 2008
Illinois College of Optometry	1872	Chicago, Ill.	165	2
New England College of Optometry	1894	Boston, Mass.	127	12
Southern California College of Optometry	1904	Fullerton, Calif.	104	6
Ohio State University College of Optometry	1914	Columbus, Ohio	67	3
Pennsylvania College of Optometry	1919	Elkins Park, Pa.	155	-5
Pacific University College of Optometry	1921	Forest Grove, Ore.	91	-1
University of California, Berkeley	1923	Berkeley, Calif.	66	0
Southern College of Optometry	1932	Memphis, Tenn.	136	12
Indiana University School of Optometry	1951	Bloomington, Ind.	68	-10
University of Houston	1952	Houston, Texas	101	1
UAB School of Optometry	1969	Birmingham, Ala.	50	5
SUNY College of Optometry	1971	New York, NY	100	24
Michigan College of Optometry	1974	Big Rapids, Mich.	37	1
Northeastern State University	1979	Talequah, Okla.	28	0
University of Missouri at St. Louis	1980	St. Louis, Mo.	45	-2
Inter American University of Puerto Rico	1981	Bayamon, PR	60*	0
Nova Southeastern University	1989	Ft. Lauderdale, Fla.	105	3
University of the Incarnate Word	2009	San Antonio, Texas	67	67
Western University of Health Sciences	2009	Pomona, Calif.	86	86
MCPHS School of Optometry	2012	Worcester, Mass.	63	63
University of Pikeville	2016	Pikeville, Ken.	60	60
Arizona College of Optometry/Midwestern	2017	Glendale, Ariz.	56	56
Chicago College of Optometry/Midwestern	2017	Chicago, Ill.	66	66
			1903	**+449**

** Enrollment in 2016. Data not available for 2017.*

cycle, applications are down 11.5% over the year prior, according to ASCO.

That doesn't leave a lot of room for schools to be selective, explains Joseph Bonanno, OD, professor and dean at Indiana University School of Optometry. Some students are being accepted who otherwise wouldn't, especially at the newer institutions. ASCO data shows that the six newest schools accept objectively lower-scoring applicants (*Table 2*).[5] In 2017, they accepted GPA averages ranging from 3.20 to 3.41 with a group average of 3.32; for the six oldest schools, it was 3.39 to 3.66 and an average of 3.49. Of the six lowest GPAs accepted in the United States last year, five come from the newest institutions (*Table 2*).

Some evidence suggests that the ripple effect of lowering admissions standards may have spread to other optometry programs, just as Dr. Lighthizer described. Students are being accepted with lower optometric admission test (OAT) scores nearly across the board compared with a decade earlier (*Table 3*).[2,3] Averaging all changes in OAT scores (i.e., increases as well as decreases) gives an overall decline of 1.75%, but individual schools saw declines as high as 5%. Of the 17 US optometry schools that existed in 2008, 14 lowered their accepted academic average OAT score by 2017—11 by five points or greater.[2,6]

What's the picture like at the end of a student's college experience? Also troubling. Optometry board pass rates published in late 2017 found a national rate of 91%, with some schools falling well below the average (*Table 4*).[6] Above-average student populations don't always correlate with below-average pass rates. Of the bottom five, Salus University's Pennsylvania College of Optometry (PCO), whose ultimate pass rate is 84.2%, has the largest class (152 candidates) and Southern California College of Optometry (SCCO) at Marshall B. Ketchum University is second (85.6% pass rate) with 97 candidates. But the other three are mid-range on class size, with Western University of Health Sciences hosting 76 candidates (only 68.4% pass), Rosenberg hosting 64 (84.4% pass) and Massachusetts College of Pharmacy and Health Sciences (MCPHS) hosting only 59 (74.6% pass).[6]

Altogether, eight schools fell below the National Board of Examiners' (NBOE) average pass rate. Among those were the five that accepted the lowest OAT scores in 2013 (the year the class of 2017 would have entered the program).[7] However, while the connection exists on the low end of the chart, the trend doesn't necessarily indicate that low OAT scores correlate directly with low ultimate pass rates. Take for instance, New England College of Optometry, which, at 90.4%, fell below the NBOE's average pass rate, but in 2013 accepted an average academic OAT score of 320 and an average total science score of 318. That's on par with the average OAT scores for the entering class of 2013 (whose academic average was 320 and average total science score was 317). Conversely, Midwestern University's Arizona College of Optometry accepted students in 2013 with average scores of 319 (academic) and 315 (total science) (tied

Source: ASCO. See ref. 5

Table 2. Profile of Accepted Students in 2017: Newest Programs vs. Oldest Programs

All scores are averages of those students accepted by each institution. Not all students matriculated.

Newest Schools	Year Founded	Academic OAT Score	Total Science OAT Score	GPA Score
Arizona College of Optometry/Midwestern	2017	327	325	3.41
Chicago College of Optometry/Midwestern	2017	311	303	3.31
University of Pikeville	2016	311	300	3.32
MCPHS School of Optometry	2012	303	291	3.2
Western University of Health Sciences	2009	325	325	3.28
Rosenberg School of Optometry	2009	319	316	3.37
GROUP AVERAGE		**316**	**310**	**3.32**
Oldest Schools	**Year Founded**	**Academic OAT Score**	**Total Science OAT Score**	**GPA Score**
Illinois College of Optometry	1872	325	323	3.42
New England College of Optometry	1894	332	331	3.39
Southern California College of Optometry	1904	344	347	3.44
Ohio State University College of Optometry	1914	341	343	3.66
Pennsylvania College of Optometry	1919	320	315	3.45
Southern College of Optometry	1932	332	329	3.57
GROUP AVERAGE		**332**	**331**	**3.49**

for fifth lowest) and a 3.37 GPA (seventh lowest) and, yet, 95.4% of its students pass boards. UAB is another example where, although its OAT scores fall below the average (academic, 315; total science, 311), 94.6% of students pass boards.[6,7] This suggests that while being selective with the students who enter the program can impact the outcome, ultimately a school has the opportunity to right its students' ship.

Numbers Don't Tell All

To wit, educators say students' personal stories can counter the notion that lower scores make for less suitable candidates (see, *"I'm More Than My GPA,"* page 45).

While GPA and OAT scores can be predictors of success in optometry school, there's a third factor that's harder to quantify. "You can't just look at GPA on face value, says Joseph Pizzimenti, OD, an educator on the admissions board at UIW's Rosenberg School of Optometry. "A physics major from University of Chicago may have only graduated with 2.95," but someone with that degree from that school "will likely perform well in optometry school," as long as their OAT scores measure up. "If that kid can communicate during an interview, I'm going to take her every day of the week and twice on Sunday," he says. "You do this long enough and you know where the quality [undergraduate] programs are."

Across the country in Pennsylvania, James Caldwell, OD, dean of student affairs at PCO, agrees about the value of communication skills. "I don't know where the study is that says you have to have a 3.7 GPA to be a better optometrist than someone with a 3.3 GPA." PCO looks for "appropriate coursework in the appropriate combination," he says. That is, a mix of multiple science courses, "pre-med quality work," participation in school organizations and community service. "You want to have a nice, solid portfolio. You don't want to be all academic and no personal skills."

In fact, in a 2008 ASCO survey, eight schools rated students' OAT scores "significant" in influencing their admission process, another eight rated it only "moderate" and one even said it had no influence at all. But they all required an in-person interview.[3]

Bright and motivated students can succeed just as well as undergraduate superstars, say educators in the trenches. But an objectively weaker pool (on purely academic measures) of candidates and rapidly evolving clinical responsibilities are causing institutions to revamp some elements of their programs, or at least contemplate doing so. Broadly speaking, three actions can keep these trends from inflicting damage to institutions, practitioners and the profession as a whole.

1. Adapt

With downward pressure on admissions standards, the education community may need to enact some short-term reforms to ensure a stronger long-term outlook.

"The fact that there are more seats available while we have the same number of candidates presents a challenge," says Dr. Damari. "And it's difficult for some programs to decrease their class sizes."

But if they did, it wouldn't be without precedent. For example, in the 1980s Southern College of Optometry (SCO) did reduce its class size. "When I came in in 1980, my class had 152 students," says Lisa Wade, OD, director at SCO's Hayes Center for Practice Excellence. "They soon reduced it to 90 over concerns about the quality of applicants" and cut tuition by 27%. "At that time, SCO was the most expensive optometry college in the country, and they realized they were on a path that could not be sustained or continue to attract quality applicants," Dr. Wade says. Today, SCO has 132 seats, up by only eight from 2008, when the current boom began.[3]

The mix of didactic vs. hands-on training might be in need of a rethink, too. "We're ready to make the most efficient modifications to make sure our students are best prepared," said PCO Dean Melissa Trego, OD, in a videotaped response to the NBOE board pass rate data, including ending a program that allows third-year students to work in an off-campus clinic in January.[8] Now, they'll remain on campus so they can be prepared for part one of the boards, which begins in March. She also stresses that the NBOE figures are only first-time scores. "Ultimately, when students graduate, they are able to pass part one," Dr. Trego adds. "We've already started the process of developing a new curriculum which provides multiple opportunities to be tested."

Some schools may have figured out a way to both bring in a high number of students, including those whose GPAs may drag down their average, and still see nearly every single student pass boards.

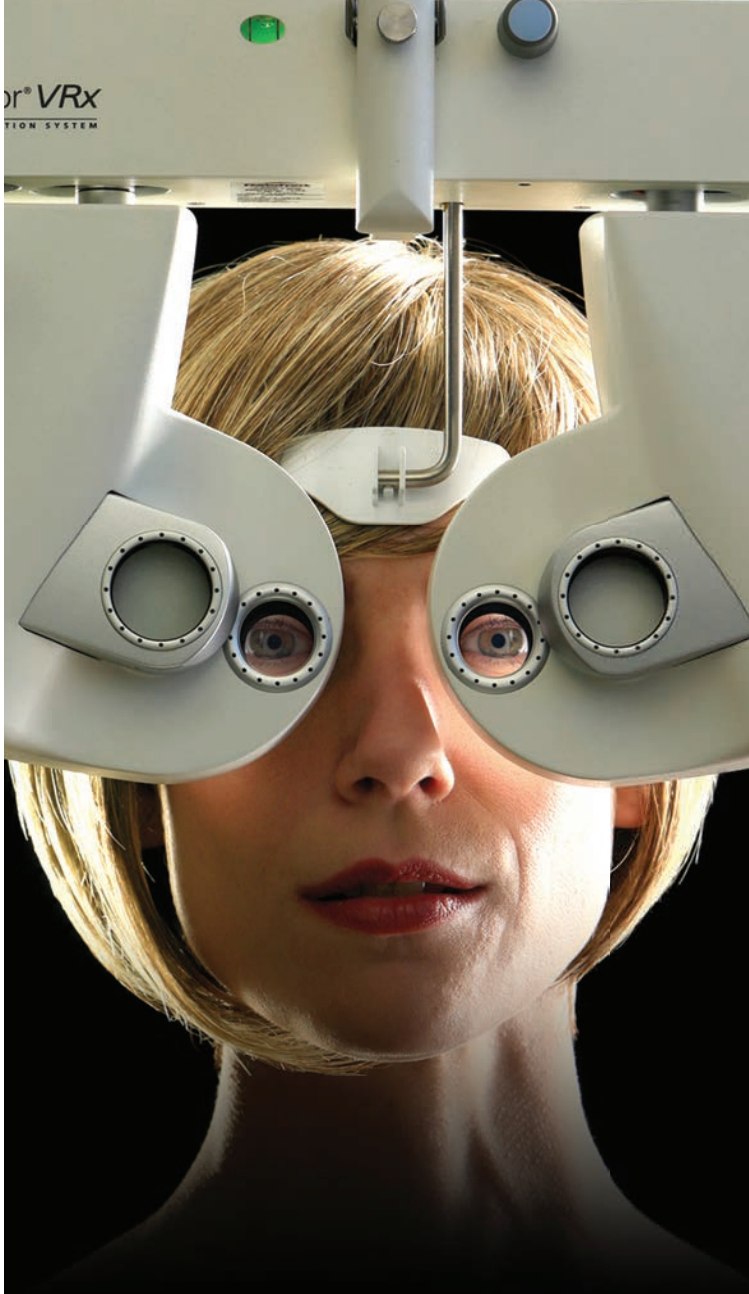
Look at Nova Southeastern University in Ft. Lauderdale, Fla., which has hosted more than 100 students per class since 2011 and its average incoming GPA in 2014 was 3.36, tied for fourth lowest. How, then, have administrators managed to keep its ultimate pass rate at 97.9%? Perhaps it has something to do with how the school evaluates students on their way in.

"I'm More Than My GPA"

As she considered what to do with her bachelor's degree, Shannon Koenders, 26, was dissuaded by some from even considering optometry school. The native of Sioux Falls, SD, doesn't blame them. "I'll be the first to admit my GPA wasn't spectacular," she says, describing it as "just scraping 3.0." But she comes from a large family and grew up helping her parents and siblings care for a brother with Down syndrome, something she says helped her develop the skills of a caring, attentive clinician. This spring, Ms. Koenders will graduate from the University of the Incarnate Word's Rosenberg School of Optometry in San Antonio, Texas.

In her time there, she's achieved the academic success that eluded her in her undergraduate days and then some. In fact, she is currently seeking to specialize in caring for the vision of special needs patients, including those with Down syndrome and autism, both conditions on the rise in the United States.

"Maybe I wasn't a competitive applicant on paper," says Ms. Koenders, reflecting on her journey into optometry. But once inside the gates, she's developed into a member of her school's Gold Key Honor Society, parlayed her involvement in Student Volunteer Optometric Services to Humanity into an upcoming internship, worked as an optical assistant and visited Oaxaca, Mexico, on a mission, for which she had to give eye exams in Spanish. "I'm more than just my GPA," she concludes.



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Source: ASCO. See refs. 2,3

Table 3. Effect of Student Expansion on Academic Standards: GPA and OAT Scores, 2008 vs. 2017

Note: "Matriculated students" are those who entered the program. "Accepted students" (Table 2) are those offered seats.

School (in order of year founded)	GPA of matriculated students, 2017	Change from 2008		Academic OAT of matriculated students, 2017	Change from 2008	
Illinois College of Optometry	3.36	-0.03	-0.88%	318	-17	-5.07%
New England College of Optometry	3.31	-0.03	-0.90%	326	-5	-1.51%
Southern California College of Optometry	3.41	0.06	1.79%	339	-1	-0.29%
Ohio State University College of Optometry	3.63	0.07	1.97%	336	-8	-2.33%
Pennsylvania College of Optometry	3.39	0.05	1.50%	310	-10	-3.13%
Pacific University College of Optometry	3.49	0.08	2.35%	326	-14	-4.12%
University of California, Berkeley	3.53	0.00	0.00%	358	-3	-0.83%
Southern College of Optometry	3.56	0.11	3.19%	331	-9	-2.65%
Indiana University School of Optometry	3.64	0.15	4.30%	322	-8	-2.42%
University of Houston	3.57	0.11	3.18%	340	4	1.19%
UAB School of Optometry	3.59	0.03	0.84%	317	-10	-3.06%
SUNY College of Optometry	3.61	0.10	2.85%	345	6	1.77%
Michigan College of Optometry	3.70	0.16	4.52%	334	-2	-0.60%
Northeastern State University	3.49	-0.21	-5.68%	315	-15	-4.55%
University of Missouri at St. Louis	3.53	0.09	2.62%	322	-10	-3.01%
Inter American University of Puerto Rico	2.94*	-0.11	-3.61%	301*	11	3.79%
Nova Southeastern University	3.36	-0.03	-0.88%	323	-10	-3.00%
University of the Incarnate Word	3.44	*Avg: 0.04*	*Avg: 1.01%*	320	*Avg: -5.94*	*Avg: -1.75%*
Western University of Health Sciences	3.20			319		
MCPHS School of Optometry	3.20			303		
University of Pikeville	3.31			306		
Chicago College of Optometry/Midwestern	3.24			318		
Arizona College of Optometry/Midwestern	3.39			320		
** 2016 scores. Data not available for 2017.*	*Avg: 3.43*			*Avg: 324*		

Both Nova and Indiana University have safety nets to catch students before they fall. "We've created a new program wherein we identify applicants we think would struggle and we put them on a five-year path that spreads out the academic burden," Dr. Bonnano says of Indiana University. "OD programs demand a lot of course hours, and they're all science courses." Undergrads aren't used to taking five science courses at once, he notes. "We're interested in students' success. We want to lower our attrition rate. I think you're going to see this popping up at other schools," he says. "The curriculum's gotten tougher, too."

It may sound a bit like coddling, but ASCO President Dr. Damari doesn't see it like that. He says it's a way to correct an imbalance. "Students from diverse economic backgrounds may not have had the best educational preparation because of the circumstances under which they grew up," he says. "This gives us the opportunity to bring more people from different backgrounds into the profession, which is extremely valuable for any health care profession if you're going to serve a diverse population."

While the students entering at the bottom of the class have a chance to catch up, those nearing graduation have an option for a different kind of fifth-year: taking on a residency. To be clear, a residency is not a fifth *academic* year but rather a chance for hands-on experience. Optometry colleges today "must teach at the broadest scope of practice," says Dr. Pizzimenti. "Students need to be able to sit for any state board in the country." That includes states such as Oklahoma, where optometrists can perform laser procedures and a nationwide trend toward optometric surgical comanagement.

Formal residency programs represent a new level of training more suited for the needs of a modern OD, with students learning a style of medical practice that would have been unrecognizable a generation ago. In 1976,

the Veteran's Affairs Medical Center in Kansas City, Mo., founded the first formally accredited one-year residency for optometrists.[9] Today, approximately 235 Accreditation Council on Optometric Education (ACOE) accredited residencies place hundreds of new ODs every year into programs as broad as family practice optometry and as narrow as vision therapy and rehabilitation.[9]

"There's only so much you can fit into a four-year curriculum," explains Caroline Beesley Pate, OD, associate professor and director of residency programs at the University of Alabama's School of Optometry in Birmingham, Ala. "Although the scope of practice has changed drastically in the last 30 years, optometric education has largely remained a four-year program," she says. Educators have to cover the same fundamental skills as in previous eras while incorporating everything that reflects a modern scope of practice, one that extends as far as injections, lasers and minor surgical procedures. "Doing a residency enables you to further expand if you're interested in those areas."

2. Incentivize

Education reform can address the changing nature of both the applicant pool and optometric responsibilities, but those new doctors still need to find a spot for themselves in the profession. The greatest need lies in rural areas, where health care demand and resources are often at their most unbalanced.

"Central Appalachia has the highest incidences of severe vision loss from other factors such as diabetes and hypertension," said a 2015 University of Pikeville press release announcing its intentions to launch an optometry program. "Our objective is to provide access and education to the people of the mountains and to address a critical health care need."[10]

"Optometrists have sort of congregated in urban areas," says Wingate University Vice Provost Robert Supernaw in a statement about its upcoming expansion into optometric education. "We thought that we could correct that." The national average is 1.3 ODs per 10,000 population, says Wingate. In North Carolina, it's just 1.1, "and many counties in eastern North Carolina have well below the national average—or no optometrists at all." The school will include a community clinic to serve indigent local residents.[1]

While those university clinics provide much-needed care, they can't meet the ballooning demand on their own. "There's plenty of people emailing me from rural areas with job opportunities, not only within Alabama, but throughout the Southeast," says Dr. Pate. "The

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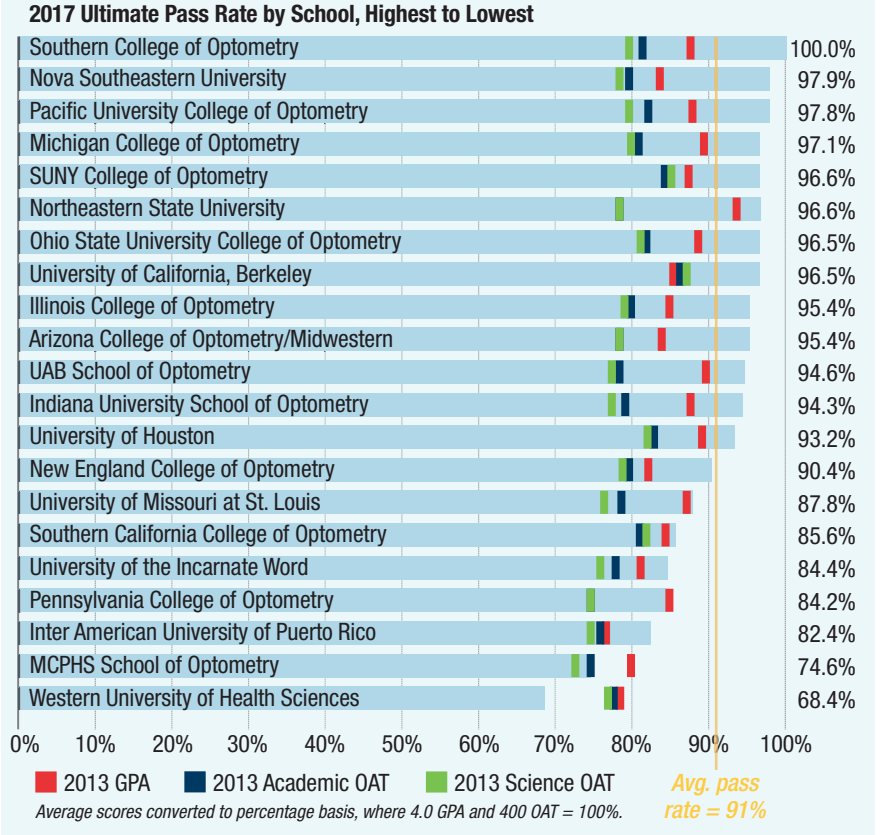
Source: ASCO. See ref. 6,7

Table 4. Admissions Standards and Pass Rates: How the Class of '17 Fared

Average OAT and GPA scores from 2013 enrolled students overlaid on 2017 board exam ultimate pass rates.



question is, are these graduates willing to go where the opportunities are?" She theorizes that optometry can withstand the influx of new students if they are willing to disperse from urban centers. As a state-funded program, a portion of UAB's support relies on accepting a higher percentage of students from within the state. But, Dr. Pate says, she knows of no real school-sponsored incentive program to keep them in that state.

Some states have experimented with incentive programs. Those help to balance the pro/con lists a new doctor draws up, but can fall short. After her own graduation from PCO, Dr. Pate's home state of Maryland agreed to reimburse her for a percentage of her tuition if she agreed to practice there for four years. But when an opportunity at UAB presented itself, she opted to forgo the deal.

Maryland may have lost Dr. Pate, but the gambit makes sense. In their mid- to late-twenties, many recent grads aren't only settling into a career but also into family life. Encouraging young doctors to stay in a particular area at a time when many are getting married, buying a home or perhaps having children, doesn't necessarily ensure they won't move after four years, or break their contract at some point before then, but it helps establish roots young doctors may be reluctant to break.

Unfortunately, Dr. Pate is not exactly an outlier. "Those kinds of programs are decreasing," says Dr. Damari. States have found that more students are willing to pay the penalty to get out of the contract, he notes.

Urban centers offer personal and professional advantages that many new optometrists deem too good to pass up, be it quality of life improvements that come with population density or professional access to the broader health care infrastructure, which, not coincidentally, also clusters in major cities. The expanding palette of optometric practice creates in many students career aspirations ill-suited to rural areas (see, *"Wanting More,"* page 49).

Of the many threads that weave together to form the complex state of optometric education in 2018, this inability to match health care needs with resources in the form of human capital is perhaps the most intractable.

3. Promote

If there's any consensus among optometric educators, it's that optometrists must do more to raise its profile and attract more qualified candidates. "As a profession, we need to brag about who we are," Dr. Caldwell says. "We need to embrace the diversity of practice opportunities and the diversity of educational programs."

"If we did a better job of letting the public know why they need vision care, not just eye disease care," says Dr. Damari, "there would be more demand out there than we could know," for both optometric care and optometric careers. "Look at what dentistry did in the 1960s and what nursing did in the 1980s and 1990s" to raise their profiles. "Our field has really lagged behind on that and we really need to step up."

Third-year ICO students Jessica Capri and Mallory Scrimger are eager to take on optometry's 21st century challenges.

Wanting More

According to Matt Geller, OD, founder of the New Grad Optometry website, "The people who put glasses up on the wall and run a little family practice—that's going to go away." That model is "simply over," he says.

Before optometry students even graduate, many are eyeing careers modeled after subspecialty practitioners who concentrate on areas such as dry eye, diabetes, pediatrics, glaucoma or low vision. "It really is beneficial to have that niche," says Dr. Pate. "It puts you in a class above the average graduate. You have that extra experience under your belt and you'll be a little more marketable, and that will open doors you might not have considered, like academics or VA hospitals."

Just look at third-year Illinois College of Optometry students Jessica Capri and Mallory Scrimger, 23 and 24, respectively. They're well aware that optometry in the 21st century offers a buffet of opportunities instead of a cookie-cutter career. "That's the really cool thing about this field—you can do so much with it," says Ms. Capri, who is considering a law degree once she graduates. "The field is changing. It's not going to be just refractions anymore. It's becoming more and more about things like medically necessary contact lenses and it's becoming more inter-professional. That's one of the things that drew me to this career"—the ability to contemplate options as varied as retail, research and specialty clinics, she explains. Ms. Scrimger adds, "It's such a multifaceted profession. I didn't realize initially that there were so many different avenues I could go down."

ODs are even finding career paths in hospital administration. After joining the surgical comanagement team at Manhattan Eye Ear & Throat Hospital, Marta Fabrykowski, OD, noticed delays in processing. She led the effort to streamline booking and intake and delays were substantially reduced. Now, she says, she's fed up working *for* the medical center—and wants to *run* the center. To that end, she has just enrolled in Yale's MBA program.

This diversity of opportunity could be a financial boon for optometrists. "There's no lack of positions out there for optometrists today," says Dr. Wade, who, as part of her position with SCO assists in plugging graduates into the working world. "We have way more opportunities than ODs" available. And, as a result, "people are offering more competitive compensation packages."

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Toward that end, Dr. Damari explains, ASCO has gone as far as to hire a public relations firm to get help growing the profession's visibility in the eyes of the public. But, he and others suggest, there are several actions individual optometrists can take today to help in the effort, including keeping their eyes open for young patients who may have what it takes to go into optometry school and making in-roads into their communities.

Inventing the Future

More new schools are coming. Although ASCO and others can advise stakeholders on the burden new educational facilities may create, optometry cannot halt private development. Those closest to the issue suggest the field grow parallel to new institutions by broadening the definition of optometry, pushing for scope of practice expansions—and trusting the next generation. "I think a lot of people are just afraid of change," says Ms. Scrimger. "But, for our generation, maybe not so much."

Optometry has always been a self-made discipline. Fifty years ago, a group of ambitious young ODs were dismayed to find many of the diagnostic and therapeutic skills they learned in optometry school could not legally be put into practice.[11] They lobbied legislators for change and transformed optometry from a refraction-based job to a primary eye care profession. "The best way to predict the future is to invent it," said computer pioneer Alan Kay. That's always been optometry's way forward. ■

1. Yost K. Wingate pursuing optometry school. www.wingate.edu/wingate-pursuing-optometry-school. October 9, 2017. Accessed February 2, 2018.
2. ASCO. Profile of the 2017 optometry entering class. https://optometriceducation.org/wp-content/uploads/2017/10/ASCO-Prof-Entering-Class-2017.pdf. Accessed Feb. 8, 2018.
3. ASCO. Annual student data report academic year 2008-2009. https://optometriceducation.org/wp-content/uploads/2013/03/2008-2009-Student-Data-Report.pdf. Accessed Feb. 8, 2018.
4. Mullin CF. The future of optometric education. www.charlesmullen.com/optometric-education-challenges-opportunities. September 14, 2017. Accessed, October 3, 2017.
5. ASCO. Profile of Applicants to OD Degree Programs for Fall 2017 Entering Class. https://optometriceducation.org/wp-content/uploads/2017/10/ASCO-Profile-of-Applicants-2017.pdf. Accessed Feb. 8, 2018.
6. ASCO. NBOE 10/2016 – 9/2017 Institutional yearly performance report. https://optometriceducation.org/wp-content/uploads/2017/12/ASCO-Report-2016-2017.pdf. Accessed Feb. 8, 2018.
7. Profile of the 2013 optometry entering class. Association of Schools and Colleges of Optometry. https://optometriceducation.org/wp-content/uploads/2013/10/Profile-of-the-Entering-Class-2013.pdf. Accessed Feb. 8, 2018.
8. National board of examiners in optometry pass rates. Salus University. www.salus.edu/Colleges/Optometry/Doctor-of-Optometry---Traditional-Program/NBEO-Pass-Rates.aspx. Accessed February 4, 2018.
9. FAQs about residencies. ASCO. optometriceducation.org/students-future-students/residency-programs/faqs-about-residencies/. Accessed February 4, 2018.
10. Kentucky College of Optometry set to recruit inaugural class. University of Pikeville News. www.upike.edu/News/Campus/Kentucky-College-of-Optometry-set-to-recruit-inaug?feed=UPikeNews. November 15, 2015. Accessed, October 3, 2017.
11. Haffner A. The La Guardia conference—the meeting that changed the profession. Hindsight: The Journal of Optometry History. 2010;41(1):17-20.

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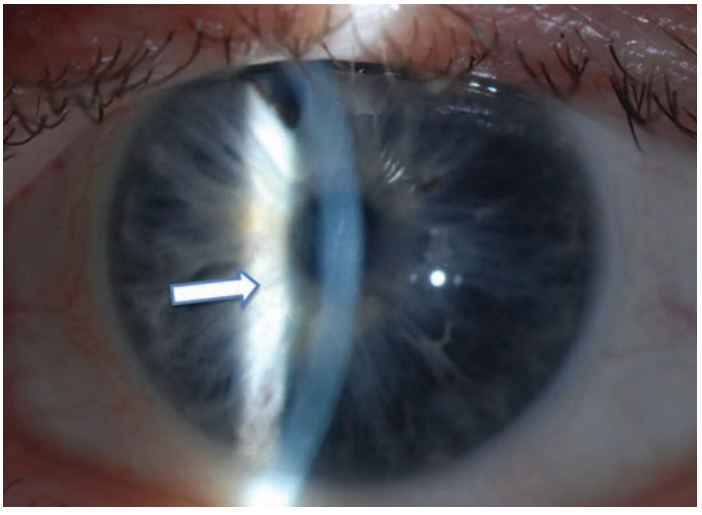
Rethinking Endothelial Repair

An eye drop that might regrow cells and a graft-free surgical technique may soon revolutionize the treatment regimen for these patients.

By Amelia C. Rohan, BA, and Kathryn Colby, MD, PhD

Historically, visually significant endothelial dysfunction, most commonly caused by Fuchs' endothelial dystrophy (FED), was treated by full-thickness corneal transplantation—penetrating keratoplasty (PK). Unfortunately, this procedure leaves the patient with a long visual recovery, a high chance of immunologic rejection, postoperative astigmatism, as well as the increased risk for wound dehiscence, even with minor ocular trauma years after the transplant.[1]



One month postoperative appearance following 4mm Descemet's stripping without endothelial keratoplasty in a 64-year-old man with Fuchs' dystrophy. The arrow highlights the edge of the descemetorhexis.

More recently, PK has been sidelined in favor of Descemet's stripping endothelial keratoplasty (DSEK) and Descemet's membrane endothelial keratoplasty (DMEK), partial-thickness transplant procedures that involve grafting of donor endothelium, with (DSEK) or without (DMEK) a small amount of donor stroma. Although visual recovery with these procedures is quicker than with traditional full-thickness PK, rejection and surgical complications still remain.[2]

With advances in surgical techniques over the past few decades, surgical outcomes for endothelial dysfunction patients have improved dramatically, but several significant problems remain in the developing world, namely a shortage of donor corneas and trained corneal surgeons. In the entire developing world, for example, fewer than 30,000 corneal transplants are performed yearly, while in the United States alone, almost 50,000 transplants were performed in 2016.[3,4] This has driven research toward finding alternatives to the classic surgical approach of tissue transplantation.

Several novel methods have been proposed to reduce the remaining complications of tissue transplantation for endothelial dysfunction, including the

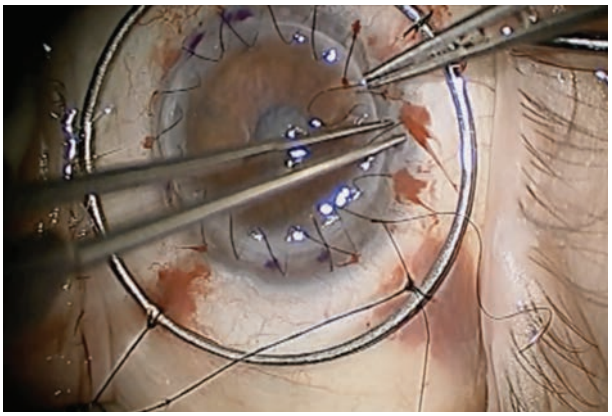


Photo: Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

Traditional PK, as seen here, comes with the risk of graft rejection, post-op astigmatism, suture management, intraocular complications and traumatic corneal rupture. Newer partial-thickness transplant procedures have helped minimize these potential complications.

use of cultured human endothelial cells (which is potentially applicable for all forms of endothelial dysfunction) and Descemet's stripping without endothelial keratoplasty, which eliminates the need for any donor tissue or cells, but is only applicable in the treatment of FED.[2,5] A potentially important innovation is the use of rho-kinase (ROCK) inhibitors, either topically or intracamerally, as an adjunct to Descemet's stripping or injection of cultured cells.[5-8]

This article briefly reviews our current understanding of the causes of endothelial dysfunction, and then delves into the status of these new and promising treatments.

Disease Basics

A firm grasp of the biology of the corneal endothelium is key to understanding the mechanisms that go awry in endothelial dysfunction. The corneal endothelium consists of a monolayer of hexagonal cells derived from neural crest cells. The endothelium maintains corneal transparency through the regulation of the hydration state of the cornea, which involves both a passive barrier function of the intact monolayer and active ATP-dependent ionic pumps.[1] These cells are halted in the G1 phase of the cell cycle, and are therefore normally prevented from dividing after birth.

Any damage to the endothelial cells from trauma (e.g., following cataract surgery) or endothelial dystrophies, such as FED, causes the remaining healthy cells to undergo changes in shape (pleomorphism) and size (polymegethism), as they spread out to

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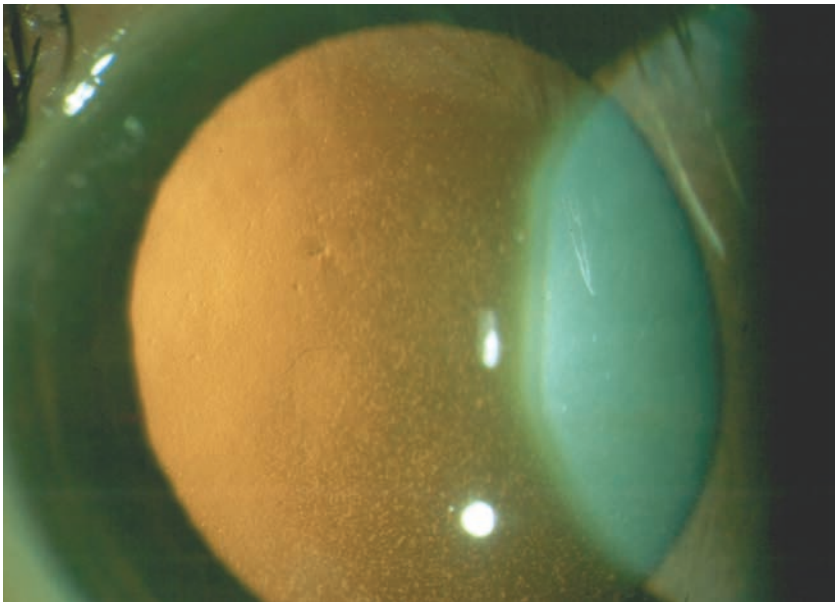


This patient has confluent central guttae from FED. Even though the cornea retained normal thickness, the patient's vision was reduced to 20/30 due to the visual degradation from the guttae.

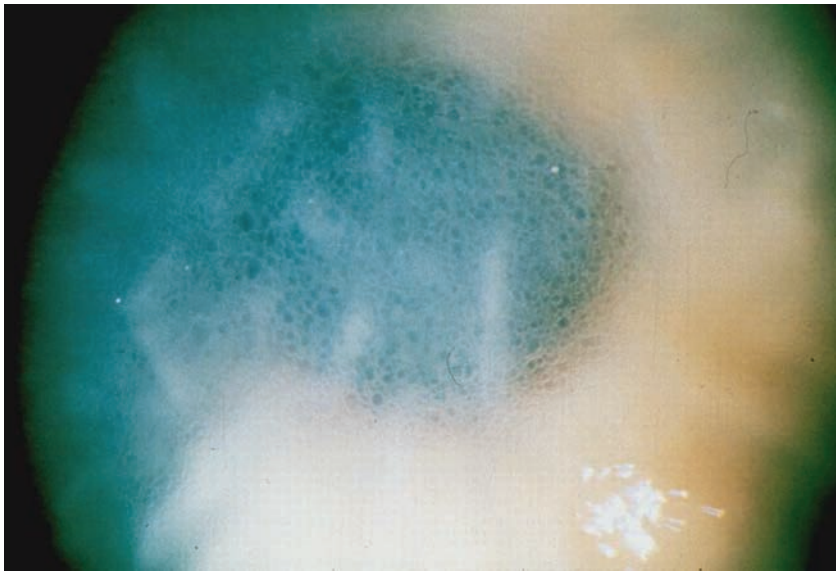
cover the damaged area.[1] Corneal endothelial cell density in a healthy adult subject is between 2,000 and 2,500 cells/mm^2. When the cell count drops to between 500 and 1,000 cells/mm^2, the endothelium begins to decompensate, and corneal edema and reduced vision develop.[6]

FED, the most common endothelial dysfunction, is usually diagnosed in patients older than 40. The disease is slowly progressive and generally requires intervention when patients reach their seventh or eighth decade of life. FED appears to follow an autosomal dominant inheritance pattern; however, older patient age during onset and variable penetrance makes a true determination of inheritance difficult. Many patients who are asymptomatic likely remain undiagnosed. Numerous genetic mutations have been associated with FED, including *COL82A*, *SLC4A11*, *ZEB1*, *LOXHD1* and *AGBL1*.[1] However, the most common genetic cause is expansion of a trinucleotide repeat in an intronic region of the *TCF4* gene on chromosome 18, which accounts for up to 75% of cases in Caucasian patients.[9]

Corneal guttae—excrescences on or within Descemet's membrane—are the initial finding of FED and can be found in up to 4% of patients in the United States, although a much smaller percentage of patients with guttae go on to develop FED.[1] Guttae themselves scatter incoming light, causing decreased visual acuity, even without severe corneal edema.[10]

If left untreated, progressive FED leads to corneal edema, reduced visual acuity and, eventually, painful blindness. With the success of modern transplantation techniques, few patients in the developed world get to this advanced stage.

Many proposed mechanisms for FED exist, including mitochondrial dysfunction, oxidative stress and a variety of genetic mutations.[1] Mitochondria play a vital role in the production of ATP, which is necessary to run the ionic pumps in the corneal endothelium that maintain proper corneal dehydration. Samples taken from human endothelial cells removed during keratoplasty have shown an increased number of mitochondria, suggesting a compensatory mechanism



This image shows advanced endothelial dysfunction with Descemet's membrane folds and microcystic epithelial edema.

to combat mitochondrial dysfunction.[11] The body naturally produces reactive oxygen species during the electron transport chain and as a response to UV exposure. When normal combative mechanisms against these substances break down, oxidative stress can damage DNA, proteins and lipids. Especially vulnerable to oxidative stress is mitochondrial DNA (mtDNA), which research shows can be damaged in the endothelium of FED patients compared with normal controls.[12] Studies also show a reduction in telomere length, a common sign of DNA damage, in FED corneas, which further supports this theory.[11]

Promising Novel Therapies

Given the significant visual effects of endothelial dysfunction—especially in FED where the guttae themselves can cause light scatter and visual degradation—and the potential complications associated with corneal transplantation, researchers continue to search for novel treatment options. A few are promising and have been used in a limited number of patients. Several of these new techniques may be enhanced by the adjuvant use of ROCK inhibitors.

Rho kinases are serine/threonine kinases that play an important role in diverse cellular pathways, including cell migration, proliferation and apoptosis.[13] Two human rho-kinase homologs exist, ROCK1 and ROCK2, whose genes reside on different chromosomes, although they have similar functional domains. The distribution of the two ROCK homologs varies in different tissues, with ROCK2 predominating in the eye.

Researchers have looked at inhibition of the action of rho kinases by ROCK inhibitors as a treatment for a wide variety of disparate diseases, including asthma, cancer, osteoporosis and neuronal degeneration.[14] In ocular disease, ROCK inhibitors are most commonly studied for glaucoma. Glanatec (ripasudil hydrochloride hydrate 0.4%, D. Western Therapeutics Institute), while not available in the United States, has been approved in Japan since 2014 for glaucoma and ocular hypertension.[15] This agent inhibits both forms of rho kinase and is believed to act on the trabecular meshwork to increase outflow facility. In December 2017, the first topical ROCK inhibitor Rhopressa (netarsudil, Aerie Pharmaceuticals) was FDA approved as a treatment for glaucoma in the United States and should be available soon.

Research also shows ROCK inhibitors may play a role in the treatment of corneal endothelial dysfunction. Several animal studies demonstrate it can

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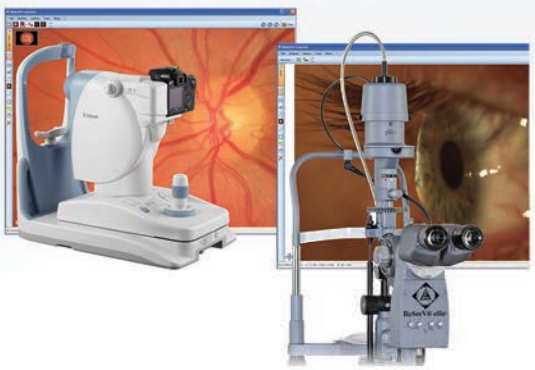

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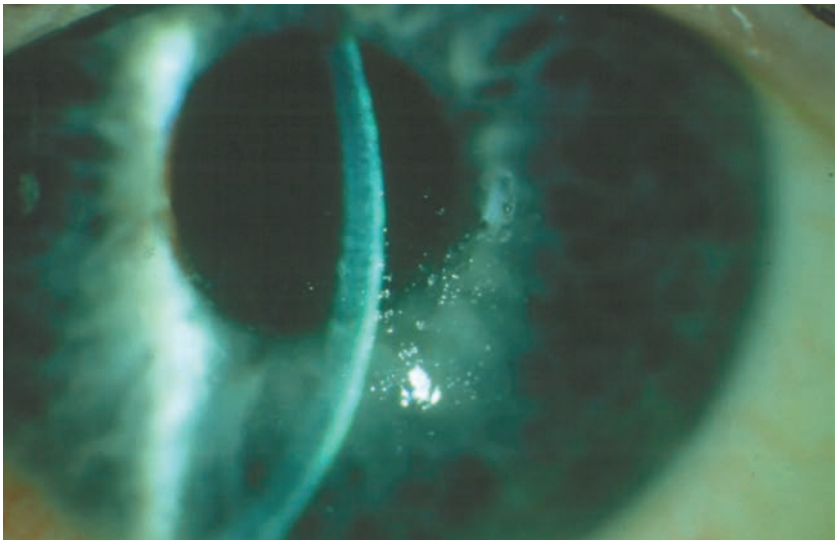


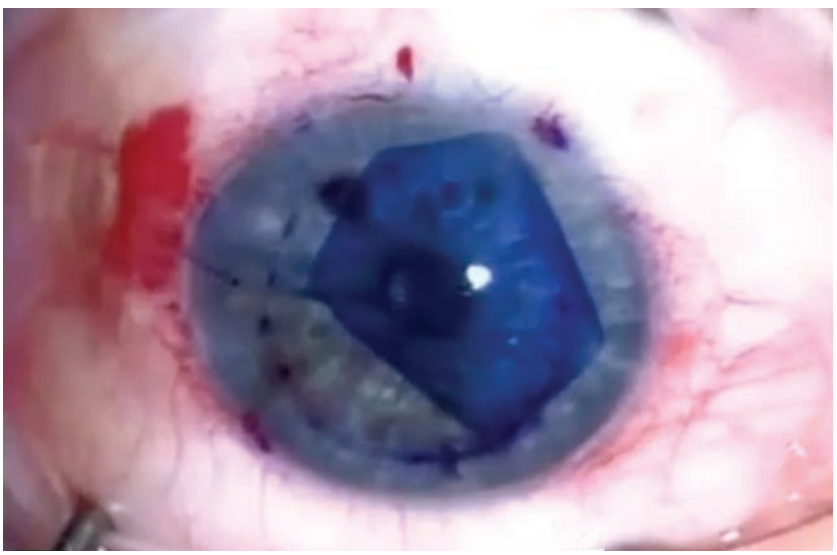
This patient was diagnosed with intermediate-stage FED with diffuse guttae and inferior stromal thickening, as demonstrated by the slit beam.

promote corneal endothelial healing in rabbit and monkey models.[5,6] After Japanese researchers demonstrated that the ROCK inhibitor Y-27632 could promote primate corneal endothelial cell proliferation *in vitro* and healing *in vivo* in animal models, they sought to test the therapy on humans.

Their 2013 case report described corneal clearance in FED after corneal endothelial denudation with cryotherapy followed by topical administration of Y-27632.[8] In this case, a 52-year-old Japanese man, diagnosed with late-onset FED, was referred as a candidate for keratoplasty. Although his right eye was clear, his left cornea showed severe edema, his vision was 20/63 OS and his central corneal thickness was 703µm OS. His midperipheral endothelial cell count was maintained at a cell count of 757cells/mm^2.

Transcorneal cryotherapy was used to destroy the central corneal endothelium and release the contact inhibition of the endothelial cells, thus allowing healthier cells in the periphery and mid-periphery to migrate to cover the denuded area. Y-27632 was compounded into an eye drop and used by the patient for one week following the endothelial destruction. By two weeks, vision had improved to 20/20 and the cornea had deturgesced. Corneal clarity was maintained throughout two years of follow-up. The researchers concluded that ROCK inhibitor eye drops may be a useful adjunct for less-invasive surgery (endothelial destruction without graft placement) for FED; however, they acknowledge they did not do the control of simply destroying the endothelium without the adjunct ROCK inhibitor and thus could not definitively isolate the effect of the ROCK inhibitor.

Photo: Jim Guzek, MD



Although advanced techniques such as DMEK have improved surgical outcomes for endothelial dysfunction patients, several novel methods—many used in conjunction with ROCK inhibitors—show promise for even better long-term visual outcomes.

This approach (endothelial destruction without placement of a corneal graft) relies on the presence of functioning endothelial cells to cover the denuded area and would not be expected to succeed in situations characterized by global depletion of endothelial cells such as pseudophakic bullous keratopathy.

Several other less-invasive therapies are under development for the treatment of endothelial dysfunction, with ROCK inhibitors used as an adjunct with several of them.

Cultured corneal endothelial cells (CECs). The same group of researchers in Japan has used CECs for transplantation following endothelial destruction in animal models.[5] Engraftment of the transplanted CECs was facilitated by

concurrent application of Y-27632. This team used monkeys whose corneal endothelium had been physically removed. Cultured monkey CECs (MCECs), in a suspension supplemented with Y-27632, were then injected into the anterior chamber, with the animal remaining in a face down position for three hours to allow the CECs to come in contact with the Descemet's membrane. Results showed that the corneal endothelium that regenerated from the combined injection of CECs and ROCK inhibitor resembled normal corneal endothelial tissue, with a monolayer of hexagonal cells and Na+/K+ ATPases. There was no indication of adverse reactions from the injection, including intraocular pressure (IOP) increases, accumulations of injected cells or rejection.[5]

Following the successful regeneration of corneal endothelial tissue using MCECs, researchers then injected cultured human CECs (HCEC) into the monkey following endothelial removal, both with and without the ROCK inhibitor.[5] While the HCEC injection without the ROCK inhibitor was unable to regenerate corneal endothelial tissue, a corneal endothelium was regenerated within one week after injection with HCECs along with the ROCK inhibitor.

Eyes injected with the HCECs and ROCK inhibitor showed corneal endothelium that resembled normal human endothelium, reaching a cell density of 2,890 cells/mm^2. Again, no increase in IOP was observed, although secondary glaucoma is a potential side effect.

As of October 2016, this team had treated 31 patients with cultured cell injection therapy and is currently preparing their human data for publication.[16] This approach opens up the possibility of dozens of CEC treatments from one donor cornea, helping to alleviate the corneal donor shortage. However, it also presents logistical challenges for regulatory approval in the United States, and peer-reviewed literature currently has little to offer on this approach in patients with endothelial dysfunction. Of course, this approach transplants donor cells and is a potential treatment for any form of endothelial dysfunction, since it does not require corneal rejuvenation by remaining host endothelium.

Rescued corneal endothelium without the use of foreign cells or tissue. The success of Descemet's stripping without endothelial keratoplasty suggests this may be a possible treatment for FED in cases where host endothelial cells are preserved in the corneal periphery.[2,7,17] In this procedure, the central area of the Descemet's membrane with confluent guttae

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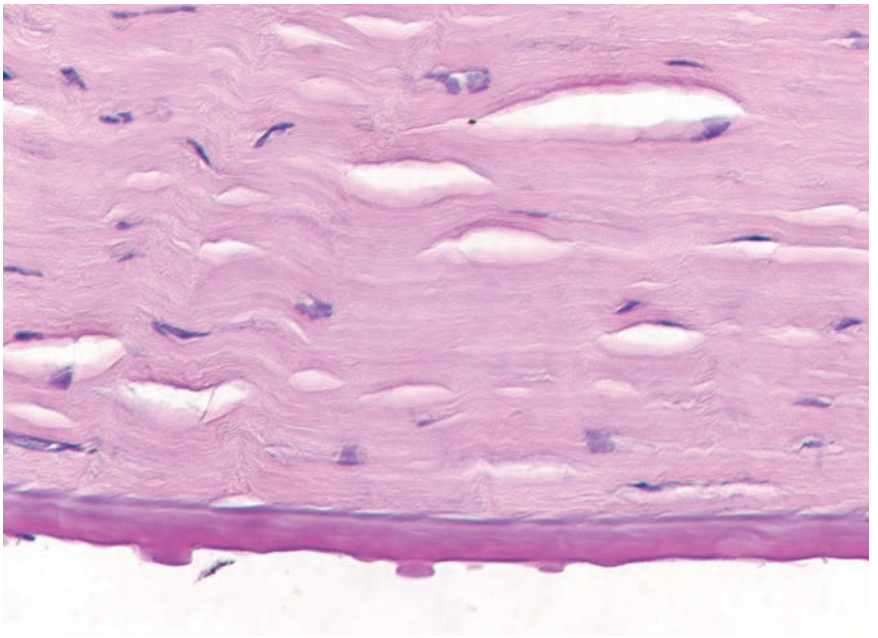

Fig. 3. This specimen, removed following corneal transplantation, shows the histopathology of corneal guttae. Note the Descemet's membrane thickening, also a hallmark of FED.

is simply removed, without placement of donor tissue. Peripheral endothelium migrates to cover the area of endothelial removal, providing corneal deturgescence without the need for foreign cells.[1,2] This approach is only applicable in conditions where there are enough host endothelial cells are preserved to rejuvenate the cornea, most commonly in FED. It will not be effective in conditions characterized by complete endothelial depletion such as pseudophakic bullous keratopathy or advanced FED.

The first large series to show success with this approach studied Descemet's stripping after cataract removal and intraocular lens insertion in patients with moderate-stage FED.[2] In this study, corneal clearance occurred in 10 of 13 cases after a 4mm central Descemet's stripping, at an average of three months (range one to six months). The three eyes that failed to clear underwent standard endothelial transplantation with good outcomes.[18]

Others have also confirmed the viability of Descemet's stripping without endothelial replacement as a treatment for FED, although it has shown variable success in a few small series by other authors.[2] Recently, one study confirmed corneal clearance in 14 of 17 FED patients after Descemet's stripping without endothelial replacement, but demonstrated a much greater success rate (10 of 10 cases) if Descemet's membrane and endothelium were removed by a smooth tear method known as descemetorhexis, rather than scoring and subsequent Descemet's stripping. This suggests physical characteristics of the area of endothelial removal (i.e., smooth rather than jagged edge) may play a role in the success of this technique.[17]

A recent study of Descemet's stripping without grafting in 12 FED eyes suggests the use of the ROCK inhibitor ripasudil as a rescue agent for patients whose corneas did not clear after endothelial removal alone.[7] In this study, nine of 12 eyes cleared spontaneously. One eye that did not clear was treated with compounded topical Y-27632 without success and eventually underwent endothelial keratoplasty with a good outcome. Two eyes had persistent edema with stalled corneal deturgescence two and three months after the Descemet's stripping. The authors applied ripasudil to these two cases and noted clearing of the corneal edema after the drop was used six times daily for two weeks.

Since this study was published in 2017, other groups have started to examine the effect of topical ROCK inhibitors as an adjunct to endothelial removal or destruction as a treatment for FED. None of these studies have reached the peer-reviewed literature yet, but preliminary data suggests topical ripasudil speeds endothelial migration (corneal clearance in six weeks rather than three months), reduces the number of cases that fail to clear and may increase the final central endothelial cell count.

Many questions remain, however, including the optimal dose and duration of ROCK inhibitors, as well as which agent is best. The recent approval of netarsudil in the United States should help the eye care community discover answers to these questions.

While modern corneal transplantation techniques are highly successful for management of endothelial failure, several novel therapies have shown promise in recent years, especially for Fuchs' dystrophy, the most common cause of endothelial dysfunction. More work

is needed to determine the optimal patient population for Descemet's stripping without endothelial keratoplasty, as well as the role of topical rho-kinase inhibitors in this procedure. Cultured endothelial cells offer the possibility of treating many patients from a single donor, although significant logistical obstacles stand in the way of widespread acceptance of this approach.

These techniques have the potential to reduce the risks associated with conventional surgical treatments, may offer options for earlier-stage endothelial dysfunction patients, especially those with Fuchs' dystrophy, and are likely to play an increasing role in the management of corneal endothelial diseases in the future. ■

Ms. Rohan is a second-year optometry student at the Illinois College of Optometry.

Dr. Colby is the Louis Block Professor and chairman of the Department of Ophthalmology and Visual Science at the University of Chicago. An internationally renown corneal surgeon, Dr. Colby is an expert in Fuchs' dystrophy and has pioneered the use of Descemet's stripping for FED management.

1. Sarnicola C, Farooq A, Colby K. Fuchs endothelial corneal dystrophy: Update on pathogenesis and future directions. Eye & Contact Lens. 2018, in press.
2. Borkar DS, Veldman PV, Colby KA. Treatment of Fuchs endothelial dystrophy by Descemet stripping without endothelial keratoplasty. Cornea. 2016;35:1267-73.
3. Alcon. Millions of patients in developing countries await corneal transplants to restore their sight. www.alcon.com/stories/millions-patients-developing-countries-await-corneal-transplants-restore-their-sight. Accessed January 4, 2018.
4. Eye Bank Association of America (EBAA). Surgical Use and Indications for Corneal Transplant Statistical Report Analysis-2016. 2017: Washington, DC.
5. Okumura N, Sakamoto Y, Fujii K, et al. Rho kinase inhibitor enables cell-based therapy for corneal endothelial dysfunction. Sci Rep. 2016 May 18;6:26113.
6. Okumura N, Kinoshita S, Koizumi N. Application of Rho kinase inhibitors for the treatment of corneal endothelial diseases. J Ophthalmol. 2017;2017:2646904.
7. Moloney G, Petsoglou C, Ball M, et al. Descemetorhexis without grafting for Fuchs endothelial dystrophy-supplementation with topical ripasudil. Cornea. 2017 Jun;36(6):642-8.
8. Koizumi N, Okumura N, Ueno M, et al. Rho-associated kinase inhibitor eye drop treatment as a possible medical treatment for Fuchs corneal dystrophy. Cornea. 2013 Aug;32(8):1167-70.
9. Du J, Aleff RA, Soragni E, et al. RNA toxicity and missplicing in the common eye disease Fuchs endothelial corneal dystrophy. J Biol Chem. 2015;290(10):5979-90.
10. Watanabe S, Oie Y, Fujimoto H, et al. Relationship between corneal guttae and quality of vision in patients with mild Fuchs' endothelial corneal dystrophy. Ophthalmology. 2015;122(10):2103-9
11. Gendron SP, Thériault M, Proulx S, et al., Restoration of mitochondrial integrity, telomere length, and sensitivity to oxidation by in vitro culture of Fuchs endothelial corneal dystrophy cells. Invest Ophthalmol Vis Sci. 2016;57(14):5926-34.
12. Jurkunas UV, Bitar MS, Funaki T, Azizi B. Evidence of oxidative stress in the pathogenesis of fuchs endothelial corneal dystrophy. Am J Pathol, 2010;177(5):2278-89.
13. Julian L, Olson MF. Rho-associated coiled-coil containing kinases (ROCK) structure, regulation, and functions. Small GTPases. 2014;5:e29846. [Epub].
14. Labandeira-Garcia JL, Rodríguez-Perez AI, Villar-Cheda B, et al. Rho kinase and dopaminergic degeneration: a promising therapeutic target for Parkinson's disease. Neuroscientist. 2015;21(6):616-29.
15. Garnock-Jones KP. Ripasudil: first global approval. Drugs. 2014;74(18):2211-5.
16. Kinoshita S. Clinical application of cultured human corneal endothelial cells. American Academy of Ophthalmology Annual Meeting. October 18th, 2016: Chicago.
17. Davies E, Jurkunas U, Pineda R 2nd. Predictive factors for corneal clearance after Descemetorhexis without endothelial keratoplasty. Cornea. Oct 17, 2017. [Epub ahead of print].
18. Rao R, Colby KA, Veldman PV. Descemet membrane endothelial keratoplasty following failed Descemet stripping without endothelial keratoplasty. Cornea. 2017;36(7):763-6.

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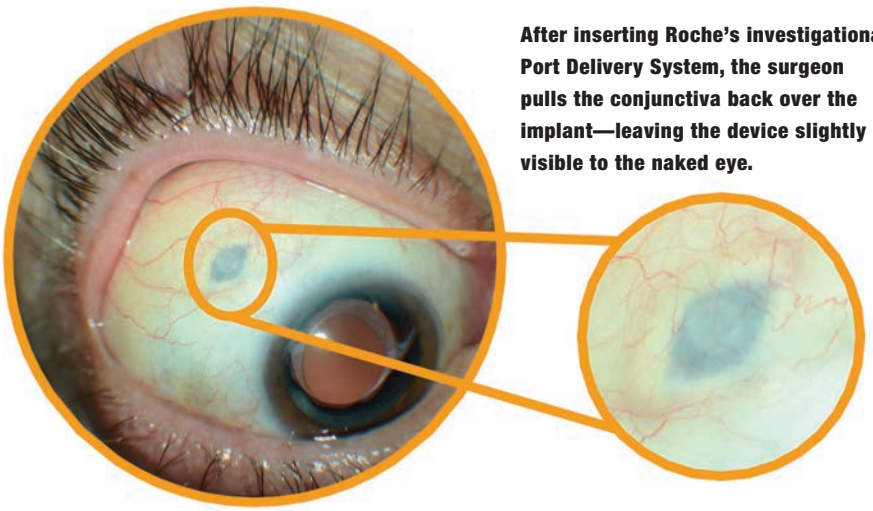
Breaking the Burden: A New Way to Deliver Anti-VEGF

An implantable drug reservoir looks to shake up today's successful but untenable protocols. **By Anat Loewenstein, MD**

Anti-vascular endothelial growth factor (VEGF) therapy has dramatically revolutionized the management of age-related macular degeneration (AMD), diabetic macular edema and retinal vein occlusions. Many trials have shown that, worldwide, the treatment has significantly changed outcomes in patients with retinal vascular disease and reduced the incidence of blindness from these conditions.[1-5] In each of these indications, the best outcomes have been shown with fixed dosing schedules.[6] However, such a regimen risks either over- or undertreatment if the chosen intervals between treatments are too short or too long.[7]



After inserting Roche's investigational Port Delivery System, the surgeon pulls the conjunctiva back over the implant—leaving the device slightly visible to the naked eye.

Numerous studies have shown the challenges of bringing the efficacy demonstrated in clinical trials into the real world.[8] Fixed monthly dosing is associated with huge burdens for the patient, the physician, the clinic and the health system. Patients experience inconvenience and infection risk each time an injection takes place. As most are elderly, they typically require the presence of a caregiver at office visits, extending the disruption to family members. Retina practices must maintain high inventories of medications and establish a tight workflow that allows high-volume dispensing. These activities consume precious physician and staff time that causes a backlog of cases to develop, delaying care for other patients. And the health case system as a whole must then process and reimburse it all.

Therefore, clinicians in everyday practice have turned to regimens such as PRN or treat-and-extend schedules.[7-9] Outcomes obtained with PRN depend largely on the

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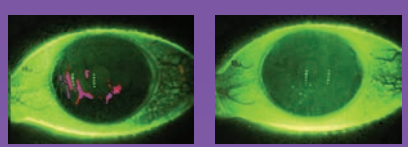

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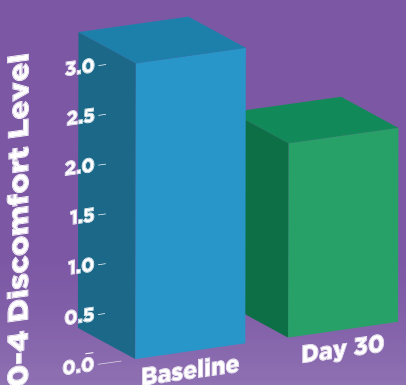
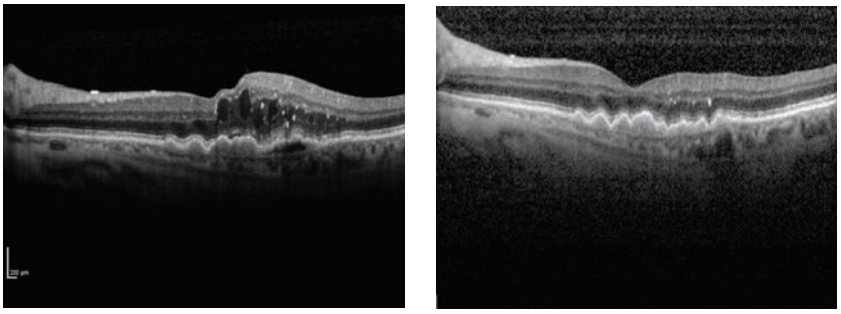


retreatment criteria employed by the treating physician and how closely these are adhered to. However, these regimens require multiple examinations, are dependent on patient compliance and do not achieve the same results as monthly treatment. The burden of treatment combined with the lack of efficacy in reactive modes of therapy creates a need for better delivery systems.

As a result, other modes of drug delivery with potentially less intervention and less need for follow up have been explored. Technologies that could extend the duration between treatments could offer a significant benefit to patients by reducing the burden of treatment while improving vision.

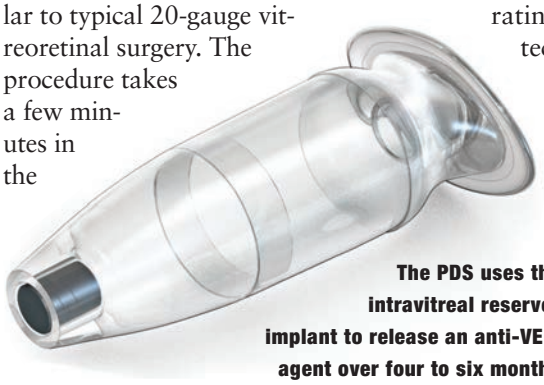


Wet AMD patient before (left) and after (right) treatment with Lucentis. The PDS hopes to maintain clinical improvements of this kind while reducing the treatment burden.

Refillable Drug Reservoir

The Port Delivery System (PDS), under development by Swiss drugmaker Roche, is a durable intravitreal reservoir implant placed through a scleral incision in the pars plana in a one-time surgical procedure.

The device, roughly the size of a grain of rice, is loaded with ranibizumab (Lucentis, Genentech) via a custom needle and then inserted using standard vitreoretinal surgical techniques. After the physician makes a small pars plana incision and inserts the device, the conjunctiva is then pulled back over the implant and secured in place, similar to typical 20-gauge vitreoretinal surgery. The procedure takes a few minutes in the operating room, followed by a short recovery period.



The PDS uses this intravitreal reservoir implant to release an anti-VEGF agent over four to six months.

The implant holds a small volume of a highly concentrated drug that is slowly released over a four- to six-month period, or perhaps longer. The device can then be refilled in the ophthalmologist's office with a custom needle that is part of the drug/device combination system.

The technology was originally developed by ForSight Vision4 to improve delivery of a variety of therapeutic payloads, including small molecules that typically stay in the eye for a short time. ForSight Vision4 is the fourth start-up company to arise through ForSight Labs, which was also responsible for developing the CyPass glaucoma stent (acquired by Alcon) and a drug-eluting periocular ring for glaucoma drug delivery (acquired by Allergan).

ForSight Vision4 began collaborating with Roche's Genentech unit in 2010. In late 2016, Roche acquired ForSight Vision4 and the PDS. The acquisition has also provided researchers the opportunity to apply the PDS technology to other molecules in the company's pipeline.

A Phase I trial involving 20 patients found that the implant was well tolerated and improved best-corrected visual acuity in a manner comparable to monthly ranibizumab injections.[10] On average, patients in this trial needed fewer than five refills over the course of a year, establishing proof of concept for the system.

Following the promising Phase I results, Genentech received FDA fast-track designation for the implant and initiated a Phase II clinical trial of the implant in patients with wet AMD. The Long Acting Delivery of Ranibizumab (LADDER) study is evaluating the safety and efficacy of the implant in patients who have previously responded to ranibizumab therapy, in hopes of reducing the number of office visits and injections while still achieving the maximum benefit possible from therapy. The multicenter, randomized controlled trial is evaluating the implant with three different ranibizumab concentrations vs. conventional delivery of ranibizumab 0.5mg.[11]

The primary study endpoint of LADDER is the time point at which a patient first requires the RPDS implant to be refilled according to protocol-defined refill criteria. The goal is to determine the pharmacodynamic relationship and the refill interval, thus defining the duration

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of the therapeutic effect between refills. The desired target for the ranibizumab port delivery system is four to six months between refills. The study also seeks to determine whether the efficacy is the same if the drug is delivered at a steady rate over long periods of time compared to the peaks and valleys of intermittent dosing. Enrollment has been completed and top-line data are expected later this year.

Using the Port Delivery System to administer ranibizumab (and potentially other molecules) in sustained-release fashion creates the potential for fewer office visits, fewer total interventions and comparable outcomes with much less burden. The technology, however, requires a scleral incision, one that should be performed for now in the operating room setting.

Different Approaches

Many other methods of reducing anti-VEGF treatment burden have been investigated. Some have failed to deliver enough promising clinical data for development to continue; others remain in testing.

One promising but ultimately disappointing effort involved looking beyond the VEGF pathway by targeting platelet-derived growth factor (PDGF), which recruits pericytes to aid formation of the neovascular matrix in wet AMD and other forms of angiogenesis. Researchers hoped to achieve an additive effect by pairing an anti-PDGF agent with conventional anti-VEGF therapy. This concept was studied for both ranibizumab in the Fovista trials and aflibercept (Eylea, Regeneron) in the Capella trial; unfortunately, Phase III and II trials respectively found that neither resulted in additional benefit to monotherapy with anti-VEGF.[12]

The topical route has also been explored. Squalamine, a small-molecule drug that targets both VEGF and PDGF, was investigated in the Phase II IMPACT study. This prospective randomized trial compared the experimental drug given topically BID in conjunction with routine ranibizumab injections vs. ranibizumab monotherapy. Though it showed encouraging results, a Phase III study of the drug published in January of this year failed to meet its primary endpoint of mean visual acuity gain at nine months.[13,14]

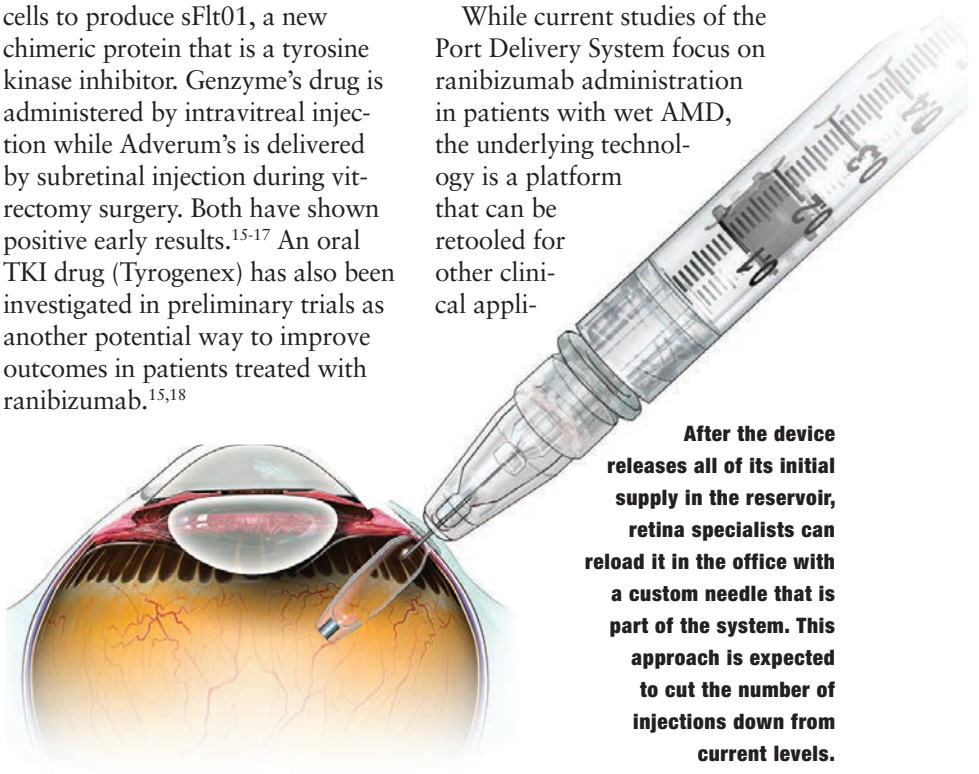
An approach actively under review involves inhibition of tyrosine kinase, a cell-signaling molecule that triggers vascular endothelial cell proliferation. Efforts seek to deliver a tyrosine kinase inhibitor (TKI) using either gene therapy or oral administration. Genzyme and Adverum Biotechnologies are each studying a promoter gene packaged in an adenoviral vector that causes cells to produce sFlt01, a new chimeric protein that is a tyrosine kinase inhibitor. Genzyme's drug is administered by intravitreal injection while Adverum's is delivered by subretinal injection during vitrectomy surgery. Both have shown positive early results.[15-17] An oral TKI drug (Tyrogenex) has also been investigated in preliminary trials as another potential way to improve outcomes in patients treated with ranibizumab.[15,18]

Researchers are evaluating numerous other receptors throughout the AMD pathophysiological cascade as potential targets in hopes of extended therapy beyond VEGF-based efforts. They are also investigating alternative forms of drug delivery for both established and investigational molecules.

Time to Evolve

The development of anti-VEGF for AMD in 2005 was revolutionary, a true "sea change" that radically reshaped the delivery of care and patient outcomes. But, more than a decade on, the weaknesses of the current model are increasingly evident. The R&D community is now engaged in a global effort to find the next big breakthrough that will maintain acuity gains for patients with greater predictability while reducing the treatment burden and associated costs. In the absence of another revolution, incremental gains are always welcome.

While current studies of the Port Delivery System focus on ranibizumab administration in patients with wet AMD, the underlying technology is a platform that can be retooled for other clinical appli-



After the device releases all of its initial supply in the reservoir, retina specialists can reload it in the office with a custom needle that is part of the system. This approach is expected to cut the number of injections down from current levels.

cations. The volume of the reservoir and the elution rate of drug release can be modified to suit different sizes and types of molecules. Thus, the PDS technology could unlock new treatment options for wet AMD patients as well as others, while also helping physicians and healthcare systems worldwide. ■

Dr. Loewenstein is a professor of ophthalmology and deputee dean of the medical school at the Sackler Faculty of Medicine, Tel Aviv University, and chair of the department of ophthalmology at the Tel Aviv Sourasky Medical Center.

1. Bloch SB, Larsen M, Munch IC. Incidence of legal blindness from age-related macular degeneration in Denmark: year 2000 to 2010. Am J Ophthalmol. 2012;153:209-13.
2. Skaat A, Chetrit A, Belkin M, et al. Time trends in the incidence and causes of blindness in Israel. Am J Ophthalmol. 2012;153:214-21.
3. Borooah S, Jeganathan VS, Ambrecht AM, et al. Long-term visual outcomes of intravitreal ranibizumab treatment for wet age-related macular degeneration and effect on blindness rates in south-east Scotland. Eye (Lond). 2015;29:1156-61.
4. Bressler NM, Doan QV, Varma R, et al. Estimated cases of legal blindness and visual impairment avoided using ranibizumab for choroidal neovascularization: non-Hispanic white population in the United States with age-related macular degeneration. Arch Ophthalmol. 2011;129:709-17.
5. Holz FG, Tadayoni R, Beatty S, et al. Determinants of visual acuity outcomes in eyes with neovascular AMD treated with anti-VEGF agents: an instrumental variable analysis of the AURA study. Eye (Lond). 2016;30:1063-71.
6. Lanzetta P, Mitchell P, Wolf S, Veritti D. Different antivascular endothelial growth factor treatments and regimens and their outcomes in neovascular age-related macular degeneration: a literature review. Br J Ophthalmol. 2013;97:1497-1507.
7. Schmidt-Erfurth U, Eldem B, Guymer R, et al. Efficacy and safety of monthly versus quarterly ranibizumab treatment in neovascular age-related macular degeneration: the EXCITE study. Ophthalmology. 2011;118:831-9.
8. Lanzetta P, Loewenstein A, Vision Academy Steering Committee. Fundamental principles of an anti-VEGF treatment regimen: optimal application of intravitreal anti–vascular endothelial growth factor therapy of macular diseases. Graefes Arch Clin Exp Ophthalmol. 2017;255:1259-73.
9. Essex RW, Nguyen V, Walton R, et al; Fight Retinal Blindness Study Group. Treatment patterns and visual outcomes during the maintenance phase of treat-and-extend therapy for age-related macular degeneration. Ophthalmology. 2016;123:2393-2400.
10. Laganovska G. et al. First-in-human results of a refillable drug delivery implant providing release of ranibizumab in patients with neovascular AMD. Paper presented at American Academy of Ophthalmology, Retina Subspecialty Day; November 9, 2012; Chicago IL.
11. Dreyer RF. Sustained delivery of ranibizumab: The LADDER trial of the ranibizumab port delivery system. Presented at: American Society of Retina Specialists 34th Annual Meeting, Aug. 9-14, 2016. San Francisco, Calif.
12. Dunn EN, Hariprasad SM, Sheth VS. An overview of the Fovista and Rinucumab trials and the fate of anti-PDGF medications. Ophthalmic Surg Lasers Imaging Retina. 2017;1;48(2):100-4.
13. Ohr Pharmaceutical. Ohr Pharmaceutical presents data from OHR-102 Phase II IMPACT Study in wet-AMD at ARVO conference. www.ohrpharmaceutical.com/media-center/press-releases/detail/446/ohr-pharmaceutical-presents-data-from-ohr-102-phase-ii. Accessed January 31, 2018.
14. Ohr Pharmaceutical. Ohr Pharmaceutical announces efficacy results from the MAKO Study in wet-AMD. https://globenewswire.com/news-release/2018/01/05/1284092/0/en/Ohr-Pharmaceutical-Announces-Efficacy-Results-from-the-MAKO-Study-in-Wet-AMD.html. Accessed January 31, 2018.
15. Falavarjani KG, Sadda SR. Hot topics in pharmacotherapy for neovascular age-related macular degeneration.
16. Avalanche Biotechnologies, Inc. announces positive top-line Phase 2a results for AVA-101 in wet age-related macular degeneration. http://investors.adverum.com/news-releases/news-release-details/avalanche-biotechnologies-inc-announces-positive-top-line-phase. Accessed January 31, 2018.
17. Heier JS. Preliminary results of phase 1 study with AAV2-sFLT01 as gene therapy for treatment of exudative AMD: one-year results of phase 1 clinical trial with rAAV.sFLT-1. The Retina Society 2014 Annual Meeting. Sept. 11-14, 2014. Philadelphia.
18. Chaudhry N. Oral VEGF receptor/PDGF receptor inhibitor X-82. Paper presented at the American Academy of Ophthalmology Annual Meeting; November 14-17, 2015; Las Vegas, NV.

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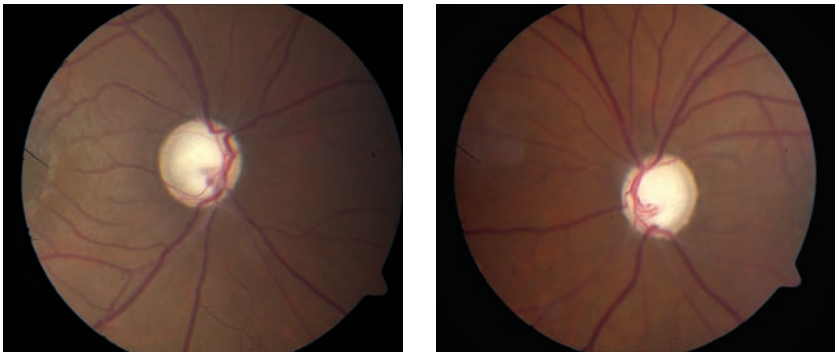
PEER • REVIEWED • RRA • ARTICLE

Prospects for Neuroprotection in Glaucoma

Investigators are gunning for glaucoma. Here, one shares some thoughts from the frontlines. **By Sylvia L. Groth, MD**

I had the pleasure of seeing the most delightful patient recently. A gentleman in his mid 70s, he was presenting for a follow-up visit for our current clinical trial of recombinant human nerve growth factor (rhNGF) eye drops at Byers Eye Institute at Stanford. The goal of this medication is to preserve the life of retinal ganglion cells (RGCs) and slow the progression of visual field (VF) loss. As I reviewed his VFs and data from the prior months, my heart sank. He had devastatingly advanced, end-stage glaucoma. His VFs were dark, except for the smallest central island. When we spoke, he began asking me several questions that we researchers ask ourselves—"Why does my disease progress even though I've never had a pressure greater than 12mm Hg? Are other factors at play aside from the eye pressure? Are we going to find a way to treat people like me?"

Finding those answers is specifically what my research team is working toward as we investigate neuroprotection and vision restoration for glaucoma.



If promising new research is on the right track, patients may be able to avoid this kind of glaucomatous optic nerve damage in the future.

Reviving Ganglion Cells

This is an exciting time to be part of glaucoma research. We have 60 patients enrolled in an eight-month, double-masked, randomized Phase Ib clinical trial testing rhNGF, including treatment and placebo controlled arms (clinicaltrials.gov, #NCT02855450). The patients included have clinical evidence of progressive glaucomatous RGC dysfunction based on VF and structural imaging modalities. The trial requires residual VF preservation in at least one quadrant—i.e., something left to save. Exclusion criteria include vision loss from other causes and recent intraocular surgery.

The design is atypical for glaucoma trials in that the primary outcome is not measured in intraocular pressure (IOP) but rather visual function. We are measuring functional endpoints including visual acuity, VF and electrophysiology, and structural endpoints including RGC layer thickness by optical coherence tomography. Translating laboratory-based research into clinical-based research, we are working with glaucoma patients at all stages. Many see this trial as their last line of hope, although other similarly

designed trials investigating other therapies are also in progress at Stanford and a few other centers. These trials are testing whether candidate treatments could revive sick, poorly functioning RGCs and enable them to avoid ultimate death, but on a non-IOP mediated basis. What we are learning will hopefully be applied to future patients at earlier stages of their disease, leading to more preservation of functional vision.

The ability to maneuver in an environment without bumping into objects, to drive a car, to read printed material are all activities that many of us take for granted. The aim of these studies is to find new therapies that could halt glaucoma before it becomes debilitating.

Under Pressure

As we know, glaucoma is a neurodegenerative disease that causes an optic neuropathy, mostly affecting RGCs and leading to visual field defects. It is irreversible and can be devastating. Estimates predict glaucoma patients worldwide in 2020 will reach 79.6 million, with 5.9 million people estimated to be bilaterally blind from primary open-angle glaucoma (POAG).[1]

Despite diligent research, we simply don't understand much about the pathophysiology of glaucoma. We have evidence that IOP is an important risk factor but, perhaps more importantly, we now realize that it is merely one factor.[2] As with my patient, some with the disease maintain low pressures yet continue to progress. Through ocular hypotensive therapy, surgical or laser intervention, or both, these patients maintain a low target pressure range; however, their glaucoma continues to advance and in some cases ultimately leads to loss of functional vision or blindness. Questions physicians should be asking include 'how much do vascular and blood flow factors affect the glaucomatous damage of the optic nerve and how can we use those factors for treatment?' 'Are we dealing with distinct pathologic processes that result in diverse trajectories and how can we identify the patients in each group?' 'Are there new therapeutic avenues we should pursue, such as neurotrophic factors, to prevent RGC death?'

As we have realized the limitations of IOP lowering in glaucoma, neuroprotection has emerged as a critical area of research. In glaucoma, neuroprotection has been defined as an intervention independent of IOP reduction that prevents RGC death.[3] As a 2012 investigation shows, the ideal time to protect neurons is before extensive damage is done.[4] We unfortunately don't catch all patients at that early stage, but intervention must at least be before near complete or complete RGC death. Though the balance of the discussion will relate to therapies for neuroprotection, early detection and prevention are essential components of glaucoma care.[3,4]

Potential Pharmaceuticals

Thanks to the persistent work of many scientists around the world, we have drug targets and candidate therapies that inspire hope for better patient outcomes. Though much of this research is still taking place in the basic science labs, it is moving into the clinical realm. These are some of the agents researchers are investigating:

Brimonidine. One of the initial trials of neuroprotection looked at brimonidine as not only an IOP-lowering medication but also as a medication with a neuroprotective effect through its action on G-protein coupled receptor (GPCR) on RGCs. Although it is difficult to uncouple the neuroprotective and IOP-lowering effects, one study showed that both brimonidine and timolol had similar measurable IOP effects, but only brimonidine demonstrated preservation of visual fields.[5] This still needs to be demonstrated in humans with repeatable prospective randomized clinical trials.

Glutamate receptors. An early neuroprotection trial looked at memantine, a selective non-competitive N-methyl-D-aspartate (NMDA) channel antagonist valuable as a treatment in Alzheimer's disease.[6] NMDA channels are ionotropic glutamate receptors, and elevated levels of glutamate can contribute to excitotoxicity and neurodegeneration.[7] Memantine is able to block higher levels of glutamate that lead to excitotoxicity.[7] Unfortunately, in the Phase III clinical trial, there was no convincing evidence of a significant effect vs. the control group with the primary outcome measure being visual field function.[7] Presently, glutamate as a neuroprotective agent is currently just a hypothesis.[8]

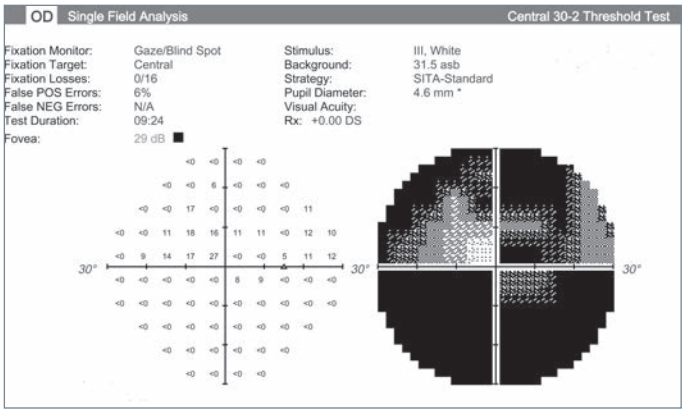
Neurotrophic factors. These promising agents halt the loss of RGCs by stopping apoptotic pathways or inhibiting intracellular death signals.[9] They act both in the central and peripheral nervous systems. Numerous classes of neurotrophic factors (NTF) modulate these pathways.[10] The challenge has been identifying which are most useful to the RGCs and how to get them into the correct location with sustained delivery or continuous secretion.[11] One theory on the advancement of glaucoma is that the increased IOP or other forces are blocking the transport of appropriate NTFs to the RGCs, causing cell death.

In addition to NTF discussed above and in clinical testing at Stanford, ciliary-derived neurotrophic factor (CNTF), brain-derived neurotrophic factor (BDNF) and glial-derived neurotrophic factor (GNTF) are all players in RGC survival in pre-clinical models.[12,13] Limitations on delivery have included short *in vivo* half-life and low bioavailability due to these biologics' larger molecular weight.[14]

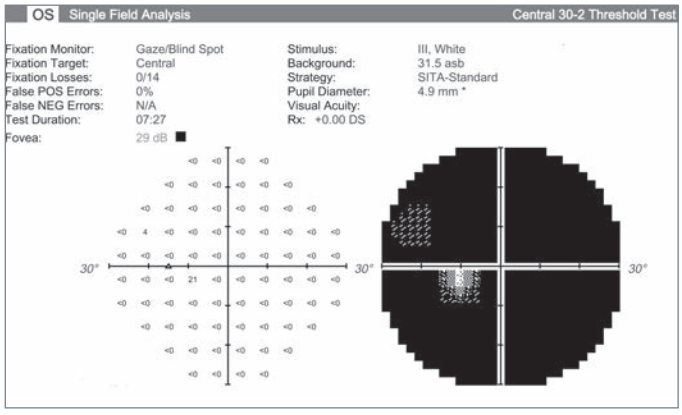
One suggested solution is to insert a slow-release device intravitreally that will continue to release the factors; for example, using encapsulated cell therapy. One such device is the CNTF secreting device NT-501, currently in a Phase 2 clinical trial at the Byers Eye Institute at Stanford, Columbia University in New York and the Glaucoma Associates of Texas in Dallas (clinicaltrials.gov #NCT02862938).

We are surgically inserting this implant into patients and then following their course of glaucoma progression. With the insert sitting in the vitreous, it is slowly releasing CTNF on a continuous basis. We hypothesize that this will have ongoing effect and prolong RGCs' survival and promote RGC function. Viral vectors have also been in development as a means to deliver CNTF and BDNF to target cells.[15]

Tumor necrosis factor-α. Another known contributor to glaucoma progression, TNF-α interacts with two main receptors, TNF-R1 and TNF-R2.[16] TNF-R1 is centrally involved in apoptosis of cells, and it was shown to have elevated expression in an ocular hypertension model in rodents.[16,17] Higher concentrations of TNF-α have been found in glaucomatous eyes vs. controls.[18] Though this connection has been described and some immune-modulating drugs are approved for clinical use in humans with other diseases, no translational clinical trials target this particular pathway for glaucoma.



Visual fields like these from an end stage glaucoma patient show the lack of function we're hoping neuroprotective advancements can halt.



Heat shock proteins (HSPs). These are one component of cellular defense, and they assist in functions as chaperones, anti-apoptosis agents and signaling transducers. Types of HSPs exist in crystalline lenses, and rodent models show a neuroprotection and neuroregeneration effect with puncture of the lens capsule.[19] A large cascade of activity was set off by the release of these proteins, including neuroinflammation and activation of glial components.[20] We have not as yet developed a way to parlay this into clinical practice, but it is an interesting natural response to this incident and seems like a hopeful pathway for future development.

Nitric oxide (NO). This can mediate IOP-lowering effects through increase of aqueous outflow through the trabecular meshwork and Schlemm's canal, the conventional pathway.[21] However, it has been hard to find evidence of NO mediating neuroprotection of the ganglion cells.

One medication being investigated, nipradilol, is an alpha-beta blocker and donor of NO, an antagonist of alpha-1 and beta-1 and 2 adrenoreceptors. We know that nipradilol relaxes smooth muscle from the release of NO and also has antagonistic properties that inhibit muscle contraction. Research shows that glaucomatous eyes may have a baseline dysfunction in their NO pathways, creating decreased blood flow to the optic nerve head.[22] Other studies demonstrate the slowing of RGC death in an optic nerve crush model in rats with nipradilol and also suggested that some of the neuroprotection may come from increased retinal blood flow properties.[23,24] In addition, increased blood flow in the optic nerve head can result from treatment with nipradilol.[25]

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There may be many benefits from incorporating NO or NO moieties into medical treatment for glaucoma. An exciting development in this area is the recent FDA approval of latanoprostene bunod, a molecule that acts as a prostaglandin analog and as an NO donor.

Stem cells. A common question patients ask me is if stem cells are able to restore their vision in glaucoma. This is a reasonable line of inquiry, since medicine has seen many advancements in the use of stem cells. While there is promise in this field, there are also substantial limitations. One of these limitations is getting those stem cells to connect with the brain itself.

The brain is a complex set of circuitry that is not easy to plug into, so we are a ways off on getting stem cells to take the place of dead or dying cells in the CNS. However, it does look like we can enhance survival of connected, living RGCs with stem cells.[26] One study using intravitreally injected mesenchymal stem cells (MSCs) in rats resulted in prolonged survival of RGC axons. There was no effect on the RGC axons when the MSCs were injected intravenously.[27] This was done in rats with glaucomatous optic nerve injury in a laser-induced ocular hypertensive model.

One mechanism by which stem cells would have beneficial effect on the RGCs is by continuous release of neurotrophic factors, which slow down the degeneration.[28] Factors that can be secreted from stem cells include CNTF, fibroblast growth factor and GNTF.[29] There may even be more factors released that have not yet been characterized. A concern some researchers have discussed: if we haven't mapped all the possible neurotrophic factors that may be released, it is possible these stem cells could be secreting compounds that actually have deleterious effect on the optic nerve. Therefore, we should be sure we have fully characterized the NTFs that come from a line of cells before we use them.[30]

A New Hope

We're living in a time of tremendous development in glaucoma research. Much of it is currently transitioning from animal models to human trials. What we should take from this and bring to the next glaucoma patient we have in our chairs is hope. We can confidently say that we are getting closer to a solution for them and their younger family members. I was happy to share that message with my patient. We don't have the complete answer today, but I now know, more than ever, that we are taking appropriate steps toward bringing these treatments to the millions of patients that so desperately need them.

That is the excitement of neuroprotection research and, at some future stage we hope, neuroregeneration itself. Now, part of the thrill of treating glaucoma is the feeling that our treatment options—and patients' prognosis for vision—are on the cusp of changing. ■

Dr. Groth is a clinical instructor of ophthalmology and glaucoma fellow at Byers Eye Institute at Stanford University.

1. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol. 2006;90(3):262-7.
2. Toris CB, Gelfman C, Whitlock A, et al. Making basic science studies in glaucoma more clinically relevant: the need for a consensus. J Ocular Pharm Therap. 2017;33:501-18.
3. Weinreb R, Levin L. Is neuroprotection a viable therapy in glaucoma? Arch Ophthalmology. 1999 Nov;117(11):1540-4.
4. Chader G. Advances in glaucoma treatment and management: neurotrophic agents. Invest Ophthalmol Vis Sci. 2012 May 4;53(5):2501-5.
5. Krupin T, Lieberman J, Greenfield D, et al. A randomized trial of brimonidine versus timolol in preserving visual function: results from the Low-Pressure Glaucoma Treatment Study. Am J Ophthalmol. 2011;151:671-81.
6. Reisberg B, Doody R, Stoffler A, et al. Memantine in moderate-to-severe Alzheimer's disease. N Engl J Med. 2003;348:1333-41.
7. Hare W, WoldeMussie E, Lai R, et al. Efficacy and safety of memantine, an NMDA-type open-chanel blocker, for reduction of retinal injury associated with experimental glaucoma in rat and monkey. Surv Ophthalmol. 2001 May;45:S284-9.
8. Casson R. Possible role of excitotoxicity in pathogenesis of glaucoma. Clin Exp Ophthalmol. 2006 Jan-Feb;34(1):54-63.
9. Chader G. Advances in glaucoma treatment management: Neurotrophic agents. 2012 May;53(5)2501-5.
10. He S, Stankowska DL, Ellis DZ, et al. Targets of neuroprotection in glaucoma. J Ocular Pharm Therap. 2017;00. [Epub].
11. Nafissi M, Foldvari M. Neuroprotective therapies in glaucoma: I. Neurotropic factor delivery. Nanomed Nanobiotechnol. 2016;8:240-4.
12. Ko M, Hu D, Ritch R. Patterns of retinal ganglion cell survival after brain-derived neurotrophic factor administration in hypertensive eyes of rats. Neurosci Lett. 2001;305:139-42.
13. Fu Q, Li X, Yip H. Combined effect of brain-derived neurotrophic factor and LINGO-1 fusion protein on long-term survival of retinal ganglion cells in chronic glaucoma. Neuroscience. 2009;162:375-82.
14. Domenici L, Origlia N, Falsini B, et al. Rescue of retinal function by BDNF in a mouse model of glaucoma. PLoS One. 2014;9:e115579.
15. Pease M, Zack D, Berlinicke C, et al. Effects of CNTF on retinal ganglion cell survival in experimental glaucoma. Invest Ophthal Vis Sci. 2009;50(5):2194-200.
16. Tezel G. TNF-alpha signaling in glaucomatous neurodegeneration. Prog Brain Res. 2008;173:409-21.
17. Yang Z, Quigley H, Pease M. Changes in gene expression in experimental glaucoma and optic nerve transection: the equilibrium between protective and detrimental mechanisms. Invest Ophthalmol Vis Sci. 2007;48(12):5539-48.
18. Tenzel G, Li L, Patil R, Wax M. TNF-alpha and TNF-alpha receptor-1 in the retina of normal and glaucomatous eyes. Invest Ophthalmol Vis Sci. 2001;42(8):1787-94.
19. Fischer D, Pavlidis M, Thanos S, et al. Cataractogenic lens injury prevents traumatic ganglion cell death and promotes axonal regeneration both in vivo and in culture. Invest Ophthalmol Vis Sci. 2000;41(12):3943-54.
20. Lorber B, Berry M, Logan A. Lens injury stimulates adult mouse retinal ganglion cell axon regeneration via both macrophage- and lens-derived factors. Eur J Neurosci. 2005;21(7):2029-34.
21. Borghi V, Bastia E, Guzzetta M, et al. A novel nitric oxide releasing prostaglandin analog, NCX 125, reduces intraocular pressure in rabbit, dog and primate models of glaucoma. J Ocul Pharmacol Ther. 2010;26(2):125-132.
22. Polak K, Luksch A, Berisha F. Altered nitric oxide system in patients with open-angle glaucoma. Arch Ophthalmol. 2007;125(4):494-98.
23. Karim Z, Sawada A, Mizuno K, et al. Neuroprotective effect of nipradiol [3,4-dihydro-8-(2-hydroxy-3-isopropylamino)-propoxy-3-nitroxy-2H-1-benzopyran] in a rat model of optic nerve degeneration. J Glaucoma. 2009;18(1):26-31.
24. Kida T, Suguyama T, Harino S, et al. The effect of nipradilol, an alpha-beta blocker, on retinal blood flow in healthy volunteers. Curr Eye Res. 2001;23(2):128-132.
25. Fukukita M, Ido M, Osawa S, et al. Retrobulbar hemodynamic effects of nipradilol in normal and normal-tension glaucoma eyes. J Ophthalmol. 2011;2011:652904.
26. Johnson TV, Martin KR. Cell transplantation approaches to retinal ganglion cell neuroprotection in glaucoma. Curr Opin Pharmacol. 2013;13(1):78-82.
27. Johnson T, Bull N, Hunt D, et al. Neuroprotective effects of intravitreal mesenchymal stem cell transplantation in experimental glaucoma. Invest Ophthalmol Vis Sci. 2010 Apr;51(4):2051-9.
28. Dahlman-Noor A, Vijay S, Jayaram H. Current approaches and future prospects for stem cell rescue and regeneration of the retina and optic nerve. Can J Ophthalmol. 2010;45(4):333-341.
29. Wang N, Zeng M, Ruan Y, et al. Protection of retinal ganglion cells against glaucomatous neuropathy by neurotrophin producing, genetically modified neural progenitor cells in a rat model. Chin Med J (Engl). 2002;115(9):1394-400.
30. Greco S, Rameshwar P. Microenvironment considerations in the application of human mesenchymal stem cells in regenerative therapies. Biologics. 2008;2(4):699-705.

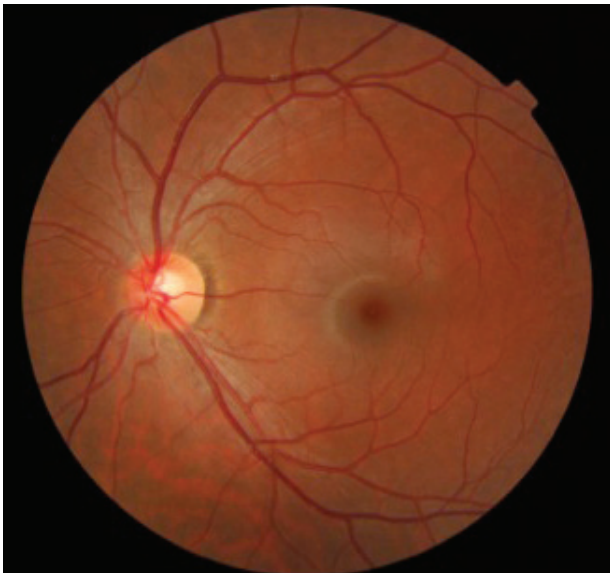
PEER • REVIEWED • PRA • ARTICLE

AI for DR: "The Digital Doctor Will See You Now"

Software that can detect ocular anomalies may offer new ways to reduce the loss of vision due to diabetic retinopathy.

By Thomas A. Wong, OD, Amy Steinway, OD, Kim Poirier, OD, and Jennifer Gould, OD

The ability to design technology that can mimic some elements of human cognition, once purely the stuff of science fiction, is becoming a reality.[1] Medicine generally and eye care specifically are among the facets of daily life that stand to be transformed by this technology, allowing doctors to become more accessible and efficient.[2] The successful optometric practice of the future will be one that chooses to see artificial intelligence (AI) not as a threat but rather as a tool to enhance uniquely human skills such as intuition and insight that are the hallmark of a good doctor.



Deep learning software can help AI systems identify the difference between a normal retina, as seen here, and one with signs of diabetic retinopathy.

"Medicine begins where the technology ends," bioethicist Edmund Pellegrino said 30 years ago, and it remains true today.[3]

But how will this disruptive technology evolve, and where will it fit into modern inter-professional health care teams? No doubt AI will affect many aspects of care, but the first frontier will be our diabetes patients. In fact, it's happening already. Efforts are underway in the United Kingdom, India, Australia and Silicon Valley, among other sites. Interestingly, at least two projects involve teen prodigies partnering with medical and computer science pros.

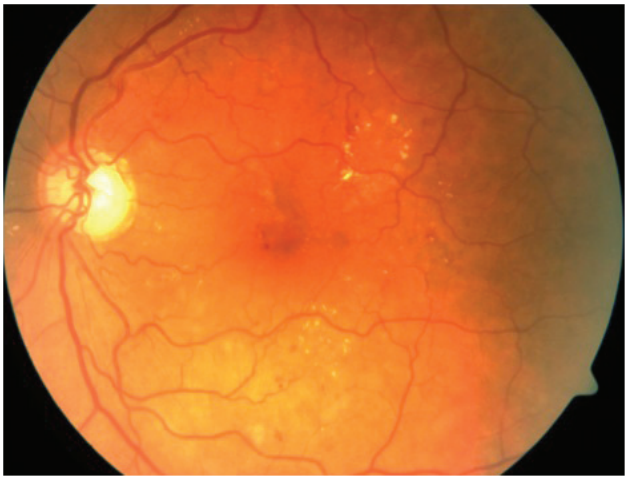
Tech-assisted Diagnosis

Diabetic retinopathy (DR) screening may be the optometric clinical responsibility best suited for augmentation by AI, given certain inescapable facts about the population affected and their access to care—or lack thereof.

For one, the prevalence of diabetes is increasing. Epidemiologically, the worldwide diabetes population grew from 153 million in 1980 to 347 million in 2008, and 25.6 million, or 11.3%, of the US population in 2010 had diabetes.[4] Estimates show that nearly 86% of Type 1 diabetes mellitus (DM) patients and 40% of Type 2 DM patients have

DR.[5] Furthermore, a high association exists between disease duration and loss of vision.

Despite these numbers, such patients are woefully underserved in the current health care system. Research estimates more than one-third of all adult diabetes cases in the United States remain undiagnosed.[6] And an American Optometric Association survey found that four out of five Americans don't realize that diabetic eye disease has no visible symptoms in the early stages.[7] Lack of awareness and difficulty accessing routine eye care leads many patients to remain undiagnosed until later in the course of the disease.



These new tools may one day provide screening services to underserved populations, giving doctors the chance to catch signs of advancing disease much earlier and avoid severe nonproliferative DR, as seen here.

Technology has already made small improvements to the care of patients with DR. Digital imaging makes serial evaluation of images easier and more collaborative. Computer-navigated surgical tools improve the safety of laser surgery for proliferative DR.[8] And at the 2017 Association for Research in Vision and Ophthalmology meeting, surgeons demonstrated the first successful use of a remote-controlled robotic system in the human eye during retinal surgery, with fewer complications in the robot-assisted group compared with the standard manual approach.[9]

But the truly revolutionary frontier lies in efforts to develop software that can screen at-risk patients and identify the earliest signs of disease. Successes in cardiology may give us a sense of what's in store for eye care. Computer scientists at Stanford University have developed algorithms that can diagnose heart arrhythmias from electrocardiography signals. The algorithm is cardiologist-level accurate, not subject to fatigue and can detect arrhythmias continuously with real-time efficiency.[10] The technology, placed in a device at-risk patients wear at all times, can alert emergency services to serious heartbeat irregularities as they happen.

Deep Thoughts on DR

The essence of computer-based health screening is reducing fundamental diagnostic dilemmas to mere data analysis problems. In DR research, AI software is fed a huge database of images and trained to distinguish between healthy and DR-affected eyes by teaching it to recognize the characteristics of each.

While the ability to compare images to normative databases and identify outliers has been a feature of modern medicine for years, newer software algorithms employ so-called deep learning, in which analysis takes place incrementally at multiple levels, beginning at the most rudimentary and progressing with ever-increasing sophistication.

In image detection, AI software may begin by assessing just a few pixels from a tiny section of the image, noting those relationships and assigning a weight or likelihood that the image meets the target identity; in this case, diabetic retinopathy. It then polls the next level of analysis (for instance, advancing from pixels to simple shapes) to see if it too fits the pattern in question, then moves up another level, and so on. Eventually, a confluence of validating findings arises among multiple levels and the image is classified.[11] Along the way, the software learns which associations are seen most often and applies that new knowledge to the next scan. It is this self-improving capability that earns it the labels *machine learning* or *artificial intelligence*. The more layers of abstraction in the algorithm, the "deeper" the learning—and the more accurate the result.

In one retrospective study, a deep-learning software algorithm analyzed a database of 1,748 retinal fundus photos from 874 diabetes patients. Results showed 96.8% sensitivity and 87% specificity for DR, which represented a 30% greater specificity than software analysis that lacked deep learning.[12]

Another project used 54 ophthalmologists to evaluate 128,175 retinal images for the presence of DR and diabetic macular edema, as well as image quality.[11] The DR severity (none, mild, moderate, severe or proliferative) was graded according to the International Clinical Diabetic Retinopathy scale. An AI algorithm was designed to identify 'referable' cases—defined as moderate or worse DR—as a way to demonstrate its viability as

a screening tool. Its performance in several tests yielded sensitivities for referable DR that ranged from 87% to 97.5%, and specificity of 94%. The study authors conclude, "these results demonstrate that deep neural networks can be trained, using large data sets and without having to specify lesion-based features, to identify diabetic retinopathy or diabetic macular edema in retinal fundus images with high sensitivity and high specificity." They also note that an algorithm's sensitivity and specificity "can be tuned to match requirements for specific clinical settings, such as high sensitivity for a screening setting."[11]

AI in the Exam Room

Several groups have met with success in modeling deep-learning AI to DR screening. Players range from big fish in computer science to high school students. It's too soon to tell which, if any, of these efforts will be commercialized and widely accepted by medical professionals, but here's a look at early movers in AI for DR.

• ***Google's DeepMind.*** London-based AI firm DeepMind, acquired by Google in 2014, is working with Moorfields Eye Hospital to develop algorithms for early detection of DR and age-related macular degeneration. The team is reviewing fundus photos and optical coherence tomography (OCT) scans from more than one million exams that took place at Moorfields in 2017.[13] In addition to imaging, the dataset will also include each patient's demographic information, disease status, treatment history, imaging devices used and time elapsed between visits. By combining imaging with exam data, the team is exploring not just automated grading of fundus photos and OCT scans, as in other AI efforts, but also the feasibility of developing "novel quantitative measures for specific disease features and for monitoring the therapeutic success."[13] The work is ongoing and results have yet to be announced.

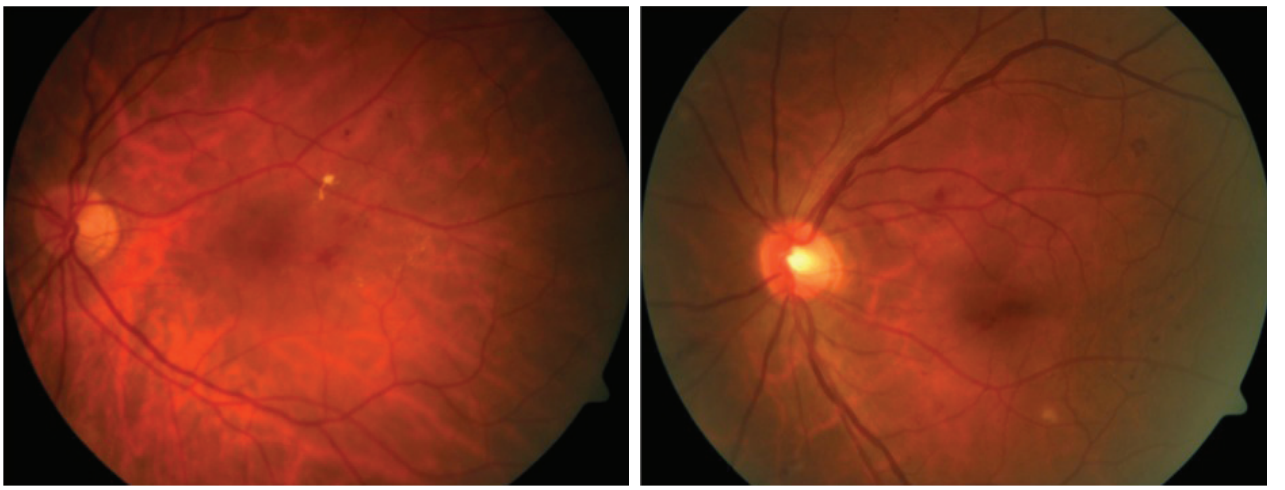
• ***EyeLogic.*** A smartphone app now under development, this uses an algorithm built from analysis of more than 75,000 fundus images found in the Eye Picture Archive Communication System (EyePACS) telemedicine database. Published results show 94% sensitivity and 98% specificity for diabetic retinopathy.[14] Given that the technology identified cases that need further evaluation and treatment with high reliability, the authors conclude that a fully data-driven AI-based grading algorithm has the potential to be used to screen fundus appearance in patients with diabetes.

Created by 18-year-old Rishab Gareya of San Jose, Calif., the work earned him a $50,000 scholarship to Stanford.

• ***Eyeagnosis.*** A second teen techie, 16-year-old Kavya Kopparapu of Herndon, Va., created an app that uses a 3D-printed lens as a smartphone accessory to enable self-administered eye exams. The AI was designed using off-the-shelf Microsoft code and a database of 34,000 retina scans from the National Institutes of Health.[15]

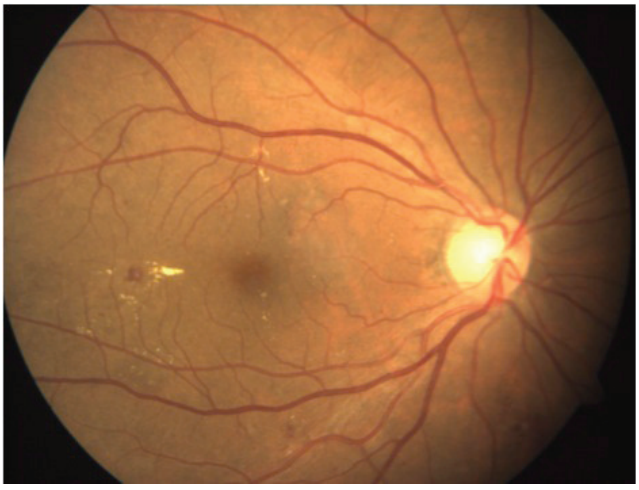
Tests performed at a hospital in Mumbai found the app can "spot diabetic retinopathy with the accuracy of a human pathologist," according to *IEEE Spectrum*.[15] It also can identify retinal microaneurysms. The app remains in testing.

• ***Dr. Grader.*** Australia's Commonwealth Scientific and Industrial



By teaching AI software to distinguish from mild (left) and moderate (right) non-proliferative DR, researchers demonstrated an AI algorithm's viability as a screening tool. The system identified 'referable' cases—moderate or worse DR—with sensitivities ranging from 87% to 97.5%, and specificity of 94%.[11]

Research Organization (CSIRO) developed this AI platform and is about to deploy it at 20 general practitioners' (GP) offices in Western Australia. The software was built from an IBM database of 88,000 retinal images in the EyePACS library. At-risk patients "would usually be referred to a specialist for screening, waiting six weeks or more—now it can potentially be done in a single 30-minute visit to a GP," according to CSIRO.[16] If successful, the app could help reduce the $14 billion annual impact of diabetes on the Australian economy, CSIRO says.



As AI technology for DR progresses, researchers hope it will one day provide screening advanced enough to properly identify any and all disease stages, even borderline cases, such as mild/moderate non-proliferative DR, as seen here.

• ***Optos/Verily partnership.*** In January 2017, another Google company, Verily Life Science, announced a partnership with Nikon and its subsidiary Optos to create new DR screening protocols.[17] No other details have been released, but the effort is expected to combine Verily's machine learning technology with Optos' ultra-widefield imaging already popular in today's practices.

The Future in Focus

The team at DeepMind note several ways AI might reduce the worldwide loss of vision from diabetic retinopathy:[18] Screening tests, either self-administered with a mobile device or used in telemedicine networks with medical professionals, can extend access to care for underserved populations. Subclinical disease may be identified early enough to allow initiation of treatment and lifestyle modifications to forestall damage in ways never before possible. And since these new software platforms are learning engines, it's reasonable to expect continued refinement of accuracy and capability.

As artificial intelligence and other technologies evolve, optometry will have opportunities to bring patients into the healthcare system earlier than before and make meaningful differences in their lives. With these innovative technologies, optometrists may one day be able to improve patient outcomes, positively impact optometric education and create new models of inter-professional care. For us to continue to positively impact the many diverse communities that optometry serves, the future starts now. ■

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Dr. Poirier is an assistant clinical professor in the Advanced Eye Care Service at the SUNY College of Optometry.

Dr. Gould is the chief of the advanced Care Service at the SUNY College of Optometry.

1. DesMarais C. Elon Musk is right, artificial intelligence is growing like crazy. November 29, 2017. www.inc.com/christina-desmarais/elon-musk-is-right-artificial-intelligence-is-growing-like-crazy.html. Accessed January 25, 2018.
2. Research at Google: Healthcare. https://research.google.com/teams/brain/healthcare. Accessed January 25, 2018.
3. Pellegrino ED, Thomasma DT. For the good of the patient: The restitution of beneficence in medical ethics. New York: Oxford University Press; 1988.
4. American Diabetes Association. Economic costs of diabetes in the U.S. in 2012. Diabetes Care. 2013;36:1033-46.
5. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013:36(suppl 1):S64-71.
6. Menke A, Casagrande S, Geiss L, et al. Prevalence of and trends in diabetes among adults in the United States, 1988-2012. JAMA. 2015;314(10):1021-9.
7. American Optometric Association. American Optometric Association's annual survey reveals misconceptions about diagnosing diabetes and its related eye diseases. October 27, 2016. www.aoa.org/newsroom/aoa-annual-survey-reveals-misconceptions-about-diabetes. Accessed January 25, 2018.
8. Kernt M, Cheuteu R, Vounotrypidis E, et al. Focal and pan-retinal photocoagulation with a navigated laser (NAVILAS). Acta Ophthalmologica. 2010;89(8):e662-4.
9. Association for Research in Vision and Ophthalmology. First use of surgical robot inside the human eye. May 1, 2017. www.newswise.com/articles/view/673836/?sc=sphr&xy=10020710. Accessed January 25, 2018.
10. Kubota T. Stanford computer scientists develop an algorithm that diagnoses heart arrhythmias with cardiologist-level accuracy. July 6, 2017. http://news.stanford.edu/2017/07/06/algorithm-diagnoses-heart-arrhythmias-cardiologist-level-accuracy. Accessed January 25, 2018.
11. Gulshan V, Peng L, Coram M, et al. Development and validation of a deep learning algorithm for detection of diabetic retinopathy in retinal fundus photographs. JAMA. 2016;316(22):2402-10.
12. Abramoff MD, Lou Y, Erginay A, et al. Improved automated detection of diabetic retinopathy on a publicly available dataset through the integration of deep learning. IOVS. 2016;57(10):5200-6.
13. De Fauw J, Keane P, Tomasev N, et al. Automated analysis of retinal imaging using machine learning techniques for computer vision [version 2; referees: 2 approved]. F1000Research. 2017;5:1573.
14. Gargeya R, Leng T. Automated identification of diabetic retinopathy using deep learning. Ophthalmology. 2017;124:962-9.
15. Bleicher A. Teenage whiz kid invents an AI system to diagnose her grandfather's eye disease. IEEE Spectrum. August 3, 2017. https://spectrum.ieee.org/the-human-os/biomedical/diagnostics/teenage-whiz-kid-invents-an-ai-system-to-diagnose-her-grandfathers-eye-disease. Accessed January 25, 2018.
16. Commonwealth Scientific and Industrial Research Organization. AI technology to help prevent blindness. September 17, 2017. www.csiro.au/en/News/News-releases/2017/AI-technology-to-help-prevent-blindness. Accessed January 25, 2018.
17. Nikon, Verily partner to diagnose diabetic retinopathy sooner. January 10, 2017. www.healio.com/optometry/business-of-optometry/news/online/%7B682c487d-edfd-4373-ac85-29064e0b129a%7D/nikon-verily-partner-to-diagnose-diabetic-retinopathy-sooner. Accessed January 25, 2018.
18. DeepMind. Researching for tomorrow: DeepMind health and research collaborations. https://deepmind.com/applied/deepmind-health/working-nhs/health-research-tomorrow. Accessed January 25, 2018.

Myopia Management in Action

These clinical pearls can help you establish a successful subspecialty within your practice. By Daniel J. Press, OD, and S. Barry Eiden, OD

Myopia is likely the most common condition encountered by optometrists in clinical practice, and the prevalence is only increasing.[1] To make matters worse, myopia is also a well-established risk factor for serious eye diseases such as retinal detachment, glaucoma and maculopathy.[2] Clinicians must prepare their practices to care for each and every patient.[3-5]

A myopia management subspecialty within your practice is an opportunity to better educate your patients and families about the implications of progressive myopia, screen for at-risk patients and select the most appropriate treatments.

Here is a primer on how to put in place the systems required to successfully integrate myopia management into your practice.

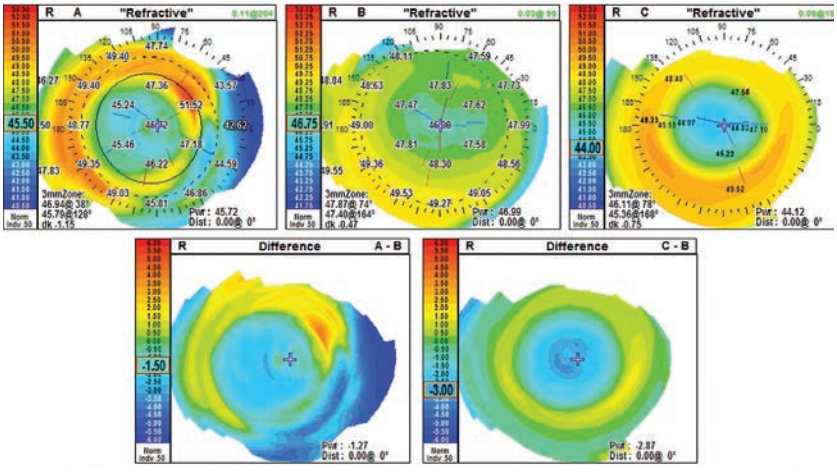

Fig 1. The refractive difference map shows the similar corneal power profile for multifocal soft lenses and corneal reshaping. In map A, the patient is wearing NaturalVue multifocal 1-day -3.00. Map B is the naked cornea, and map C is the same patient after a night of corneal reshaping treatment.

Mind Shift

Clinicians ready to dive into myopia management as a subspecialty should start with the right mindset: focus on the condition you are treating, not the treatments you offer.

For example, in our office, patients no longer make an appointment for a "corneal reshaping evaluation." Instead, our staff schedules a "myopia management evaluation." Likewise, we establish a comprehensive myopia management baseline before making treatment recommendations.

The more we learn about myopia and its implications, the more motivated we are to slow its progression. But when you have a child diagnosed with or at a high risk for myopia sitting in your exam chair, how do you proceed?

The answer depends on a number of factors—the most critical of which is how you frame the discussion. Our first recommendation is to stop referring to myopia as

Table 1. Binocular and Accommodative Testing Performed in Myopia Management Assessment and Periodic After Care

1. Amplitude of accommodation
2. Binocular accommodative facility
3. Monocular accommodative facility
4. Negative relative accommodation
5. Positive relative accommodation
6. Lag of accommodation
7. Stereopsis
8. Cover test at distance and near
9. Near point of convergence
10. Fusional vergence ranges at near
11. Vergence facility at near

"nearsightedness." This describes the subjective experience of blurred distance vision relative to near vision. Myopia is a condition that results in light being focused anterior to the macula, often due to excessive axial length.

Think of it this way: if a patient had LASIK for 7D of myopia and their post-surgical refraction was plano, are they still myopic? They may no longer be nearsighted, but when it comes to risk factors associated with axial length, they are still myopic. Society's lexicon equating nearsightedness with myopia has led to a ho-hum attitude that only communicates blurred vision. In the exam room, we try to stick to the medical term *myopia*.

Myopia is more than an inconvenience of sight, and often patients and caregivers are more motivated to intervene when we take the time to explain myopia more completely. For one, an increase is most commonly due to an increase in axial length, which is the most likely cause of the increased risk of eye disease with higher myopia. Research estimates that if we successfully reduce progression by 33%, 73% of those children will be below 5.00D—a threshold associated with an increased risk of choroidal neovascularization, retinal detachment, glaucoma and cataract.[6] Further reducing progression by 50% would mean 90% of those patients will remain below 5.00D.[6]

In our office, myopia education doesn't start with the first "minus" refractive measurements. If a child has a high risk of myopia, including a positive family history and a documented reduction of hyperopia, then the conversation is started.

Clinical First Steps

Clinicians can screen at-risk patients using a simple, single measure of refractive error. A child at six years of age with +0.75 or less of hyperopia has a significantly higher risk of developing myopia, and clinicians should recommend more frequent monitoring.[7] Common recommendations for at-risk cases include increasing time outdoors and general visual hygiene such as monitoring working distance and taking breaks from accommodating at near for extended periods of time.[8,9]

Once a child is diagnosed with myopia and both the patient and parents understand myopia as a medical concern, our clinicians schedule a comprehensive myopia baseline evaluation. This includes measurements and procedures, beyond a routine eye exam, that reveal visual findings potentially contributing to myopia development and amenable to interventions (*Table 1*). The specialty evaluation

Table 2. Advanced Technology Used in Myopia Management

1. Specular microscopy
2. Infrared pupillometry
3. Scheimpflug corneal tomography
4. Placido corneal topography
5. Axial length measurement
6. Aberrometry

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BHVI Resources

By Monica Jong, PhD, BOptom

Clinicians can access many helpful myopia management tools provided by the Brien Holden Vision Institute (BHVI), including:

Managing Myopia (North America). This COPE-approved course aims to help practitioners deliver the broadest and most effective management options and offers an opportunity to engage with leaders in the field during a live webinar. The course will be available March 21 through April 11, 2018, and can be found at https://academy.brienholdenvision.org/browse/usa/courses/myopia-us-2018.

An evidence-based myopia calculator. This can help practitioners demonstrate the impact of various myopia management strategies on the predicted level of myopia up to the age of 17. Practitioners can access the calculator at https://calculator.brienholdenvision.org.

The International Myopia Institute. This global group of experts is tasked with generating a consensus on key areas of myopia that can help guide practitioners towards an evidence-based approach to myopia management. Membership is free and open to all. The white papers will be freely accessible in late 2018. For more information, visit www.myopiainstitute.org.

Dr. Jong is a senior research fellow at the Brien Holden Vision Institute.

guides the informed decision on which therapy is most appropriate and provides important data for comparison over time.

Binocular vision and accommodation are important to monitor in school-age children, as deficits can lead to difficulties with academic behaviors.[10] The myopia management programs offered in our practice have the potential to impact binocular vision and accommodation, which necessitates a baseline that includes those measurements.

Practice pearl. This diagnostic battery is considered advanced testing, and the doctor should be compensated appropriately. In our practice, patients are financially responsible for the myopia management evaluation as a non-covered service billed outside of any managed care insurance program.

The Evaluation

The ocular health of our patient is our number one concern, and detailed corneal analysis is invaluable, especially for considering for corneal reshaping and multifocal contact lens treatments. Clinicians should want the most comprehensive evaluation of ocular and visual findings that may impact myopia management now and into the future. That means clinicians should test beyond the standard evaluation of the cornea under the biomicroscope with fluorescein staining (*Table 2*).

In our practice, we gather information on endothelial cell function via specular microscopy and elevation data with corneal tomography, topography or both. Documenting pupil size in dim and bright lighting conditions is particularly important for treating with low-dose atropine. We perform infrared pupillometry, although less precise pupil size measurements are also useful.

Wavefront aberrometry, included in our baseline myopia management evaluation, documents any corneal higher-order aberrations (HOAs). Recent research suggests a correlation between HOAs and myopia progression, with larger HOAs associated with less myopia progression and smaller axial elongation.[11] Comprehensive myopia management should include periodic axial length monitoring. If you are going to develop a myopia management subspecialty, axial length measurement is crucial and will likely be considered standard of care one day.

Practice pearl. In our practice, an ophthalmic technician gathers many of the measurements while the doctor discusses the pros, cons and efficacy of each treatment option to the parent. Common questions that come up include: *How long will my child be treated? Is technology influencing myopia? What would you do?*

A practitioner developing a myopia management practice should be prepared to address these questions. Regarding the last question, we have found that the following answer is effective: "I treat every child that I see as if they were my own. I would not recommend anything for your child that I would not do for my own children." Of course, clinicians must truly believe it before using that statement.

Another important consideration is to include the child in the conversation once they are back in the exam room. It makes little difference if you and the parent are on the same page if the child isn't. Ensuring the child understands the discussion will help improve compliance rates.

Choosing the Right Therapy

Research suggests slowing myopia progression is accomplished via two mechanisms of action: optical defocus and biochemical influence. Our practice has three evidence-based myopia management programs, two based on optical defocus and one on biochemical influence: corneal reshaping, soft multifocal contact lenses and low-dose atropine.

Other options may help to reduce myopic progression, but the evidence is not as compelling as it is for these three strategies. Although at times we still use other methods of myopia intervention, the formalized programs are limited to these three. Of course, all of these options are technically off-label uses, as none of them are FDA approved specifically for myopia control.

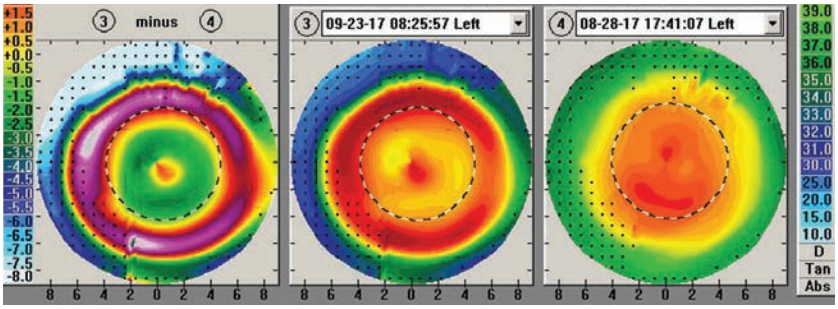

Fig. 2. Measurements taken during a corneal reshaping patient's first morning show a central island, indicated by the small zone of central corneal steepening.

While the three options are presented, the majority of our patients still choose corneal reshaping as the primary treatment option. At our practice, approximately 80% of new myopia patients are fit in corneal reshaping lenses, 15% are fit in multifocal contact lenses and 5% are prescribed low-dose atropine. Corneal reshaping is our primary treatment option because our practice is involved in clinical research, and our doctors have a high comfort level with its safety and efficacy.[12]

Given the two mechanisms of action at play, it would seem logical that the combination would lead to an increased treatment effect. While our clinical experience suggests this is true, research has yet to provide evidence-based support for this modality. Thus, we reserve combination treatments for children with significant myopia progression.

Marketing Your Programs

Our practice has established a packet of information for each of our myopia management programs. The packet is organized in a branded, double-sided folder with information for the patient on one side and the treatment program contract on the other. The contract includes:

- Introduction to the treatment modality, including its off-label use
- Program fees
- Definition of what's included in the fees
- Potential additional fees
- Ongoing care fees after the first year
- Refund and credit policies
- Consent to treat document
- Research reference list

Our program fees are all-inclusive for the first year of treatment, and we do not separate professional services from materials. The exception is the low-dose atropine program, where the fee for the medication is handled by the patient and the compounding pharmacy and is not included in our program fee.

The fees for our programs are tiered and are based on several factors beyond a traditional refractive cutoff, including the estimated time investment for each patient, degree of myopia, rate of progression, patient age, complexity of the fit (for corneal reshaping and multifocal contact lenses), anticipated material costs and personality of the patient and parent. The more time we anticipate spending and the more challenging the case, the higher the quoted tier.

When patients return for their second year in the program, they are responsible for another fee that covers a second comprehensive myopia evaluation. They can also enroll in an aftercare program the same day as their annual comprehensive eye examination. The aftercare program covers office visits related to myopia management for the year. If they do not enroll in the program, they must pay individually for each myopia management visit the following year.

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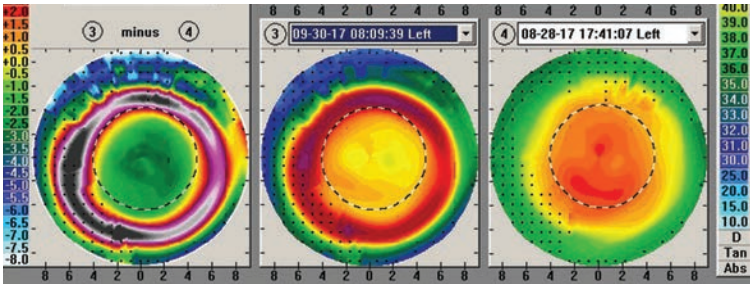

Fig. 3. The same patient's one week follow up shows complete resolution of the central island—without any changes to the prescribed lens.

Corneal reshaping. Research suggests this treatment option slows myopia progression, on average, by 45% with the added lifestyle benefit of not needing optical compensation during the day.[13] However, proceed with caution with children who are outside of the FDA-approved parameters. For reference, the Paragon CRT system is FDA-approved for up to -6.00D of myopia and -1.75D of astigmatism. In our practice, if a patient is beyond that refraction, they are not candidates for the corneal reshaping program. Despite the excellent safety profile of corneal reshaping lenses, some parents are not comfortable with the concept of overnight contact lens wear.[14] These patients may do better with one of the other treatment programs.

Multifocal contact lenses. This treatment option slows progression of myopia by approximately 50% and is a good option for patients who are averse to or have dropped out of corneal reshaping treatment or who are outside of the FDA-approved range for corneal reshaping.[15,16] When fitting children in soft contact lenses, we are most comfortable with the single-use lens modality. Center-distance designs are our first choice, considering the optical effect is closest to mimicking the optics of a post corneal reshaping cornea (*Figure 1*).

Currently, only one single-use lens on the market in the United States has a center-distance design, the NaturalVue Multifocal 1-Day (Visioneering Technologies). It has a high plus effect that creates myopic defocus in the peripheral retina. Research shows myopic defocus in the peripheral retina can be a powerful stimulus to slow myopic progression and is the leading theory on the effectiveness of corneal reshaping and multifocal contact lenses.[17]

Low-dose atropine. This treatment program can reduce progression by approximately 50%. However, studies show it slows myopia progression after two years of treatment, one year off treatment, then two years on or off treatment, depending on progression—making generalization challenging.[18,19]

Regardless, treatment effectiveness is not the only factor under consideration when making myopia management recommendations. Patient and parent philosophies are also taken into account, and patients and their parents may prefer to avoid long-term medication use. While atropine has been a popular myopia control method for years, optometrists are often hesitant to recommend it due to concerns about side effects.[19] The current literature, with the longest follow up of five years, reports no long-term side effects on accommodation or retinal function associated with the use of atropine.[20,21] Clinically, we have yet to see a case with any significant complication due to therapy with low-dose atropine. A small percentage of cases experience mydriasis and reduced accommodative amplitude, both of which are addressed by either a further reduction in the atropine concentration or discontinuation of atropine therapy. Low-dose atropine may be a useful option for cases that are outside of the FDA-approved parameters for corneal reshaping, for those who are not appropriate multifocal contact lens candidates and in combination therapy. A thorough discussion of the benefits and risks of low-dose atropine can help patients and parents develop a comfort level with this option. In all studies to date, an inverse relationship of atropine concentration and myopia progression exists, where higher concentrations result in less myopia progression. Prior to the ATOM 2 study, the philosophy was to prescribe the highest dosage that did not result in symptomatic side effects. However, since the ATOM 2 study, we start at 0.01% atropine and consider increasing the concentration if there is evidence of inadequate myopia control.[20]

Ongoing Care

The follow-up schedule is similar across all modalities with the exception of corneal reshaping patients, who require a first morning follow up. The programs include visit intervals at one week, one month, three months and then every six months. More frequent visits are included in the cost of the program. Clinicians should evaluate eye health and side effects at every office visit and repeat refractive and axial length measurements at six-month intervals.

A few clinical pearls on corneal reshaping are important to share. For one, clinicians should not rush to modify the fit of a corneal reshaping lens after the first morning. Often, small concerns will disappear at the one-week visit without any

modifications (*Figures 2 and 3*). In addition, clinicians should avoid taking visual acuities monocularly in free space. While perhaps an unusual clinical pearl, you will encounter parents who are not satisfied with one eye that is 20/20 while the other eye is 20/20-1. Taking binocular visual acuities in free space can eliminate any angst associated with slight variations. You can record monocular acuities, but with the patient behind the phoropter with no lenses before commencing refraction.

Measuring Success

Since the area of myopia management is still in its relative infancy, clinicians will have to look to the literature to help define acceptable myopia control as more information is published. In our practice, we refer to experts in the field who reference control groups with average axial length elongations of 0.22mm to 0.24mm. Thus, we use greater than 0.23mm of axial elongation in one year as our benchmark for inadequate control for patients enrolled in one of our management programs. In those cases, we consider combined myopia management options. Unfortunately, there are times when, despite our best efforts, we are not achieving what we consider to be acceptable myopia control.

Adding formal myopia management programs has given us the opportunity to provide our patients with another area of specialized care that they have come to expect from our practice. A mother with three children undergoing myopia management in our practice recently shared, "I cannot adequately express in words what these treatment options mean for our family. Thank you." With the right tools, you too can make a lasting impact on your myopic patients and their families. ■

Dr. Press is the director of Pediatric Eye Care, Binocular Vision and Vision Therapy Services at North Suburban Vision Consultants in Illinois.

Dr. Eiden is the president and medical director of North Suburban Vision Consultants and the president and cofounder of the International Keratoconus Academy.

1. Holden BA, Fricke TR, Wilson DA, et al. Global prevalence of myopia and high myopia and temporal trends from 2000 through 2050. Ophthalmology. 2016;123(5):1036-42.
2. Saw SM, Gazzard G, Shih-Yen EC, Chua WH. Myopia and associated pathological complications. Ophthalmic Physiol Opt. 2005;25(5):381-91.
3. Holden BA, Wilson DA, Jong M,et al. Myopia: a growing global problem with sight-threatening complications. Community Eye Health. 2015;28(90):35.
4. Holden B, Sankaridurg P, Smith E, et al. Myopia, an underrated global challenge to vision: where the current data takes us on myopia control. Eye (Lond). 2014;28(2):142-46.
5. Flitcroft DI. The complex interactions of retinal, optical and environmental factors in myopia aetiology. Prog Retin Eye Res. 2012;31:622-60.
6. Brennan NA. Predicted reduction in high myopia for various degrees of myopia control. Cont Lens Anterior Eye. 2012 Dec;35(Suppl):e14-e15.
7. Zadnik K, Sinnott LT, Cotter SA, et al. Prediction of juvenile-onset myopia. JAMA Ophthalmol. 2015;133(6):683-9.
8. Xiong S, Sankaridurg P, Naduvilath T, et al. Time spent in outdoor activities in relation to myopia prevention and control: a meta-analysis and systematic review. Acta Ophthalmol. 2017;95(6):551-66.
9. Saw SM, Chua WH, Hong CY, et al. Nearwork in early-onset myopia. Invest Ophthalmol Vis Sci. 2002;43(2):332-9.
10. Borsting E, Mitchell GL, Kulp MT, et al. Improvement in academic behaviors after successful treatment of convergence insufficiency. Optom Vis Sci. 2012;89(1):12-18.
11. Hiraoka T, Kotsuka J, Kakita T, et al. Relationship between higher-order wavefront aberrations and natural progression of myopia in schoolchildren. Sci Rep. 2017;7:7876.
12. Davis RL, Eiden SB, Bennett E, et al. Stabilizing Myopia by Accelerating Reshaping Technique (SMART)-Study three year outcomes and overview. Adv Ophthalmol Vis Syst. 2015;2(3):00046.
13. Sun Y, Xu F, Zhang T, et al. Orthokeratology to control myopia progression: a meta-analysis. PLoS One. 2015;10(4):e0124535.
14. Liu Y, Xie P. The safety of orthokeratology-a systematic review. Eye Contact Lens. 2016;42(1):35-42.
15. Li SM, Kang MT, Wu SS, et al. Studies using concentric ring bifocal and peripheral add multifocal contact lenses to slow myopia progression in school-aged children: a meta-analysis. Ophthalmic Physiol Opt. 2017;37(1):51-9.
16. Cooper Vision. Three-year study indicates pioneering contact lens therapy effective in slowing myopia progression in children by 59%. Press Release. June 10, 2017. https://coopervision.com/our-company/news-center/press-release/three-year-study-indicates-pioneering-contact-lens-therapy. Accessed January 8, 2018.
17. Smith EL 3rd, Hung LF, Arumugam B. Visual regulation of refractive development: insights from animal studies. Eye (Lond). 2014;28(2):180-8.
18. Chia A, Lu QS, Tan D. Five-year clinical trial on atropine for the treatment of myopia 2: myopia control with atropine 0.01% eyedrops. Ophthalmology. 2016;123(2):391-9.
19. Cooper J, Schulman E, Jamal N. Current status on the development and treatment of myopia. Optometry. 2012;83(5):179-99.
20. Chia A, Chua WH, Cheung YB, et al. Atropine for the treatment of childhood myopia: safety and efficacy of 0.5%, 0.1%, and 0.01% doses (atropine for the treatment of myopia 2). Ophthalmology. 2012;119(2):347-54.
21. Luu CD, Lau AM, Koh AH, Tan D. Multifocal electroretinogram in children on atropine treatment for myopia. Br J Ophthalmol. 2005;89(2):151-3.

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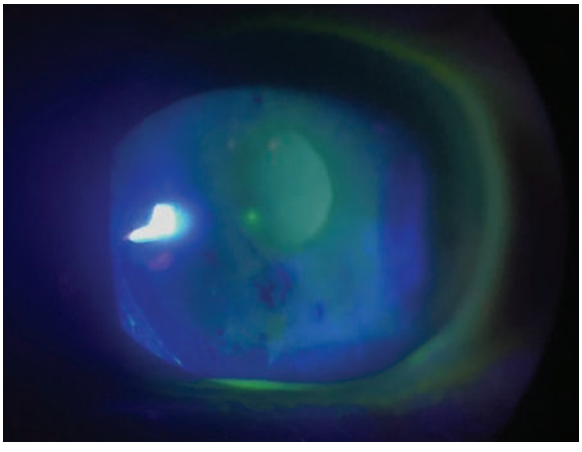
THYROID DISEASE:
A DELICATE BALANCE DISRUPTED

This diagnosis isn't as cut-and-dry as you think; be prepared to manage both hyper- and hypothyroid patients. **By Matt Dixon, OD**

The thyroid gland is critically important for our overall health and function—for one, hormones produced by the thyroid gland play a major role in energy production and metabolism at the cellular level. Every tissue and organ requires optimal thyroid hormone (TH) levels to work properly, especially the heart and brain.[1]

While optometrists are prepared to recognize Graves' disease, a major cause of hyperthyroidism, thyroid dysfunction can manifest in other ways as well—all of which can negatively impact patients. A deeper understanding of the thyroid gland and its many dysfunctions is essential for not only a more holistic view of our patients, but also improved ocular health and quality of life.



Dry eye is the most common ocular side effect of both hyper- and hypothyroidism.

The American Thyroid Association (ATA) suggests thyroid disorders affect at least 20 million Americans, and 60% of patients with the disorder are unaware.[2] Moreover, more than 12% of Americans will develop a thyroid condition during their lifetime.[2] These numbers emphasize the inevitability of one of these patients seeking care in your office. Even once patients are diagnosed, their symptoms often linger. One study found that, among patients taking thyroid medication, only 60% were within the normal hormone range.[3] These researchers concluded that "thyroid dysfunction is common,

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Goal Statement: Thyroid dysfunction can manifest in many ways—all of which can negatively impact patients. A deeper understanding of the thyroid gland and its many dysfunctions, particularly hypothyroidism, is essential for maintaining patients' ocular health and quality of life. This article will help ODs better understand the various thyroid conditions and their ocular effects.

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may often go undetected and may be associated with adverse health outcomes that can be avoided by serum thyroid stimulating hormone (TSH) measurement."[3]

This article will help you better understand both hyper- and hypothyroidism and the ocular effects, as well as how to detect, treat and monitor these patients.

The Ins and Outs of the Thyroid

An elaborate feedback loop involving the hypothalamus and pituitary glands controls the output of the thyroid, making up the hypothalamic–pituitary–thyroid (HPT) axis. When the body requires energy to perform a specific function, requiring TH, the hypothalamus releases thyrotropin-releasing hormone (TRH), which initiates the production of TSH in the pituitary gland and signals the production of thyroxine (T_4). Serum T_4 is monitored by the HPT axis, which, in normal individuals, will generate the correct amount of TH.

T_4 is known as a "storage hormone" because it is inactive and not a direct source of energy. T_4 is converted into triiodothyronine (T_3) as the necessary source of energy for the body.[1] The normal thyroid gland will generate 90% T_4 and only about 10% T_3. Most of the conversion to T_3 occurs in other tissues in the body such as the brain, kidney and liver.[1]

Nutrients such as iodine and selenium play a key role in this conversion. The body requires about 60mcg of iodine daily for adequate production of TH, and the average intake in the United States is about 1,000mcg.[4] However, about a third of the world's population live in iodine-deficient areas and are at an increased risk of iodine deficiency disorders such as goiter, hypothyroidism, mental disability, increased perinatal mortality and retarded physical development.[5] Selenium is also crucial in thyroid health, as it acts as one of the catalysts for the conversion of T_4 to T_3 and then helps to control thyroid hormone metabolism.[6]

Hyper vs. Hypo

The thyroid can malfunction in two ways: by creating too much TH, hyperthyroidism, or by creating too little, hypothyroidism (HT). Each of these conditions can cause significant systemic and ocular effects, and it's critical to know which one is at play when caring for these patients.

Hyperthyroidism. Between 1% and 3% of Americans are diagnosed with hyperthyroidism, with Graves' disease as the number one cause in the United States, where iodine is sufficient.[7] The second leading cause is toxic multinodular goiter, in which the nodules become a source of TH independently and do not respond to TSH signaling.[7]

Graves' disease is characterized by the presence of hyperthyroidism, goiter and, sometimes, ophthalmopathy and dermopathy. The

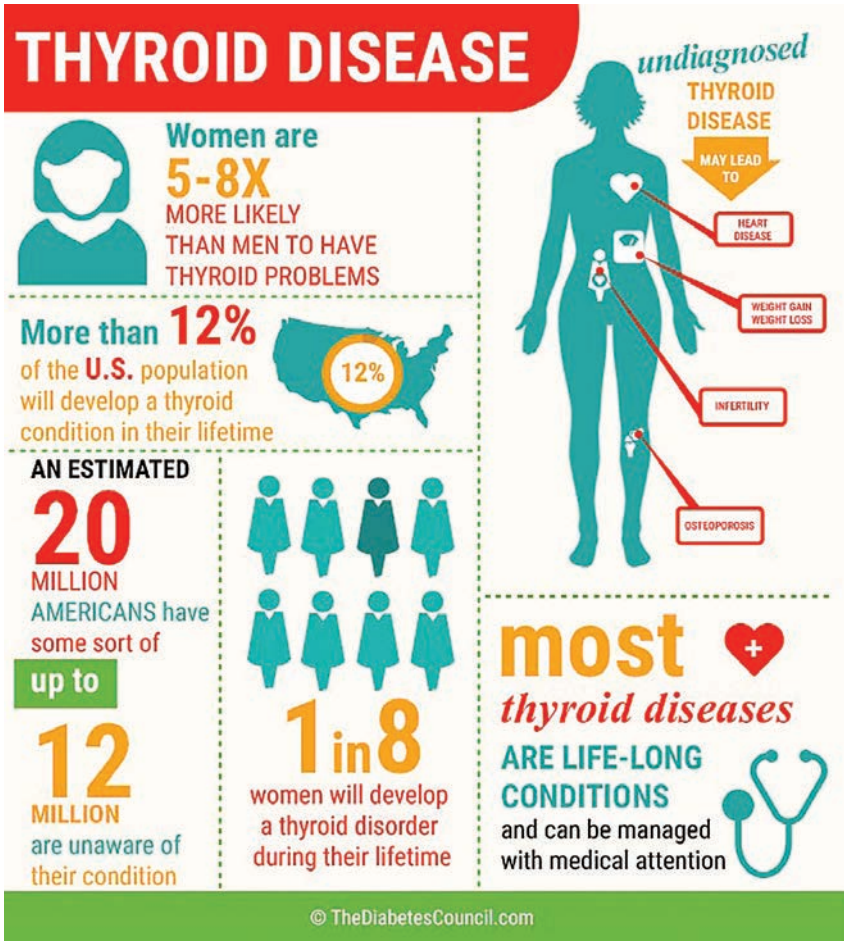

Image: The Diabetes Council

In-Office Numbers

In my own primary care practice, we frequently encounter as many as six patients a day who are taking medication for hypothyroidism. While I rarely see patients with hyperthyroidism or Graves' disease, I have more than 600 patients who are taking medication for HT. About 550 are being treated with Synthroid (levothyroxine, AbbVie) and 50 are treated with natural desiccated thyroid. Similarly, a local nursing home pharmacy supplier shared that it cares for 7,215 patients, of which 1,600 (22%) take thyroid medication, and only 11 take a medication other than Synthroid.

A Complete Thyroid Function Test

Test	Significance	Normal Adult Ranges*
TSH	This is released by the pituitary gland and controls the production of thyroid hormone.	0.5mIU/mL to 3.0mIU/mL (differs in pregnancy)
Total T_4	This is a pro-hormone, which is converted into T_3 by 5'-iodinase (rarely measured).	5µg/dL to 10.8µg/dL
Total T_3	Checks levels of triiodothyronine, the active thyroid hormone (rarely measured).	75ng/dL to 200ng/dL
Free T_4	Only a small fraction of the circulating hormone is free (unbound) and biologically available, hence measuring concentrations of free thyroid hormones is of greater diagnostic value.	0.7ng/dL to 2.5ng/dL
Free T_3	The most active thyroid hormone.	2.5pg/mL to 6.5pg/mL
Reverse T_3	This is the biologically inactive form of T_3. Conversion from T_3 to rT_3 can be protective in periods of illness or to protect from elevated T_3 in hyperthyroidism. In hypothyroidism, high rT_3 indicates problems such as too much T_4 and not enough T_3.	9ng/dL to 35ng/dL (Genova Diagnostics)
Thyroglobulin	The storage form of TH, this is primarily used as a tumor marker in thyroid cancer.	Athyrotic: <0.1ng/mL Intact thyroid: ≤33ng/mL (Mayo Clinic)
TPO and TGAb	Thyroid peroxidase antibody (anti-TPO) is the most common test for autoimmune thyroid disease; it can be detected in Graves' disease or Hashimoto's thyroiditis. Thyroglobulin antibody (anti-TGAb) targets thyroglobulin.	TPO: 0.0IU/mL to 150IU/mL TGAb: <4.0IU/mL
TRAb, TSHRAb, TSI	Thyroid receptor antibody, TSH receptor antibody and thyroid stimulating immunoglobulin are helpful in suspected Graves' disease.	

*Observed dried blood spot ranges based on collected laboratory data. Adapted from ZRT Laboratory. www.zrtlab.com/images/documents/Essential%20Thyroid%20Sample%20Report%202015.pdf. Accessed December 15, 2017

cause of Graves' disease is thought to be multifactorial, but researchers believe it arises, in part, from the loss of immunotolerance and the development of auto-antibodies that stimulate thyroid follicular cells by binding to the TSH receptor.[8] These antibodies upregulate the receptor by mimicking TSH, causing excessive TH production.

Symptoms of hyperthyroidism include heart palpitations, fatigue, tremors, anxiety, disturbed sleep, weight loss, heat intolerance, sweating and polydipsia.

Hyperthyroidism can be confirmed by low serum TSH, elevated T_4 and T_3 or both. Ultrasound and radioactive iodine uptake may also aid in diagnosis.

Hypothyroidism. Clinical experience suggests underactive thyroid is the more common of the two thyroid dysfunctions; however, data regarding prevalence is lacking and varies greatly between countries. A landmark study based in the United Kingdom found a prevalence of around 9.5%.[9] The Colorado Thyroid Disease Prevalence Study also found that 9.5% of individuals had a TSH above 5.1mIU/L, while also acknowledging the diagnostic challenges of establishing accurate numbers in the United States.[3]

Research does show that women are five times more likely than men to have HT, and women with HT are twice as likely to have a heart attack than women without the disease. In addition, 6% of miscarriages are associated with HT, and autism and low IQ in pediatric patients are linked to HT during pregnancy.[10,11]

Hashimoto's thyroiditis is the most common cause of HT, followed by thyroidectomy, amiodarone-induced hypothyroidism and postpartum thyroiditis. As much as 95% of patients with HT have Hashimoto's disease.[12]

In most cases of Hashimoto's thyroiditis, blood tests will reveal one or two types of anti-thyroid antibodies: thyroid peroxidase antibody (in up to 95% of those with Hashimoto's) and antibodies against thyroglobulin (around 80%). Because these antibodies may appear decades before a change in TSH, screening is always crucial in suspected thyroid disease.[12]

Symptoms of HT are numerous and include feeling cold, depression, fatigue, anxiety, hair loss, dry skin and eyes, thinning eyebrows temporally, brain fog and impaired memory, high cholesterol, slow pulse, irregular menstrual cycles, insomnia, edema and constipation. Practitioners can uncover most of these concerns with a symptom questionnaire (from the Institute for Functional Medicine, for example) for every confirmed or suspected HT patient.[13] Anecdotally, most patients taking medication for HT will confirm as many as 10 residual symptoms that can be linked to underactive TH, and they frequently list dry, gritty eyes as

one of the symptoms.

This disease is also known to exacerbate adverse effects of heart disease, hypertension, elevated cholesterol, cognitive deficits, autism, infertility, fibroids and neuromuscular dysfunction. One study found that, as thyroid function decreased, LDL cholesterol went up. Of patients taking thyroid medication, 40% still had abnormal TSH values—suggesting a need for better monitoring and disease control.[3]

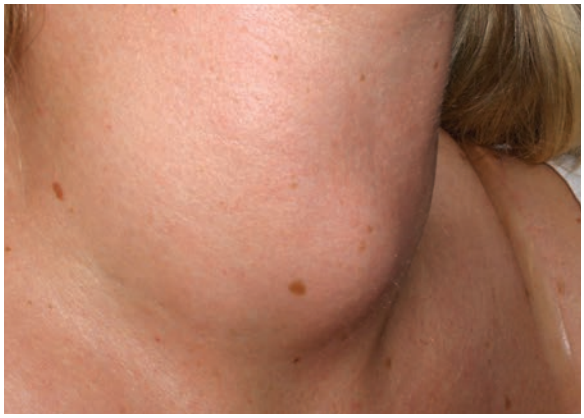

Image: Drahreg01, wikimedia commons

Patients with Graves' disease may present with hyperthyroidism, goiter (seen here) and, sometimes, ophthalmopathy and dermopathy.

Despite these findings, the American Academy of Family Physicians has concluded that routine HT screenings are not helpful.[14]

The ATA and the American Association of Clinical Endocrinology (AACE) consider TSH to be the best marker for diagnosing and treating HT. However, no consensus exists on normal TSH reference ranges. More than a decade ago, the National Academy of Clinical Biochemistry recommended an upper limit of 2.5mIU/L because 95% of normal individuals fall under this value.[15] They recognized compounding factors such as age, gender and pregnancy, as well as a diurnal variation of as much as 50%, with levels being highest during sleep.[15]

However, the 2014 ATA guidelines define subclinical (mild) HT as elevated TSH greater than 10mIU/L and a normal free T_4, while overt (severe) HT is characterized by an elevated TSH greater than 10mIU/L and abnormal free T_4.[16] Although it makes sense that hypothyroidism is acquired slowly over time, treatment is reserved until TSH reaches a level the individual clinician considers abnormal. For patients with signs and symptoms of HT, close monitoring of labs (including antibodies that indicate autoimmune involvement) may help doctors detect this disease before serious consequences occur.

While some doctors may not treat the disease until TSH is above 10mIU/L, this upper limit has been falling in recent years, and many labs now set it at 4.5mIU/L. Regardless of the number, making treatment decisions based only on the preferred lab's normative range for TSH, ignoring the entire clinical picture and especially symptoms, often leads to underdiagnosis.

ODs should inquire about the patient's last TSH level, as they would for HbA1c in diabetes patients, but remember it should be viewed on an individual basis in light of symptoms and other factors.

Ocular Involvement

Depending on whether a patient has too much or too little TH in their body, they can present with any number of ocular side effects, often beginning with dry eyes.

Hyperthyroidism. Graves' disease, an autoimmune state, may lead to thyroid-associated orbitopathy (TAO), formerly known as thyroid eye disease, in 25% of patients. Approximately 80% of TAO cases are associated with hyperthyroidism, although the onset may not coincide with the onset of the hyperthyroid state. Euthyroid TAO refers to the 5% to 10% of Graves' disease patients who do not develop hyperthyroidism. The final 10% of TAO patients have primary autoimmune *hypo*thyroidism.[17]

Typically, the first sign of TAO is ocular surface disease (OSD), although patients may also have eyelid retraction/proptosis, lagophthalmos, periorbital edema and diplopia. TAO may appear prior to diagnosis of a thyroid condition, so ordering a thyroid panel and comanaging with an endocrinologist is necessary. Still, orbital involvement progresses independently of thyroid disease and at various phases. In most cases, TAO is self-limiting.

The most serious risk of TAO is optic neuropathy with vision loss. While optometrists should seek comanagement with a specialist, they should also provide supportive measures to reduce edema, limit photophobia and protect the ocular surface. Management also includes CT or MRI to rule out optic neuropathy from compression related to

Masquerading Disease

Patients with Graves' disease have thyroid receptor antibodies (TRAb), which can cause some clinical confusion. These antibodies provide constant stimulation to the TSH receptor and result in a TSH close to zero. Radioactive iodine (RAI) does not eliminate the antibodies and may even increase them. Therefore, after RAI you may still have TRAb antibodies, even though you now have little thyroid gland function. This mechanism of action results in a TSH that is close to zero, leading a clinician to think the patient is hyperthyroid, when they are in fact severely hypothyroid. Because free T_3 and free T_4 will be extremely low in this situation, their measurements are the only way to properly determine thyroid levels, not TSH.

Graves' Disease Patients have TSH receptor antibodies (TRab). Tiredthyroid.com. Accessed November 30, 2017.

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Thyroid Screening Questionnaire

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Patient Name_____ Date_____

Put a check by the following statements that apply to your family history, your personal history, and the symptoms that you may have.

HISTORY

- ☐ My family (parent, sibling, child) has a history of thyroid disease
- ☐ I've had a thyroid problem (i.e., hyperthyroidism, Graves' disease, Hashimoto's thyroiditis, post-partum thyroiditis, goiter, nodules, thyroid cancer) in the past
- ☐ A member of my family or I have currently or in the past been diagnosed with an autoimmune disease
- ☐ I have had radiation treatment to my head, neck, chest, tonsil area, etc.
- ☐ I grew up, live, or work near or at a nuclear plant
- ☐ Women: I have a history of infertility or miscarriage

SIGNS AND SYMPTOMS

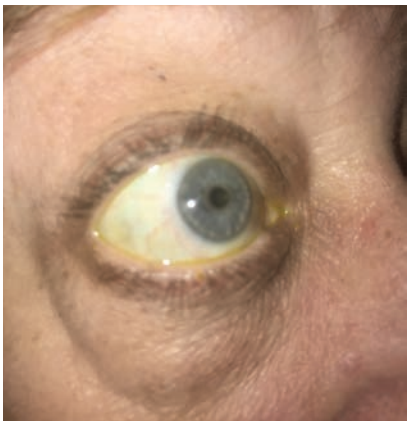
- ☐ I am gaining weight for no clear reason or am unable to lose weight with a diet and exercise program
- ☐ My "normal" body temperature is low (below 98.2° when I take it)
- ☐ My hands and feet are cold to the touch and I frequently feel cold when others do not
- ☐ I feel fatigued or exhausted more than normal
- ☐ I have a slow pulse, and/or low blood pressure
- ☐ I have been told I have high cholesterol
- ☐ My hair is rough, coarse dry, breaking, brittle, or falling out
- ☐ My skin is rough, coarse, dry, scaly, itchy, and thick
- ☐ My nails have been dry and brittle, and break more easily
- ☐ My eyebrows appear to be thinning, particularly the outer portion
- ☐ My voice has become hoarse and/or 'gravelly'
- ☐ I have pains, aches, stiffness, or tingling in joints, muscles, hands and/or feet
- ☐ I have carpal tunnel syndrome, tendonitis, or plantar fasciitis
- ☐ I am constipated (less than 1 bowel movement daily)
- ☐ I feel depressed, restless, moody, sad
- ☐ I have difficulty concentrating or remembering things
- ☐ I have a low sex drive
- ☐ My eyes feel gritty, dry, light-sensitive
- ☐ My neck or throat feels full, with pressure, or larger than usual, and/or I have difficulty swallowing
- ☐ I have puffiness and swelling around the eyes, eyelids, face, feet, hands and feet
- ☐ Women: I am having irregular menstrual cycles (longer, or heavier, or more frequent)

Case Example

A 66-year-old black female diagnosed with hypothyroidism was being treated for moderate to severe DED. She was taking levothyroxine 100mcg once daily for the thyroid condition and Restasis (cyclosporine, Allergan), artificial tears and pilocarpine HCL 5mg once daily for her dry eye. Her slit lamp examination showed 2+ punctate epithelial erosions and reduced tear film break-up time bilaterally. Her Ocular Surface Disease Index (OSDI) score was 26. Despite treatment, she showed minimal improvement.

She was referred back to her endocrinologist with a recommended change in therapy to natural desiccated thyroid or similar. She was switched to Armour Thyroid (levothyroxine, liothyronine, Allergan) 60mg once daily.

Many of her symptoms improved within a few months. After two years she no longer uses Restasis, and her OSDI score has improved to 14.



Graves' ophthalmopathy often presents with exopthalmos, or bulging eyes, as seen in this patient.

the enlargement of the extraocular muscles. In addition, visual field testing may reveal defects caused by optic neuropathy.

To date, no effective means of preventing Graves' disease or reliably altering its course exist. Current therapeutic options for mild TAO include observation, patient education, lifestyle changes (i.e., smoking cessation, salt restriction, sun protection) and ocular surface lubrication. Symptoms of moderate disease can be managed with topical cyclosporine, nighttime eyelid taping, moisture goggles, prism glasses or selective ocular patching and moderate-dose oral steroid therapy.[17,18]

Active ocular involvement often requires the use of systemic corticosteroids to manage inflammation. Depending on side effects and patient tolerance, they may be tapered over three to six months.[19]

Once a patient has progressed to severe disease, high-dose corticosteroids, external beam radiation and steroid-sparing immunosuppressive agents for reducing the inflammation may be necessary. In addition, surgery may be indicated to correct the residual abnormalities secondary to fibrosis in the inactive state of the disease. These interventions are aimed at the consequences of the disease, rather than targeting its cause. Unfortunately, they do not prevent or reverse the pathological changes in the orbital tissues.[17,18]

Hypothyroidism. Optometrists likely see a high percentage of HT patients due to signs and symptoms of significant dry eye disease (DED), which is extremely common in this patient population. While studies documenting the association between OSD and Graves' orbitopathy are numerous, those linking HT and DED are lacking. However, one study that identified DED comorbidities revealed that patients with DED were twice as likely to have HT compared with patients without DED.[20]

Systemic Treatment

Patients diagnosed with a thyroid disorder can pursue any number of therapies ranging from medical management to surgical intervention:

Hyperthyroidism. While surgical thyroidectomy is the most successful treatment for Graves' disease, medicinal options include the antithyroid drugs propylthiouracil and Tapazole (methimazole, Pfizer). Beta-blockers may also be needed in cases of thyroid storm, or thyrotoxic crisis, which is an acute, life-threatening, hypermetabolic state induced by excessive release of TH in individuals with thyrotoxicosis. The clinical features are fever, tachycardia, hypertension and neurological and GI abnormalities.[21]

Another possible treatment is radioactive iodine therapy, although it is contraindicated for moderate to severe Graves' orbitopathy.

Hypothyroidism. Patients with Hashimoto's disease can manage many of their symptoms nutritionally by addressing selenium and vitamin D deficiencies and by maintaining proper iron and iodine levels. Patients should also consider eliminating gluten from their diet, as research suggests gluten intolerance, or Celiac's disease, is associated with Hashimoto's thyroiditis.[22]

Oxidative stress is also a factor in Hashimoto's, and efforts to increase antioxidant status could be helpful.[23] Specifically, glutathione, perhaps the body's most potent endogenous antioxidant, is reduced in Hashimoto's. While direct supplementation of glutathione is often ineffective, n-acetylcysteine (NAC) supplementation can aid in the endogenous biosynthesis of glutathione. NAC and alpha lipoic acid are excellent strategies to lower oxidative stress.[24] Nuclear factor-erythroid 2 p45-related factor-2 (Nrf2) activation, a mechanism for cellular defense, also increases antioxidant

This screening questionnaire is a useful tool for patients at risk for hypothyroidism or those currently taking thyroid medications who have continuing symptoms.

production, such as glutathione, in response to reactive oxygen species.[25]

Although HT as a whole is really a deficiency of active T_3 in tissues such as the eye, patients are treated based on the TSH output from the pituitary gland, in response to TRH from the hypothalamus. However, evidence suggests a normal TSH may still exist when serum T_3 is low, indicating only specific tissues are in a hypothyroid state.[26]

HT treatment goals include maintaining "normal" TSH levels and resolution of symptoms. Some guidelines establish a therapeutic goal between 0.4mIU/L and 4.0mIU/L.[16] Typically, clinicians seek to regulate TSH and fail to inquire about a patient's quality of life—perhaps the main reason for continued symptoms such as dry eye, which is difficult to treat with unbalanced TH levels.

Medical management is dominated by monotherapy with levothyroxine (LT4) for all HT conditions, as it is the only recommended treatment by the 2014 ATA and AACE guidelines. LT4 is now among the most prescribed drugs in the United States, ranking in the top two monthly among all prescriptions.[27] Few clinicians, including endocrinologists, will go outside ATA guidelines, even when patients fail to reach a euthyroid state. Unfortunately, the therapy often falls short due to the shortcomings of solely relying on TSH to manage the disease.

One study asserts that normalizing T_3 should be a "biological priority" and that many patients fail to achieve a normal T_3 on LT4 monotherapy, despite a normal TSH.[28] When given the option, many patients do well with combination therapy such as Nature-Throid (RLC Laboratories) or Armour Thyroid (levothyroxine, liothyronine, Allergan).

More than 50 years ago, prior to the synthesis of LT4, the only thyroid hormone replacement available was natural desiccated thyroid sourced from porcine thyroid glands. Several brands are still available in the United States today, as is a synthetic T_3, available as Cytomel (liothyronine sodium, Pfizer) and Triostat (liothyronine, JHP Pharmaceuticals). Clinicians rarely encounter patients on these products or natural desiccated thyroid. With such discrepancies in the guidelines, clinicians should treat each patient individually and take into account not only TSH levels, but also patient symptoms and quality of life.[29]

Optometrists are certainly familiar with a Graves' disease diagnosis and are comfortable in the role we play in its treatment and monitoring. However, patients with subclinical or uncontrolled hypothyroidism are far more common in optometric practice than you might think. We can have a life-long impact on thousands of patients if we learn to recognize the implications of the various thyroid dysfunctions and help patients better understand their treatment options. Take a functional approach and take an interest in getting to the root cause, especially when it comes to the top symptom, DED. ■

Dr. Dixon practices at Advanced EyeCare Center in Perry and Bonaire, Ga., and is a graduate of Asbury College and the University of Alabama School of Optometry.

Clinical Pearls

Dry eye patients who might be on concomitant treatment for HT may be difficult to spot in your practice. The first step is to closely observe each patient for signs of a lower body temperature (cold hands) and thinning eyebrows temporally, in addition to their dry eye signs and symptoms. Although exceptions exist, a typical hypothyroid patient is female, fatigued, has dry skin and hair loss to the point the scalp is visible and struggles with weight and depression. They are frequently taking low-dose LT4 and will tell you their levels are fine according to their primary care physician.

However, when patients say their blood work is fine, they might mean TSH is in the "normal" range—practically a moving target. TSH normative ranges vary and must be in the optimal range for each patient, just as we select a target IOP for our glaucoma patients. TSH is the best marker we have, but not the only biochemical parameter that clinicians should monitor. Other biochemical markers, such as free T_3, can provide more pertinent information.

When multiple symptoms or clinical signs of DED exist, patients on thyroid medication who report "normal" TH levels may not be on optimal treatment that ensures sufficient T_3. These uncontrolled patients are common in our practices and appreciate a conversation about the possible relationship to their thyroid condition.

Clinicians should open a line of communication with the treating provider to confirm whether or not the patient is compliant with treatment. This is a great time to inquire about recent laboratory testing, as it will aid in the decision making process for concomitant ocular therapies. Here is a sample letter to a treating clinician you can use in your office:

MD, NP, PA:

I am treating our mutual patient _____ for chronic dry eye disease.

Studies have shown a correlation with hypothyroidism and dry eye. As we consider treatment options for this patient, would you mind sharing their thyroid lab results? Please include whether or not the TSH target has been obtained, and if Hashimoto's disease is present based on antibody levels.

I appreciate the opportunity to share in the management of this patient. You may fax lab results to xxx-xxx-xxxx.

Sincerely,

Your comanaging optometrist

1. Mullur R, Liu YY, Brent GA. Thyroid hormone regulation of metabolism. Physiol Rev. 2014;94(2):355-82.
2. American Thyriod Association. General information. www.thyroid.org/media-main/about-hypothyroidism. Accessed December 6, 2017.
3. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado Thyroid Disease Prevalence Study. Arch Intern Med. 2000;160(4):526-34.
4. Mathur R. Thyroid and Iodine-Part 1. MedicineNet. www.medicinenet.com/script/main/art.asp?articlekey=18119. Accessed December 6, 2017.
5. de Benoist B, Andersson M, Takkouche B, Egli I. Prevalence of iodine deficiency worldwide. Lancet. 2003;362(9398):1859-60.
6. Negro R. Selenium and thyroid autoimmunity. Biologics. 2008 Jun;2(2):265-73.
7. Hollowell JG, Staehling NW, Flanders WD, et al. Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab. 2002;87(2):489-99.
8. Gleeson H, Kelly W, Toft A, et al. Severe thyroid eye disease associated with primary hypothyroidism and thyroid-associated dermopathy. Thyroid. 1999;9(11):1115-8.
9. Tunbridge WM, Evered DC, Hall R, et al. The spectrum of thyroid disease in a community: the Whickham survey. Clin Endocrinol (Oxf). 1977 Dec;7(6):481-93.
10. Lyons T, Siri K. Cutting-edge therapies for autism 2011-2012. New York: Skyhorse Publishing; 2012.
11. Murphy NC, Diviney MM, Donnelly JC, et al. The effect of maternal subclinical hypothyroidism on IQ in 7- to 8-year-old children: A case-control review. Aust N Z J Obstet Gynaecol. 2015;55(5):459-63.
12. Wentz I. Hashimoto's and TPO Antibodies. The Thyroid Pharmacist. February 4, 2015. https://thyroidpharmacist.com/articles/hashimotos-and-tpo-antibodies. Accessed November 9, 2017.
13. Institute for Functional Medicine. Thyroid screening questionnaire. www.hbdnutrition.com/wp-content/uploads/2014/11/Thyroid-Screening-Questionnaire.pdf. Accessed December 7, 2017.
14. Gaitonde DY, Rowley KD, Sweeney LB. Hypothyroidism: an update. Am Fam Physician. 2012;86(3):244-51.
15. Wartofsky L, Dickey RA. The evidence for a narrower thyrotropin reference range is compelling. J Clin Endocrinol Metab. 2005;90(9):5483-8.
16. Jonklaas J, Bianco AC, Bauer AJ, et al. Guidelines for the treatment of hypothyroidism: prepared by the ATA Task Force on Thyroid Hormone Replacement. Thyroid. 2014;24(12):1670-751.
17. Maheshwari R, Weis E. Thyroid associated orbitopathy. Indian J Ophthalmol. 2012;60(2): 87-93.
18. Tellez M, Cooper J, Edmonds C. Graves' ophthalmopathy in relation to cigarette smoking and ethnic origin. Clin Endocrinol (Oxf). 1992;36(3):291-4.
19. Gould DJ, Roth FS, Soparkar CNS. The diagnosis and treatment of thyroid-associated ophthalmopathy. Aesth Plast Surg 2012;36:638-48.
20. Wang TJ, Wang IJ, Hu CC, Lin HC. Comorbidities of dry eye disease: a nationwide population-based study. Acta Ophthalmol. 2012;90(7):663-8.
21. Chiha M, Samarasinghe S, Kabaker AS. Thyroid storm: an updated review. J Intensive Care Med. 2015 Mar;30(3):131-40.
22. Liontiris MI, Mazokopakis EE. A concise review of Hashimoto thyroiditis (HT) and the importance of iodine, selenium, vitamin D and gluten on the autoimmunity and dietary management of HT patients. Points that need more investigation. Hell J Nucl Med. 2017;20(1):51-56.
23. Rostami R, Aghasi MR, Mohammadi A, Nourooz-Zadeh J. Enhanced oxidative stress in Hashimoto's thyroiditis: inter-relationships to biomarkers of thyroid function. Clin Biochem. 2013;46(4-5):308-12.
24. Kerksick C, Willoughby D. The antioxidant role of glutathione and n-acetyl-cysteine supplements and exercise-induced oxidative stress. J Int Soc Sports Nutr. 2005;2(2):38-44.
25. Tebay LE, Robertson H, Durant ST, et al. Mechanisms of activation of the transcription factor Nrf2 by redox stressors, nutrient cues, and energy status and the pathways through which it attenuates degenerative disease. Free Radic Biol Med. 2015 Nov;88:108-46.
26. Sherine M, Bianco A, Bianco AC. Defending plasma T3 is a biological priority. Clin Endocrinol (Oxf). 2014;81(5):633-41.
27. The top 50 prescription drugs filled in the U.S. Lowest Med. www.lowestmed.com/top-50-prescription-drugs-filled. Accessed November 9, 2017.
28. Sherine M. Bianco A, Bianco AC. Defending plasma T3 is a biological priority Clin Endocrinol (Oxf). 2014 Nov;81(5):633-41.
29. Waise A, Price HC. The upper limit of the reference range for thyroid-stimulating hormone should not be confused with a cut-off to define subclinical hypothyroidism. Ann Clin Biochem. 2009;46(2):93-8.

OSC QUIZ

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

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1. The normal thyroid gland secretes T4 and T3 in the ratio of:
a. 90/10.
b. 60/40.
c. 30/70.
d. 10/90.

2. The active thyroid hormone the body needs for energy is:
a. T_4.
b. T_3.
c. Calcitonin.
d. TSH.

3. The number of United States citizens with a thyroid disorder may be as high as:
a. One million.
b. Five million.
c. 20 million.
d. 60 million.

4. The US prevalence of hyperthyroidism is:
a. 10%.
b. 5%.
c. 1% to 3%.
d. 0.1% to 0.3%.

5. The number one cause of hyperthyroidism in the United States is:
a. Iodine deficiency.
b. Graves' disease.
c. Goiter.
d. Thyroidectomy.

6. TAO occurs in ____ of Graves' patients.
a. 100%.
b. 75%.
c. 50%.
d. 25%.

7. Symptoms of hyperthyroidism include all of these, except:
a. Weight gain.
b. Heat intolerance.
c. Increased heart rate.
d. Disturbed sleep.

8. The most common initial symptom in Graves' disease is:
a. Lid retraction.
b. Ocular surface disease.
c. Periorbital edema.
d. Diplopia.

9. Graves' disease is most successfully treated with:
a. Methimazole.
b. Radioactive iodine.
c. Orbital decompression.
d. Surgical thyroidectomy.

10. Roughly 80% of TAO cases are associated with:
a. Goiter.
b. Graves' disease.
c. Hyperthyroidism.
d. Hypothyroidism.

11. According to the National Academy of Clinical Biochemistry, 95% of normal individuals have a TSH level below:
a. 10mIU/L.
b. 5.5mIU/L.
c. 2.5mIU/L.
d. 1mIU/L.

12. Symptoms of hypothyroidism include:
a. Dry, gritty eyes.
b. Fatigue.
c. Cold intolerance.
d. All of the above.

13. The Colorado Thyroid Disease Prevalence Study found that, among patients taking thyroid medicine, only _____ were in the normal TSH range.
a. 10%.
b. 30%.
c. 60%.
d. 90%.

OSC QUIZ

14. Levothyroxine is the _____ most prescribed medication in the United States.
a. 2nd.
b. 20th.
c. 200th.
d. 2,000th.

15. Comorbidities of hypothyroidism include:
a. Heart disease and cholesterol issues.
b. Fibroids and infertility.
c. Autism and low IQ.
d. All of the above.

16. One study found that patients with DED were __ more likely to have hypothyroidism.
a. 1.5x.
b. 2.0x.
c. 2.5x.
d. 3.0x.

17. The most common cause of hypothyroidism in the United States is:
a. Iodine insufficiency.
b. Pharmceuticals.
c. Thyroidectomy.
d. Hashimoto's disease.

18. The second most common cause of hypothyroidism in the United States is:
a. Iodine insufficiency.
b. Pharmceuticals.
c. Thyroidectomy.
d. Hashimoto's disease.

19. Diagnostic workup for suspected or treated hypothyroid patients should include:
a. TSH.
b. Free T_3 and free T_4.
c. Antibodies.
d. All of the above.

20. When treating patients for DED, optometrists should realize:
a. Levothyroxine always restores patients to a normal thyroid state.
b. A "normal" TSH ensures that the patient will be asymptomatic.
c. Nutritional deficiencies are not linked to hypothyroidism.
d. Many patients continue to have persisting symptoms, including dry eyes, while taking levothyroxine.

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Examination Answer Sheet

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Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ
5. Ⓐ Ⓑ Ⓒ Ⓓ
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15. Ⓐ Ⓑ Ⓒ Ⓓ
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. Better understand the function of the thyroid gland and its many dysfunctions. ① ② ③ ④ ⑤
22. Become familiar with the differences between hypo- and hyperthyroidism. ① ② ③ ④ ⑤
23. Improve my clinical ability to screen for and diagnose thyroid disorders. ① ② ③ ④ ⑤
24. Increase my understanding of the ocular effects of thyroid conditions. ① ② ③ ④ ⑤
25. Increase my knowledge of systemic treatments for various thyroid disorders. ① ② ③ ④ ⑤
26. Improve my understanding of the diagnostic testing necessary to properly identify thyroid conditions. ① ② ③ ④ ⑤

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

27. The content was evidence-based. ① ② ③ ④ ⑤
28. The content was balanced and free of bias. ① ② ③ ④ ⑤
29. The presentation was clear and effective. ① ② ③ ④ ⑤
30. Additional comments on this course:

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

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Don't Stress

Endothelial blebs be a sign of a problematic scleral lens fit. Here's what you should do to avoid them. **Edited by Joseph P. Shovlin, OD**

Q What is the real significance of endothelial blebs and how do I best view them in my scleral lens wearers?

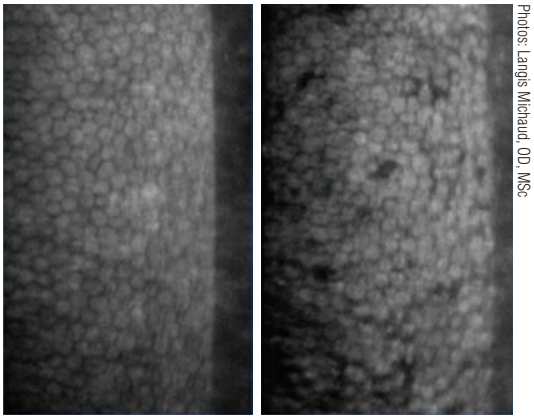
A These are edematous cells "visible under high magnification slit lamp examination or, preferably, through specular microscopy," says Langis Michaud, OD, MSc, chief of the contact lens department at the University of Montreal. "Cell-warped apical surfaces cause the loss of the light ray reflection, making the cell appear black. Consequently, it is possible to observe a transient disappearance of endothelial cells from the mosaic observed in specular reflection."

Blebs occur shortly after a contact lens is fit, creating hypoxic stress, and they disappear once the lens is removed or the cornea adapts to the lens, says Dr. Michaud. "This is called the bleb response, and it has been reported after lens wear with oxygen impermeable polymethyl methacrylate, rigid gas permeable, hydrophilic and, most recently, scleral contact lenses." In a recent study, researchers found higher central tear fluid thickness was associated with a greater number of blebs during scleral lens wear.[1]

"This represents proof that hypoxic stress occurs during scleral wear," says Dr. Michaud. He also believes it validates theoretical models that predict hypoxia will be generated if a scleral lens is too thick (>250µm) and its fit has more than 200µm of central clearance.[2]

Despite this, induced edema remains clinically invisible because it does not exceed 3% during lens wear and affects only the central cornea (except in compromised corneas).

"Some authors consider this induced edema as benign or not clinically significant, and others compare it to physiological edema," says Dr. Michaud. However, he considers this misleading because the cornea recovers from physiological edema within an hour post-awakening. In sclerals, 1% to 3% of edema remains for the entire wearing period, and because the lenses are inserted shortly after awakening, the cornea can never recover, Dr. Michaud says. "Nobody can tell, at this point, how a cornea will react to this chronic hypoxic stress long-term, but corneal edema moved the entire soft lens industry to research and find highly permeable materials, so it should do the same for scleral lenses, where optimal designs are needed."



Photos: Langis Michaud, OD, MSc

At right, the black spots are endothelial blebs. At left, the same cornea without blebs prior to lens wear.

Solving the Problem

To minimize hypoxic stress, Dr. Michaud offers these guidelines: keep the lens as thin as possible and align it in every quadrant to alleviate flexure; keep central corneal clearance under 200µm after lens stabilization; select the highest Dk material; and monitor clinical signs.

When monitoring, practitioners should perform pre- and post-fit global corneal pachymetry to establish comparative maps and track swelling; check for microcysts and bullae or loss of transparency, especially if the endothelial layer is compromised or if the cell count is reduced; note any increased symptomatology of glare and haloes, especially at lens removal; and observe the endothelial layer under high magnification slit lamp 15 to 20 minutes after lens wear to track the presence of endothelial blebs.

"Whenever possible, consider redesigning scleral lenses to alleviate hypoxic stress," says Dr. Michaud. "In some cases, however, such as when the endothelial cell count is less than 1000 cells/mm^2, you may need to consider options other than scleral lenses, especially in challenged graft patients." ■

1. Giasson CJ, Morency J, Melillo M, Michaud L. Oxygen tension beneath scleral lenses of different clearances. Optom Vis Sci. 2017;94:466-75.
2. Compañ V, Aguilella-Arzo M, Edrington TB, Weissman BA. Modeling corneal oxygen with scleral gas permeable lens wear. Optom Vis Sci. 2016;93(11):1339-48.

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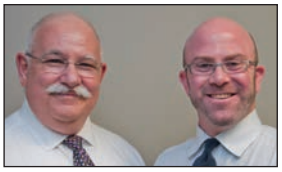


Hone Your Astigmatic Refraction

Use these tips to refine your skills and perform this task more swiftly.

By Marc B. Taub, OD, MS, and Paul Harris, OD

"Which is better, one or two?" You've probably heard that repeated back to you at parties any time you mention what you do for a living. But you know it's more than a catchphrase; it represents the astigmatic portion of a patient's refraction. You also know that it can be a real time thief. This month, we identify several steps you can take to save time on refractions from start to finish, without ignoring that vital refractive data, so you can get out of the exam lane and back to the party.

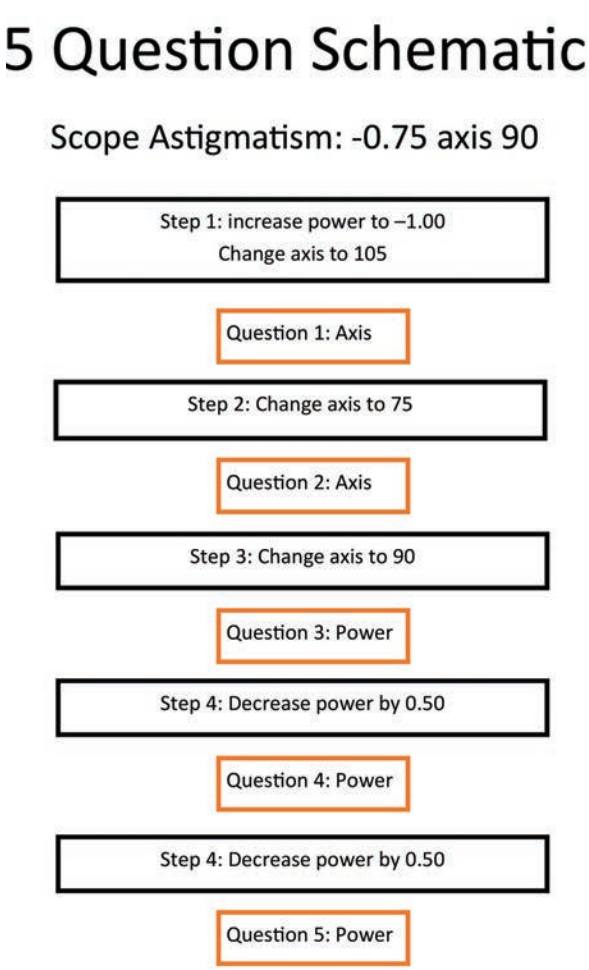
Get Out Your Retinoscope

Any refraction's foundation comes from a retinoscopy exam. Obtaining this starting point accurately sets the stage for the most efficient refraction, particularly in the portion of the refraction where we address astigmatism. We tell our students that, for every 20 seconds you spend getting a good starting point for the refraction with your retinoscopy, you may save two minutes.

When working with novices at retinoscopy, consider these two points: (1) Make sure they move only the retinoscope. Some of our students tend to bob their head up and down or side-to-side to move the retinoscopy light through the pupil. This actually makes the assessment more difficult. The key is to stay steady in space and pivot the scope off the forehead for both movements. (2) When scoping axes which are away from 90 or 180, make sure they rotate the scope. To shift the scope up and down through axis 90, the bottom is moved slightly away from the chin and towards the chin. A common mistake is to move the light either too far or too fast to make the direction of the movement easy to see. This is one of those instances when slowing down can actually speed up the entire process.

Start by getting the reflex centered and stable. As you move the bottom of the retinoscope slowing towards or away from your chin, you should be able to note the direction of movement before you complete even a half of a cycle. The second movement is a pure twist of the hand, rotating the retinoscope along its long axis. After getting things settled, a smooth slow rotation of the scope should allow you to see the direction of movement before you complete even half a cycle.

With regards to those off-axis cylinders, I don't know too many people who can adjust the collar to axis 120, keep the handle straight up and down and perform the movement necessary to move the light purely through the axis 120 meridian. A simpler method is to keep the collar



Focusing on ways to streamline aspects of your astigmatic refraction exam can help you see more patients in less time.

orthogonal to the scope itself and to rotate the arm to axis 120, keeping the top of the scope against the forehead. The physical movements then remain the same. To get axis 120, move the bottom of the scope away and towards the lower part of your face, but do so directly along the axis of the scope itself. To scope the opposite axis, perform the same twisting motion of the scope along the axis of the scope itself.

Choosing a JCC Power

Ever wonder how the +/- 0.25 Jackson Cross Cylinder (JCC) became so widely accepted as the best choice? The old Bausch & Lomb Greens Phoropter came with a set of auxiliary lenses, which included three powers of JCC lenses; +/- 0.25, 0.37, and 0.50 which could be easily swapped in and out as needed.

Having used the Greens in practice for many years, we found the +/- 0.50 allowed for faster refinement of both the cylinder axis and power than either of the other two powers. The differences between choices one and two were simply bigger and easier to see. Does the smaller power JCC allow for more refined refractive endpoints? In our experience, only in a very small percentage of patients we see. For most, the +/- 0.25 JCC does not make a big enough difference between the choices for them to detect or report actual differences and we find it is actually more time consuming as patients incessantly ask to review the options again. We recommend +/- 0.50 JCC lenses.

The Five Questions

Set a goal to see if you can do this testing with a maximum of five questions. When you achieve this, you will know that your scope gave you a great starting place and the differences you were showing your patient were big enough for them to make simple and swift choices.

Say, for instance, you have scoped a cylinder of -0.75 at axis 90 and have confirmed that the sphere power is at the right level of accommodative stimulus to begin your cylinder testing. Let us also assume that we are not dealing with someone having worse best-corrected visual acuity of 20/20 for the sake of working through these routines. In this case, we suggest a single 20/40 line be the target.

Here are the steps and the questions we would ask this patient. Note that what you see in the parentheses below is not to be said aloud, but is to help you understand what we are asking based on the optics.

Step 1: Increase the cylinder power to -1.00 and turn the axis to 105. Set the JCC to test for AXIS.

Question 1: Which is better, this way "1" (towards where I scoped) or this way "2" (away from where I scoped)?

Patient: Towards.

Step 2: Turn the axis to 75. Test for AXIS.

Question 2: Which is better, this way "1" (towards where I scoped) or this way "2" (away from where I scoped)?

Patient: Towards.

Step 3: Turn the axis back to 90 and set the JCC to test for POWER

(Remember the power of the cylinder is -0.25 higher than what we scoped.)

Question 3: Which is better, this way "1" (more than more than what I scoped) or this way "2" (less than more than what I scoped)?

Patient: Less.

Step 4: Reduce the power of the cylinder by 0.50, from -1.00 to -0.50, in this example

Question 4: Which is better, this way "1" (less than less than what I scoped) or this way "2" (more than less than what I scoped)?

Patient: More.

Step 5: Since we now know that the axis of this small cylinder is between 75 and 105, and the power is less than -1.00 and more than -0.50, we can increase the power of the cylinder by 0.25, from -0.50 to -0.75, in this example

Question 5: Which is better, this way "1" (less than what I scoped) or this way "2" (more than what I scoped)?

Cylinder Exchange

If in answering these questions the patient hesitates, you can assume the two choices look similar. To us, this is not evidence that the power needs to be higher, they have not earned the -0.75 so we go back down to -0.50 and move on.

If the patient quickly says that the "less than what I scoped" option is better, we also drop down to the -0.50 and move on. If, and only if, rather quickly, at the same speed or only slightly slower than the previous four responses, the patient responds that the "more than what I scoped" option was better, we kick the cylinder back up to -0.75 and move on.

With higher-power cylinders, we move the initial testing axis away from our scoped axis less. For example, with a -2.50 cylinder, we may shift the axis only +/- 5 degree to either side of where we scoped the patient.

If some of the ideas here are new to you, give them a try and see if you can get to your endpoints faster and still prescribe with confidence. ■

Foretold in the Stars

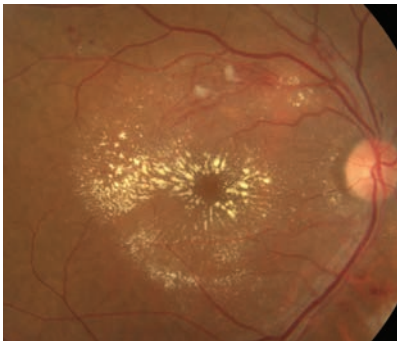
How do this patient's comorbidities inform your diagnosis?

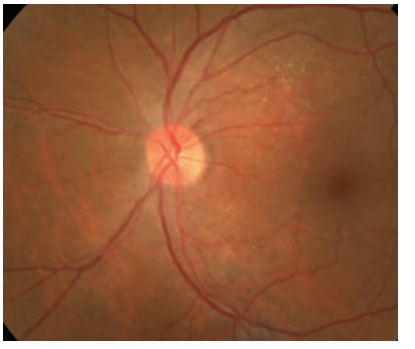
By Mark T. Dunbar, OD

A 54-year-old Hispanic male presented with complaints of blurred vision in his right eye for the past month. The patient has never had eye problems in the past and has always enjoyed good vision.

The patient's medical history is significant for being HIV positive and having hypertension. He also has had Kaposi's sarcoma as a consequence of HIV and is being treated with chemotherapy. He is on a host of medications for his HIV and hypertension.

On examination, his best-corrected visual acuity was 20/40 OD and 20/20 OS. Extraocular motility testing was normal. Confrontation fields were full-to-careful-finger-counting OU. His pupils were equally round and reactive to light, with no afferent pupillary defect. His anterior segment exam was unremarkable OU. He had no anterior chamber cells or vitreous cells. His intraocular pressures measured 14mm Hg OU. Dilated fundus exam revealed changes as seen in the fundus photos (*Figures 1 and 2*). An OCT was performed and is also available for review (*Figure 3*).





Figs. 1 & 2. Note the obvious finding in the right eye (at left) of our patient, but don't overlook the finding in the left eye.

Take the Retina Quiz

1. How would you characterize the macular changes in the right eye?
a. Exudative maculopathy.
b. Macular star formation.
c. Neuroretinitis.
d. Crystalline maculopathy.

2. What additional testing would be most useful in confirming the diagnosis?
a. Fluorescein angiography.
b. Indocyanine green angiography.
c. Blood pressure.
d. Blood serology including indirect fluorescent antibody assay for *Bartonella henselae*.

3. What is the likely diagnosis?
a. Branch retinal vein occlusion with macular edema.
b. Malignant hypertension or hypertensive emergency.
c. Cytomegalovirus retinitis.
d. Cat scratch neuroretinitis.

4. How should this patient be managed?
a. Referral for anti-viral therapy.
b. Visual field and neuro-ophthalmology consult.
c. Lumbar puncture and MRI.
d. Immediate referral to the ER for blood pressure control.

5. What is the likely visual prognosis for this patient after treatment?
a. Very poor.
b. Moderate.
c. Above average.
d. Excellent.

For answers, see page 100.

Discussion

The macular changes that are seen in the right eye represent a macular star. Because of the exudate, our patient also has a neurosensory retinal detachment, as seen on the SD-OCT. In addition, we noticed other vascular-related changes. Superior to the macula, flame hemorrhages and, possibly, a small branch retinal vein occlusion are visible. Also, there is a small hemorrhage nasal to the disc and, in the left eye, there is also a flame hemorrhage superiorly and resolving exudate. So, what is going on with our patient?

The presence of a macular star could be consistent with a neuroretinitis, but there were no vitreous cells, and the vascular changes that are seen in our patient are bilateral.

What's more, when we measured his blood pressure, it was 220/110! Based on the clinical finding and elevated blood pressure, our patient has malignant hypertension, or as it is now more accurately referred to, a hypertensive emergency with end organ damage (eye).

The Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure has provided a classification scheme for hypertension that employs systolic and diastolic criteria. Systolic blood pressure greater than 179mm Hg or diastolic blood pressure greater than 109mm Hg is classified as hypertensive crisis. The condition can be further categorized as either hypertensive emergency or hypertensive urgency.[1] End organ damage in the presence of significantly elevated blood pressure is classified as hypertensive emergency, whereas hypertensive crisis occurs in the absence of end organ damage. End organ damage may manifest in the central nervous system, eye, heart (left ventricular dysfunction) and kidney. Hypertensive encephalopathy is associated with severe headaches, change in mental status, transient convulsions, stupor and coma.[2]

"Malignant hypertension" was used in the past to describe grade IV hypertensive retinopathy including elevated blood pressure and papilledema; however, this term has been removed from National and International Blood Pressure Control Guidelines and is now referred to as hypertensive emergency.[2]

Hypertensive crisis occurs in approximately 1% of the hypertensive population.[3] The presence and extent of end organ damage is related to the level and rate of rise of blood pressure, and the level of underlying hypertensive damage and arterial/capillary change prior to the acute rise in blood pressure. However, organ dysfunction is uncommon with a diastolic blood pressure lower than 120mm Hg.[4] It is likely our patient's blood pressure had previously been even higher than what was measured on the day of his eye exam.

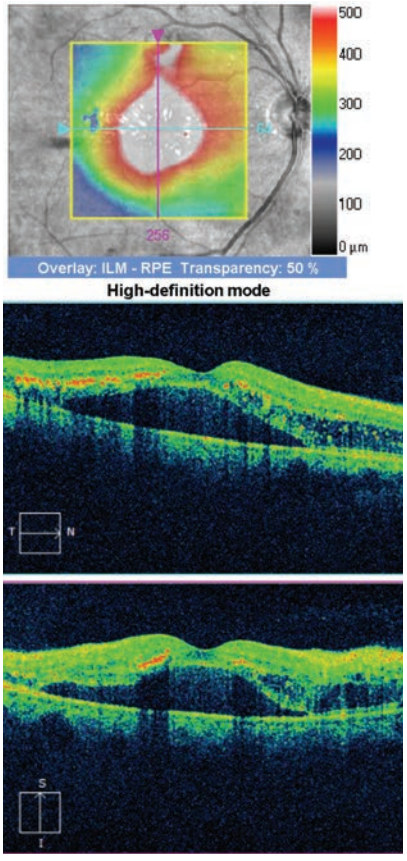


Fig. 3. SD-OCT of our patient's right eye.

Plan of Action

Our patient presented with a macular star formation and a neurosensory detachment in the right eye as a result of the massive exudate. During a hypertensive crisis, there is a loss of autoregulation of the retinal arterioles, which leads to a breakdown of the blood-retina barrier with leakage of plasma and proteins. Interestingly, our patient didn't have disc swelling, which is commonly seen with a hypertensive emergency, but that doesn't mean he didn't ever have it. In fact, he probably did have disc swelling as evidenced by the flame hemorrhages adjacent to the optic nerve and the resolving exudate in the left eye, but he was in a resolving phase at the time of presentation. It is also interesting to note that the retinal arteries and veins were not significantly attenuated as you would expect to see. In fact, the caliber of the vessels seemed pretty normal, though he did have a prominent arterial light reflex.

Patients in hypertensive emergency must have blood pressure lowered immediately via intravenous infusion of antihypertensive medications in order to avoid further end organ damage and morbidity. Conversely, patients with hypertensive urgency should have blood pressure lowered slowly over 24 to 48 hours with oral hypertensive agents to avoid subsequent ischemia and infarction secondary to the rapid change in blood pressure and loss of arteriole autoregulation.[5]

Our patient admitted to having poorly controlled blood pressure and, in fact, was having kidney failure. He was sent to the emergency room, but was lost to follow up for more than two years. When he presented again for an eye evaluation, his best-corrected visual acuity was 20/20 in each eye and he had no evidence of the hypertensive emergency two years prior. He is now undergoing dialysis, but his blood pressure is still poorly controlled. ■

1. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med. 1993;153(2)154-83.
2. Kitiyakara C, Guzman NJ. Malignant Hypertension and Hypertensive Emergencies. J Am Soc Nephrol. 1998 Jan;9(1):133-42.
3. Lee AG, Beaver HA. Acute bilateral optic disk edema with a macular star figure in a 12-year-old girl. Surv. Ophthalmol. Jan-Feb 2002:47(1):42-49.
4. Marik PE, Varon J. Hypertensive crisis, diagnosis and management. CHEST. 2007;131(6):1949-62.
5. Hajjar I, Kotchen TA. Trends in Prevalence, Awareness, Treatment, and Control of Hypertension in the United States, 1988-2000. JAMA. 2003;290:199-206.

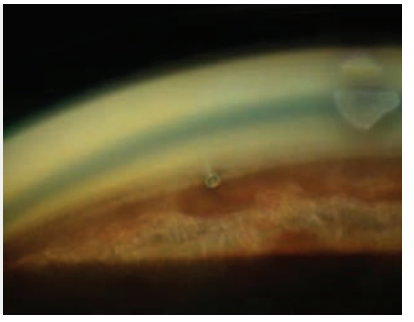
Two Conditions, One Implant

Non-medical options may be more appropriate when glaucoma patients also have ocular surface disease. **By James L. Fanelli, OD**

A 59-year-old Caucasian female presented for a scheduled glaucoma follow-up visit with complaints of decreased vision, foreign body sensation and "uncomfortable" eyes. She was initially seen four years earlier with similar complaints and was diagnosed then as a glaucoma suspect (subsequent testing indicated that she had frank glaucoma, and medication was initiated in both eyes). At that time she was also noted to have generalized ocular surface disease (OSD) with superficial punctate keratitis (SPK), a decreased tear break-up time, a scant tear prism and worsening comfort as the day progressed. She was also found to have anterior and posterior cortical spoking of the crystalline lenses, off the visual axis. Her decreased vision was attributable to the OSD rather than cataracts, and the OSD was addressed appropriately.

Therapies

Over the next several years, her symptoms of irritation fluctuated. Occasionally, her OSD flared, which required topical steroid treatment. Given a glaucoma patient's propensity to be a steroid responder, it is important to select a steroid with limited ocular penetration, and my go-to drug in these cases is fluorometholone. While this agent is a potent steroid, its potency is limited to the ocular surface, making it an ideal drug to use in glaucoma patients with OSD.



A postoperative cataract patient with an iStent in place.

But, because of its poor penetrability, it is not an appropriate steroid to use when the anterior chamber is inflamed. Accordingly, she was periodically medicated with the topical steroid to quell her symptoms. Complicating the recovery was the medical management of her glaucoma. While the patient responded nicely to ocular hypotensive agents, their introduction to her eyes every day certainly did not help the OSD complaints.

Examination

At the recent follow-up visit, her entering acuities were 20/50 OD and 20/50- OS. Her ocular medications consisted of unpreserved 0.5% timolol QAM OU, Restasis (cyclosporine, Allergan) BID OU, and unpreserved artificial tears on a PRN basis. Her systemic medications included warfarin, simvastatin and over-the-counter fish oil. She had no known allergies to medications.

A slit lamp evaluation of the anterior segments was remarkable for diffuse SPK in the left eye more so than the right, which, when compared with previous visits, was deemed an average presentation for her. The anterior chambers were clear in both eyes. Through dilated pupils, her crystalline lenses were characterized by progression of the anterior and posterior cortical spoking, now involving the visual axis. We noted minimal nuclear cataract progression in both eyes.

Her cup-to-disc ratios were stable at 0.5 x 0.65 OD and 0.4 x 0.65 OS, each nerve with thinning of the neuroretinal rim inferotemporally. At this visit, Heidelberg retinal tomography (HRT 3) and optical coherence tomography imaging demonstrated stability of the neuroretinal rims, Bruch's membrane openings, the perioptic retinal nerve fiber layer and the macular ganglion cell scans in both eyes. Her baseline central corneal thicknesses, obtained several years earlier, were 524µm OD and 531µm OS.

Macular evaluations were normal, with only fine retinal pigment epithelium granulation. We noted bilateral posterior vitreous detachments of several years' duration. The retinal vasculature was characterized by mild arteriolarsclerotic retinopathy consistent with her systemic cardiovascular history, and there were scattered arteriovenous crossing changes, which were deemed stable. Her peripheral retinal evaluations were normal, and the scattered cystoid was stable in both eyes.

Previous visual field studies demonstrated early glaucomatous field loss as obtained by flicker defined form threshold strategy testing, which was not seen on SAP white-on-white perimetry. Repeat testing over several years demonstrated no progression of the field defects. Earlier gonioscopic evaluations demonstrated open angles in both eyes, though ultrasound biomicroscopy imaging demonstrated a slight plateau iris configuration. Her angle studies also have remained stable over the past few years.

Discussion

The patient is presenting with complaints of decreased vision as well as chronic issues with discomfort in both eyes. She was found to have both progressed cataracts as well as SPK on the visual axis. Her glaucoma was stable.

Two important questions must be answered in this case to plot a proper course of action: what's causing her decreased vision, and what's her target intraocular pressure (IOP)?

Regarding the question of the decreased vision, this case highlights the overlap of both ocular surface disruption and progressing cataracts, and their effects on vision. Certainly, if there were no cataracts involved, we would see fluctuations in vision commensurate with the degree of OSD findings. These will be seen at various glaucoma follow-up visits, and over time, a clinician will get a feel for what is "normal" for the patient. This, of course, is no different if cataracts were present, but the challenge is to discern how much of the decrease in vision is attributable to the cataract progression vs. how much is related to the OSD. The answer ultimately determines which course to follow—cataract surgery or more aggressive management of the ocular surface disease.

The second question carries even more importance. Remember, the target IOP set for an individual, ideally, is the highest pressure at which no further damage occurs. While IOP reduction may actually be lower than is necessary to meet that threshold, setting a target pressure accomplishes two things: it helps avoid overmedicating the patient, and it gives us a feel for the aggressiveness of the disease process. If a patient's glaucoma is stabilized with a post-treatment IOP of 15mm Hg, is it really necessary to drive IOP down to 12mm Hg? Certainly, if the treatment involves only one topical glaucoma medication and that results in an IOP of 12mm Hg, we are doing quite well. But if one medication got us to that 15mm Hg threshold, would we really need to add a second medication? I don't think so (in the hypothetical example here).

While this patient originally presented with glaucoma, we've been able to stave off further progression of her disease with a mild-to-moderate reduction in IOP.

In general, I begin glaucoma therapy with a prostaglandin for most patients. And as most patients do, this patient had a significant reduction in intraocular pressure once the prostaglandin was initiated. But, not surprisingly, the addition of any glaucoma medication can aggravate ocular surface disease issues, which is exactly what happened in this case. Ultimately, we settled on unpreserved timolol 0.5% as the glaucoma drug of choice that was tolerable from an ocular surface perspective. While it did not drop IOP as much as other prostaglandins that were prescribed, it did achieve a target level IOP where no further damage was seen.

Given that target IOP in this case does not need to be reduced by 10mm Hg or more and the patient has OSD, we have the perfect scenario to manage both her glaucoma and cataracts with the placement of an iStent (Glaukos) during cataract surgery. iStent placement is estimated to result in IOP lowering of up to 20% from baseline and as much as 10% more than cataract surgery alone provides.[1-3] Furthermore, iStents play a significant role in reducing the medication load, which is helpful for patients with concurrent OSD.[4,5]

With the development of minimally invasive glaucoma surgery techniques, it is more important than ever that the optometrist play an integral role in the cataract surgery planning. The optometrist, after all, has been managing the patient for many years and intimately knows the nuances of their glaucoma. I've seen too many instances where optometrists, in their referral to a cataract surgeon, simply state "consider an iStent." Ultimately, that is a decision best suited for the provider managing their glaucoma; namely, the optometrist. Your cataract surgeon should follow your lead in determining that an iStent is appropriate; if not, perhaps you should be looking for another surgeon. ■

1. Wellik S, Dale E. A review of the iStent trabecular micro-bypass stent: safety and efficacy. Clin Ophthalmol. 2015 Apr;9:677-84.
2. Samuelson T, Katz L, Wells J, et al. Randomized evaluation of the trabecular micro-bypass stent with phacoemulsification in patients with glaucoma and cataract. Ophthalmology. 2011;118(3):459-67.
3. Craven ER, Katz LJ, Wells JM, et al. Cataract surgery with trabecular micro-bypass stent implantation in patients with mild-to-moderate open-angle glaucoma and cataract: two-year follow-up. J Cataract Refract Surg. 2012;38(8):1339-45.
4. Fea A. Phacoemulsification versus phacoemulsification with micro-bypass stent implantation in primary open-angle glaucoma: randomized double-masked clinical trial. J Cataract Refract Surg. 2010;36(3):407-12.
5. Arriola-Villalobos P, Martínez-de-la-Casa JM, Díaz-Valle D, et al. Combined iStent trabecular micro-bypass stent implantation and phacoemulsification for coexistent open-angle glaucoma and cataract: a long-term study. Br J Ophthalmol. 2012;96(5):645-9.

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

Lumps and Bumps Be Gone

Shave excision can be a simple answer to your patient's eyelid margin lesion.

By Leonid Skorin, Jr., DO, OD, MS, and Laura Goemann, OD

Various eyelid lesions are a common occurrence among all patient populations, and their causes are numerous, with the vast majority being benign. Clinicians should always identify the etiology of the lesion—whether it's inflammatory, infectious, structural or neoplastic—as it will direct the proper management.

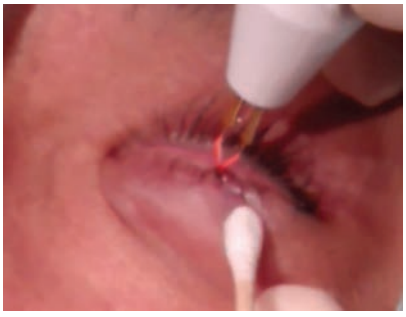
When a benign neoplasm, such as a papilloma, is present on the eyelid margin, a shave excision is often the most appropriate course of action. Although most do not pose any visual compromise, the lesions can cause irritation such as foreign body sensation. Patients presenting with complaints of eyelid lesions are often looking for a cosmetic as well as therapeutic intervention. Clinicians should not dismiss cosmetic concerns, as patients may feel self-conscious about their appearance, and removal can be pivotal to their quality of life.

Removing these lesions is a beneficial procedure for these patients and can be done in the office. Check with your state board for specific regulations regarding optometrists performing this procedure.

Rule Out Malignancy

While most lesions are benign and can simply be removed, each should be evaluated for the possibility of malignancy prior to removal. If a lesion is suspicious or of unknown etiology, refer to an ophthalmic surgeon for excision and biopsy to rule out malignancy. Signs of eyelid malignancy include madarosis and alterations to the normal eyelid integrity.[1] Malignant lesions also tend to grow faster than those that are benign. They can be painful, bleed more easily and form ulcers or crusts in the center of the lesion.

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



After the excision, cauterize the wound to control any bleeding.

Margin Integrity

When lesions are present on the eyelid margin, extra surgical care is warranted, as the structural integrity of the eyelid margin is essential to appearance and functionality. A shave excision is useful when biopsy is warranted, but a level of precision is necessary to maintain margin integrity. It provides the surgeon with histological control over the tissue being excised.

All excised tissue specimens should be sent to a pathology lab for histopathologic analysis. Results are usually returned in one to two weeks, after which you should follow up with the patient to discuss.

Procedure Basics

Prior to excision, the lower eyelid should be anesthetized with lidocaine 2% with 1:100,000 epinephrine. This also induces vasoconstriction of the surrounding vessels, limiting blood loss following the procedure. The area is prepped with betadine, draped and tested for anesthetic effect.

After confirmation of anesthesia, the lesion is grasped with forceps, exposing its base. An 11-degree surgical blade is used to remove the lesion at its base on the lid margin. Care is taken to ensure no notches are made in the margin itself. The removed specimen is placed into container and medium supplied by the lab and sent for analysis.

Cautery helps to control bleeding, and ophthalmic antibiotic ointment is used to prevent infection.

Following the procedure, the patient is instructed to use antibiotic ointment twice a day for five days.

A simple procedure, shave excision can have a significant impact on patients who are encumbered by common eyelid lesions. ■

Dr. Skorin is a consultant in the Department of Surgery, Community Division of Ophthalmology in the Mayo Clinic Health System in Albert Lea, MN.

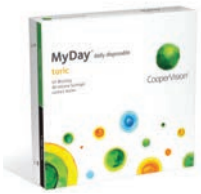
Dr. Goemann received her degree at Pacific University College of Optometry and practices at Eye Q Vision in Mankato, MN and Family Eye Care in Fairmont, MN.

1. Carter S. Eyelid disorders: Diagnosis and management. Am Fam Phys. 1998:2695-2702.

Product Review

Contact Lenses

New Toric Option



Patients with astigmatism can also consider CooperVision's MyDay toric daily disposable contact lenses, which are designed to optimize oxygen permeability with a lens that is only 4.4% silicone, the company says. The new lenses have a base curve of 8.6mm, a diameter of 14.5mm and are currently available in sphere powers from plano to -6.00D (0.25D steps), and -6.00D to -10.00D (0.50D steps), according to the company.

Visit coopervision.com.

Daily Disposables

SynergEyes, through a partnership with Tangible Science, is adding daily disposable soft contact lenses to its portfolio. As part of the partnership, the lenses will also include Tangible Science's Hydra-PEG coating, which has reduced contact lens discomfort for patients wearing gas permeable lenses, the company says. The lenses will be available later in 2018, according to SynergEyes.

Visit synergeyes.com.

Unlimited Scleral Exchanges

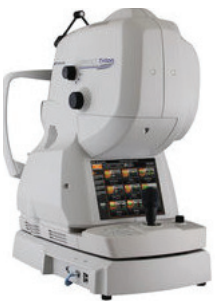
Clinicians who fit Atlantis scleral lenses from X-Cel Specialty Contacts now have a warranty structure that includes unlimited exchanges for 90 days and true paperless returns, according to the company. In addition, all standard, toric and large diameter Atlantis scleral designs now have a single price structure.

Visit www.xcelspecialtycontacts.com.

Diagnostic Technology

New OCT

Those thinking of upgrading their office's optical coherence tomography (OCT) can consider Topcon's DRI OCT Triton Series, which recently received FDA 510(k) clearance. The instrument features a 1µm, 1,050nm light source with a scanning speed of 100,000 A-scans/second and a built-in retinal camera with eye tracking for selected scans. It also combines color, red-free, FA and FAF imaging with swept-source OCT and wide-field scanning, according to the company.



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■ **4-9.** *32nd Annual EyeSki Conference.* Shadow Ridge Conference Center, Park City, UT. Hosted by: EyeSki. Key faculty: Joseph Pizzimenti, Leonard Messner, Tom Arnold, Mile Brujic, James Fanelli. CE hours: 22. For more information, email Tim Kime at tandbkime@buckeye-express.com or go to www.eyeskiutah.com.

■ **7-11.** *Ocular Therapeutics in Cancun.* Fiesta Americana Condesa All Inclusive Resort, Cancun, Mexico. Hosted by: Ocular Therapeutics CE. Key faculty: Anthony Litwak, Diana Shechtman, James Thimons. CE hours: 20. For more information, email Anthony Litwak at info@otce.ne or go to www.otce.net.

■ **8-18.** *Tropical CE Australia 2018.* Sydney & Great Barrier Reef, Sydney & Cairns, Australia. Hosted by: Tropical CE. Key faculty: Eric Schmidt, Simon Chen. CE hours: 20. For more information, email Stuart Autry at sautry@tropicalce.com, call (281) 808-5763 or go to www.tropicalce.com.

■ **15-18.** *International Vision Expo & Conference East.* Jacob Javits Center, New York City. Hosted by: Reed Exhibitions and The Vision Council. Key faculty: Ben Gaddie, Mark Dunbar, Kirk Smick, Jack Schaeffer, Dave Ziegler, Douglas Devries. CE hours: 275 total, 30 per OD. For more information, go to east.visionexpo.com.

■ **24-25.** *Symposium on Ocular Disease.* Tysons Corner Marriott, Tysons Corner, VA. Hosted by: PSS EyeCare. Key faculty: Randall Thomas, Ron Melton, Elliot Kirstein, Damon Dierker, Willian Jones, Robert Rebello. CE hours: 20. For more information, email Sonia Kumari at education@psseyecare.com, call (203) 415-3087 or go to www.psseyecare.com.

April 2018

■ **5-7.** *OAOP Vision Summit.* Norman, OK. Hosted by: Oklahoma Association of Optometric Physicians. Key faculty: Justin Schweitzer, Nathan Lighthizer, Joseph Sowka. CE hours: 35 total, 20 per OD. For more information, email Heatherlyn Burton at heatherlyn@oaop.org or go to www.oaop.org.

■ **6-8.** *New Technologies & Treatments in Eye Care.* Nashville Marriott at Vanderbilt, Nashville. Hosted by: *Review of Optometry*. Key faculty: Paul Karpecki. CE hours: up to 19. For more information, email Lois DiDomenico at reviewmeetings@jobson.com, call (866) 658-1772 or go to www.reviewofoptometry.com/nashville2018.

■ **10-14.** *COVD 2018 Annual Meeting.* Hyatt Regency Bellevue, Bellevue, WA. Hosted by: College of Optometrists in Vision Development. CE hours: 28. For more information, email info@covd.org or call (330) 995-0718.

■ **18-21.** *CE in Italy/Europe.* Residence Le Santucce, Tuscany, Italy. Hosted by: James Fanelli. Key faculty: Joseph Pizzimenti, Lorraine Lombardi, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinitaly.com, call (910) 452-7225 or go to www.ceinItaly.com.

■ **19-21.** *MWCO Annual Congress.* Aria Resort & Casino, Las Vegas. Hosted by: Mountain West Council of Optometrists. Key faculty: Alison Bozung, John McGreal, Julie Rodman, Jessica Steen, Jim Thimons, Rob Wooldridge. CE hours: 56 total, 24 per OD. For more information, email Tracy Abel at mountainwestcouncil@gmail.com or go to www.mwco.org.

■ **25-29.** *16th Annual Educational Seminar.* Kingston Plantation, Myrtle Beach, SC. Hosted by: American Academy of Optometry-New Jersey Chapter. Key Faculty: Barry Eiden, Steven Ferrucci. CE hours: 16. For more information, email Dennis Lyons at dhl2020@aol.com or call (732) 920-0110.

■ **26-29.** *New Technologies & Treatments in Eye Care San Diego/OCCRS Joint Symposium.* San Diego Marriott Del Mar, San Diego. Hosted by: *Review of Optometry* & OCCRS. Key faculty: Paul Karpecki, David Friess. CE hours: up to 28. For more information, email Lois DiDomenico at reviewmeetings@jobson.com, call (866) 658-1772 or go to www.reviewofoptometry.com/sandiego2018.

■ **29-May 3.** *ARVO 2018.* Hawaii Convention Center, Honolulu, HI. Hosted by: Association for Research in Vision and Ophthalmology. For more information, go to www.arvo.org/annual_meeting/2018.

May 2018

■ **2-4.** *CE in Italy/Europe.* Hotel Torbräu, Munich, Germany. Hosted by: James Fanelli. Key faculty: Joseph Pizzimenti, Lorraine Lombardi, Leonard Messner, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinitaly.com, call (910) 452-7225 or go to www.ceinItaly.com.

■ **5-7**. *CE in Italy/Europe.* Kongresshaus, Heidelberg, Germany. Hosted by: James Fanelli. Key faculty: Joseph Pizzimenti, Lorraine Lombardi, Leonard Messner, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinitaly.com, call (910) 452-7225 or go to www.ceinitaly.com.

■ **17-20.** *New Technologies and Treatments in Eye Care Orlando 2018.* Disney's Yacht Club, Orlando, FL. Hosted by: *Review of Optometry.* Key faculty: Paul Karpecki. CE hours: 18. For more information, email Lois DiDomenico at reviewmeetings@jobson.com call (866) 658-1772 or go to www.reviewofoptometry.com/orlando2018.

■ **30.** *The Eleventh Central Jersey Optometric Seminar.* CentraState Medical Center, Freehold, NJ. Hosted by: Optometry on West 44th. Key Faculty: Walter Whitley. CE hours: 4. For more information, go to www.optometryonwest44th.com.

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Glaucoma's New Foe, Explained

How a recently approved formulation aims to improve pressure control.

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

A 73-year-old woman presented for her glaucoma progress evaluation. She had been diagnosed with primary open-angle glaucoma (OAG) four years earlier and started on latanoprost 0.005% in each eye, which she tolerated well. She was 20/25 OU and her vision was commensurate with her mild cataracts.

Her visual fields showed very mild loss OD and a moderately extensive inferior arcuate defect OS. Recent visual field analysis confirmed no changes in either eye. Central corneal thickness was 562µm OD and 568µm OS. Intraocular pressures (IOPs) were 19mm Hg OU. Her best IOP reduction from latanoprost was 15mm Hg OD and 17mm Hg OS.

To Treat or Not To Treat?

Today's reading was especially troubling in that her peak IOP was 22mm Hg OD and 23mm Hg OS.

Previously, it was felt the IOP reduction from latanoprost was insufficient, and she had been prescribed a beta-blocker, carbonic anhydrase inhibitor and an alpha adrenergic agonist. She had either local ocular or systemic adverse effects to each, necessitating discontinuation, leaving latanoprost as maximal tolerable medical therapy.

While her IOP rising into the upper teens was disconcerting given her low baseline untreated pressures, it was comforting to see that visual fields, optic discs and retinal nerve fiber layer were all stable. This left a conundrum: either monitor a seemingly unchanging patient with current therapy or go on to more invasive laser or surgical options based simply on suboptimal IOP reduction.

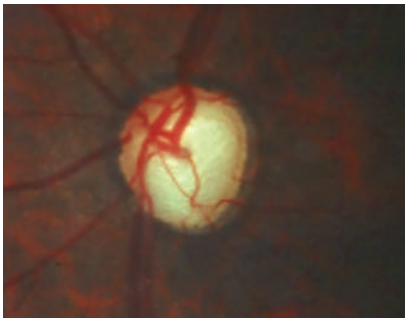
Recently, a new option for patients like ours, Vyzulta (latanoprostene bunod 0.024%, Bausch + Lomb and Nicox), was approved for the reduction of IOP in patients with OAG and ocular hypertension (OHTN).

Mechanism of Action

Vyzulta is a nitric oxide-donating prostaglandin analog designed to exploit both the uveoscleral aqueous outflow pathway (through the prostaglandin action) and the trabecular outflow channels (through the nitric oxide actions).[1] Until now, enhancing outflow through the trabecular pathway has been limited to the effects of laser trabeculoplasty and poorly tolerated miotics.

Vyzulta works by metabolizing into two moieties, latanoprost acid and butanediol mononitrate. The latter subsequently releases nitric oxide in human trabecular meshwork cells after application of latanoprostene bunod.[1] Nitric oxide has many physiological roles and is involved in inflammation, pain sensation, rheumatoid arthritis, immune system modulation and gastroprotection. It is also an antioxidant.[2]

Nitric oxide released from latanoprostene bunod elicits trabecular meshwork cell relaxation that, in turn, enhances aqueous outflow through the trabecular meshwork, Schlemm's canal and distal scleral vessels. It does so by inducing cytoskeletal relaxation via the soluble guanylyl cyclase-cyclic guanosine monophosphate (sGC-cGMP) signaling pathway.[3] Targeting the conventional outflow tissues using nitric oxide-donating drugs represents an opportunity to restore trabecular outflow, a medically untapped area in glaucoma management with the added benefit of promoting a healthy trabecular meshwork.[1,4]



Glaucomatous optic nerves such as this one now have a new potential lifeline.

Galaxy Quest

Several pivotal trials show Vyzulta's efficacy, including the VOYAGER and CONSTELLATION studies, which compared Vyzulta with latanoprost and timolol, respectively.[5,6] VOYAGER concludes once-daily dosing of Vyzulta provided IOP reductions greater than those from latanoprost.[5] In CONSTELLATION, both Vyzulta and timolol decreased IOP, but Vyzulta produced a greater mean reduction in nighttime IOP and improved diurnal ocular perfusion pressure compared with both baseline and timolol.[6]

The APOLLO study—which includes OAG and OHTN patients with baseline IOP between 25mm Hg and 36mm Hg—revealed that mean IOP with Vyzulta was significantly lower compared with timolol at all time points, a finding maintained during the open-label phase and confirmed in the crossover group from timolol to Vyzulta.[7]

The LUNAR study found mean IOP with Vyzulta was significantly lower compared with timolol at all points except at the 8am week-two point. IOP lowering was maintained during the open-label treatment and confirmed in the crossover group.[8]

The JUPITER study showed IOP decrease of 22% that lasted for a year (in a low baseline IOP group).[9]

A study involving 24 healthy, normotensive Japanese male volunteers evaluated the effect of Vyzulta instilled once daily.[10] Following 14 days of treatment, investigators saw a mean pressure reduction of 27% from baseline over the 24-hour diurnal.[10] The authors concluded that studies of Vyzulta in patients diagnosed with normal tension glaucoma are warranted.[10] Safety and tolerability has been established with no serious adverse events reported.[11] The most common adverse event noted was transient hyperemia, which is largely well tolerated.[11]

With its dual mechanism of action, once-daily dosing and favorable efficacy and side effect profiles, Vyzulta gives optometrists and our patients cause to be hopeful. ■

Dr. Sowka is on Bausch + Lomb's Vyzulta advisory board.

1. Cavet M, DeCory H. The role of nitric oxide in the intraocular pressure lowering efficacy of latanoprostene bunod: review of nonclinical studies. J Ocul Pharmacol Ther. 2017 Aug 7. [Epub ahead of print].
2. Kumar S, Singh RK, Bhardwaj TR. Therapeutic role of nitric oxide as emerging molecule. Biomed Pharmacother. 2017 Jan;85:182-201.
3. Kaufman PL. Latanoprostene bunod ophthalmic solution 0.024% for IOP lowering in glaucoma and ocular hypertension. Expert Opin Pharmacother. 2017 Mar;18(4):433-44.
4. Aliancy J, Stamer WD, Wirostko B. A review of nitric oxide for the treatment of glaucomatous disease. Ophthalmol Ther. 2017 Dec;6(2):221-32.
5. Weinreb RN, Ong T, Sforzolini S, et al. A randomised, controlled comparison of latanoprostene bunod and latanoprost 0.005% in the treatment of ocular hypertension and open angle glaucoma: the VOYAGER study. Br J Ophthalmol. 2015;99(6):738-45.
6. Liu JH, Slight JR, Vittitow JL, et al. Efficacy of latanoprostene bunod 0.024% compared with timolol 0.5% in lowering intraocular pressure over 24 hours. Am J Ophthalmol. 2016;169:249-57.
7. Weinreb RN, Sforzolini S, Vittitow J, Liebmann J. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: The APOLLO study. Ophthalmology. 2016;123(5):965-73.
8. Medeiros FA, Martin KR, Peace J, et al. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. Am J Ophthalmol. 2016;168:250-59.
9. Kawase K, Vittitow JL, Weinreb RN, et al. Long-term safety and efficacy of latanoprostene bunod 0.024% in Japanese subjects with open-angle glaucoma or ocular hypertension: the JUPITER study. Adv Ther. 2016;33(9):1612-27.
10. Araie M, Sforzolini BS, Vittitow J, Weinreb RN. Evaluation of the effect of latanoprostene bunod ophthalmic solution, 0.024% In lowering intraocular pressure over 24 h in healthy Japanese subjects. Adv Ther. 2015 Nov;32(11):1128-39.
11. Garcia GA, Ngai P, Mosaed S, Lin KY. Critical evaluation of latanoprostene bunod in the treatment of glaucoma. Clin Ophthalmol. 2016 Oct 18;10:2035-50.



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Questions may be directed to:
Julie DeKinder, OD (Director, Academic Programs)
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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**
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- Participating in leadership roles in state, regional, and national optometry organizations;
- Participating on College and University committees, as assigned;
- Participating in College and University service activities.

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 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 optometric state license in the state in which the college is located. Primary eye care clinical expertise is also required.

Contact information: 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. Inquiries may be directed to Dr. Melissa Suckow, Dean; Midwestern University: msucko@midwestern.edu.

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Diagnostic **Quiz**

She's on a Losing Streak

By Andrew S. Gurwood, OD

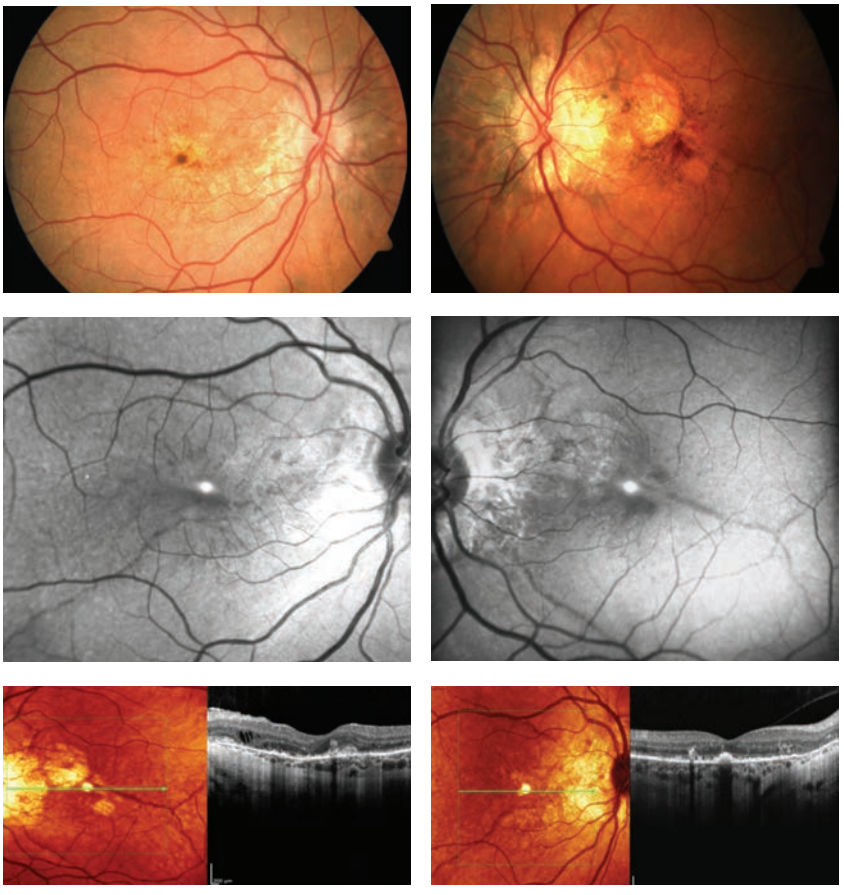
History

A 52-year-old white female presented to the office with a chief complaint of blurry vision in both eyes at distance and near. She reported that the right eye had been blurry for months and the left eye became blurred one month prior to this appointment. She reported no use of ocular medications; her systemic medications consisted of bisacodyl for constipation. She reported no known allergies to medications or anything else.

Diagnostic Data

Her best-corrected entering visual acuities were 20/80 OD and 20/150 OS. Her pupils were round, equal in size and reactive to light without an afferent pupillary defect. Extraocular muscles were full and confrontation visual fields were full to finger counting with a central blur OU when performing the "facial" Amsler. Refraction was completed with no improvement at either distance or near. Biomicroscopy was remarkable only for trace nuclear sclerosis in both eyes.

Her intraocular pressures using Goldmann applanation tonometry measured 13mm Hg OD and 12mm Hg.



A 52-year-old woman complained of progressive blurriness in both eyes. What can these images and her history combined tell you about her possible diagnosis?

Your Diagnosis

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

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

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Redefining vision by fusing science, art and technology designed to achieve revolutionary solutions for presbyopia, myopia and astigmatism.

NaturalVue® (etafilcon A) Multifocal 1 Day Contact Lenses

- Innovative Neurofocus Optics® Technology inspired by advanced camera optics
- One Universal Extended Depth of Focus center-distance design
- Fit like a Single Vision Sphere

See Naturally®

Call us today at **1-844-VTI-LENS**
(1-844-884-5367, ext. 116)
or visit **vtivision.com**

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AIR OPTIX® PLUS HYDRAGLYDE® CONTACT LENSES

2 UNIQUE TECHNOLOGIES 1 OUTSTANDING LENS

SMARTSHIELD® TECHNOLOGY **EXCELLENT DEPOSIT PROTECTION[1,2]** + HYDRAGLYDE® MOISTURE MATRIX **LASTING LENS SURFACE MOISTURE[3,4]**

FOR A LIMITED TIME, NEW WEARERS CAN

SAVE UP TO $100

ON AN ANNUAL SUPPLY VIA MAIL-IN REBATE*
WITH THE AIR OPTIX® CHOICE PROGRAM!

Visit AIROPTIXCHOICE.com to learn more

PERFORMANCE DRIVEN BY SCIENCE®

*Rebate is in the form of an Alcon VISA® Prepaid Card. Certain criteria must be met to be eligible for the full rebate. Must be a new patient to the AIR OPTIX® family of contact lenses or an existing patient that is switching lenses within the AIR OPTIX® family. Must purchase an annual supply (four 6-ct boxes) of AIR OPTIX® brand contact lenses (excluding AIR OPTIX® AQUA lenses) within 90 days of eye exam or contact lens fitting. Rebate submission must be postmarked (or submitted electronically) within 60 days of lens purchase date. Valid on purchases made at participating retailers through 12-31-17. Visit AIROPTIXCHOICE.com for complete terms and conditions.

Important information for AIR OPTIX® plus HydraGlyde® (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness. Risk of serious eye problems (i.e. corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

References: 1. Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci.* 2010;87:E-abstract 105110. **2.** Nash WL, Gabriel MM. Ex vivo analysis of cholesterol deposition for commercially available silicone hydrogel contact lenses using a fluorometric enzymatic assay. *Eye Contact Lens.* 2014;40(5):277-282. **3.** *In vitro* study over 16 hours to measure wetting substantivity; Alcon data on file, 2015. **4.** *In vitro* wetting analysis: out-of-pack and wetting substantivity; Alcon data on file, 2014.

Alcon A Novartis Division

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

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Rx only