Optometric Study Center — **Ocular Health: A Matter of the Heart,** P. 122

REVIEW® OF OPTOMETRY

October 15, 2015

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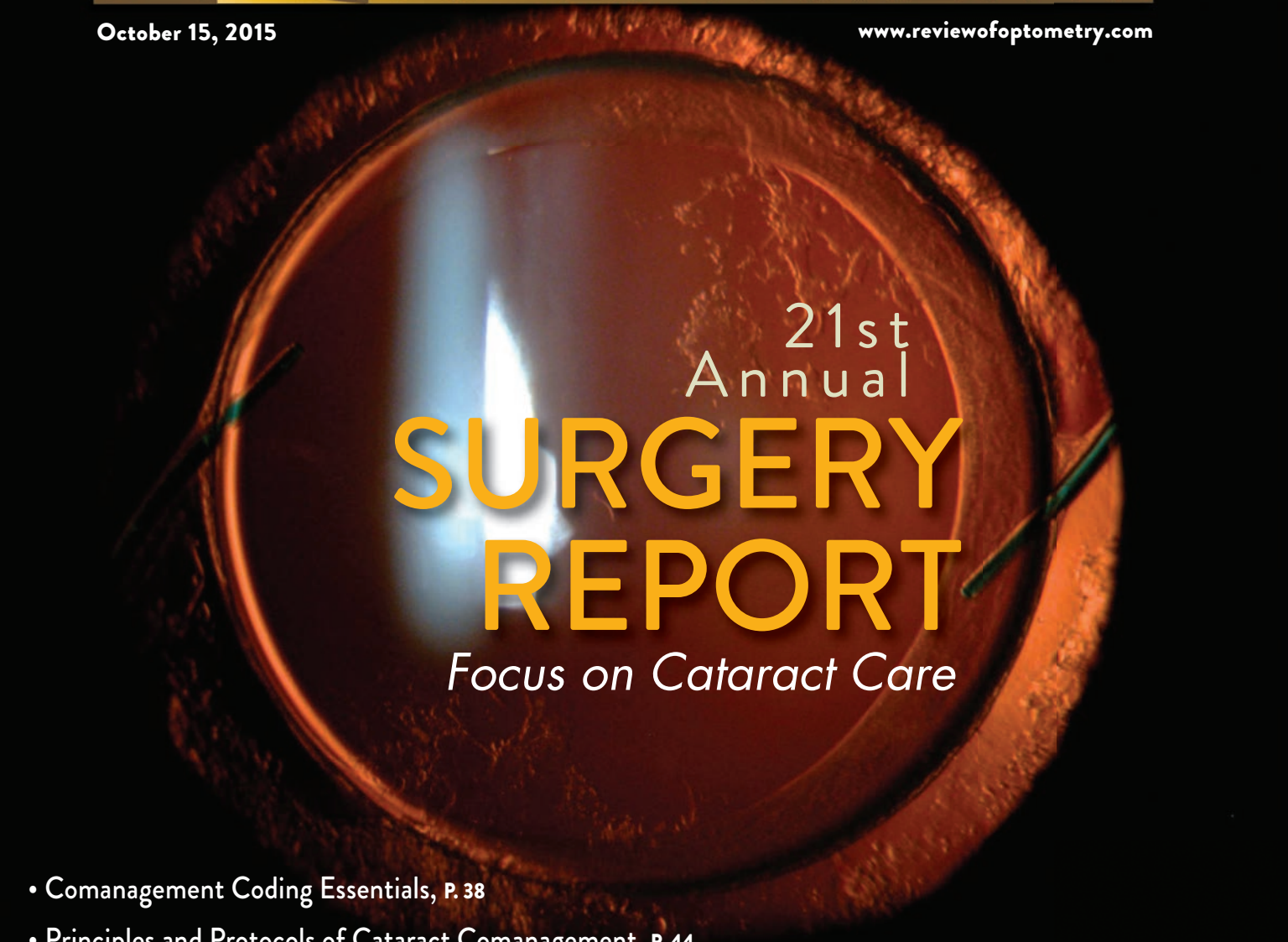

21st Annual SURGERY REPORT

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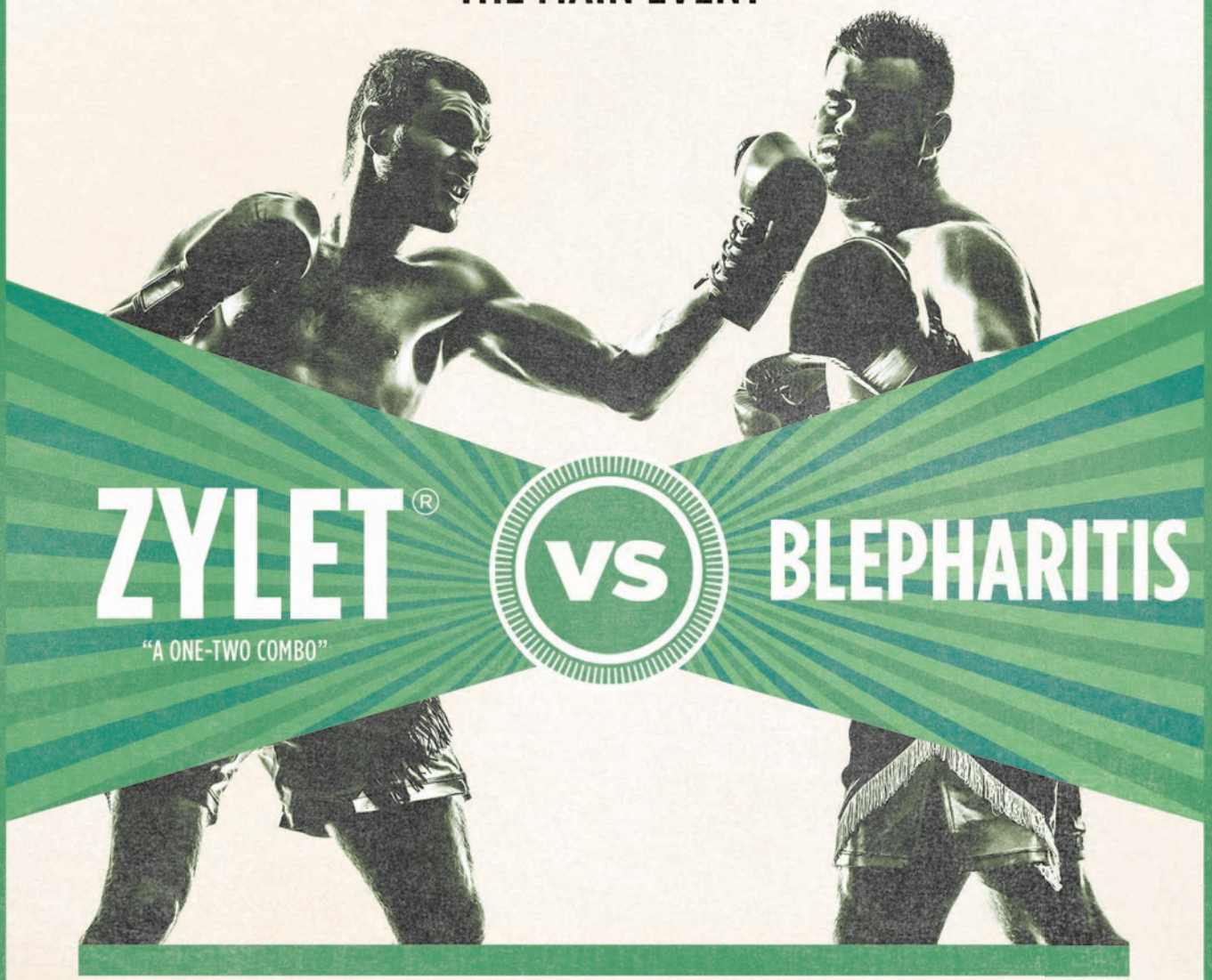

HELP PUT RELIEF IN YOUR CORNER

INDICATIONS AND USAGE

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

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

INDICATIONS AND USAGE (continued)

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

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

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

IMPORTANT SAFETY INFORMATION

- ZYLET® is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.
- Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. Steroids should be used with caution in the presence of glaucoma. If this product is used for 10 days or longer, intraocular pressure should be monitored.
- Use of corticosteroids may result in posterior subcapsular cataract formation.
- The use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification such as a slit lamp biomicroscopy and, where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infections. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.
- Employment of corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.
- Most common adverse reactions reported in patients were injection and superficial punctate keratitis, increased intraocular pressure, burning and stinging upon instillation.

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

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

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

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

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

Initial U.S. Approval: 2004

DOSAGE AND ADMINISTRATION

2.1 Recommended Dosing

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

2.2 Prescription Guideline

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

CONTRAINDICATIONS

4.1 Nonbacterial Etiology

Zylet, as with other steroid anti-infective ophthalmic combination drugs, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

5.1 Intraocular Pressure (IOP) Increase

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

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

5.2 Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

5.3 Delayed Healing

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

5.4 Bacterial Infections

Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.

5.5 Viral Infections

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

5.6 Fungal Infections

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

5.7 Aminoglycoside Hypersensitivity

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

ADVERSE REACTIONS

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

Zylet:

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

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

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

Loteprednol etabonate ophthalmic suspension 0.2% - 0.5%:

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

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

Tobramycin ophthalmic solution 0.3%:

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

Secondary Infection:

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

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

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

USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

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

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

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

8.3 Nursing Mothers

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

8.4 Pediatric Use

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

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

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

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

8.5 Geriatric Use

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

NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

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

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

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

PATIENT COUNSELING INFORMATION

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

MANUFACTURER INFORMATION

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VOL. 152 NO. 10 ■ OCTOBER 15, 2015

IN THE NEWS

Investigators at the Schepens Eye Research Institute of Mass. Eye and Ear have found that any **binocular central field loss** could delay a driver's ability to detect moving hazards in time to take safe, corrective action. To make this discovery, they studied **participants with AMD** using a state-of-the-art driving simulator in an effort to better understand the effects of vision loss on driving. The results were published in two phases, first in March 2013 in *JAMA Ophthalmology* and second on September 2, 2015 in *PLOS ONE*.

A new study, published online in *JAMA Ophthalmology*, found that women who are **vitamin D deficient** and have a specific high-risk genotype are **6.7 times more likely to develop age-related macular degeneration (AMD)** than women without the genotype and who had sufficient levels of vitamin D. Researchers at the University at Buffalo's School of Public Health and Health Professions made the discovery after studying 1,230 women ages 54 to 74 who participated in the **Carotenoids in Age-related Eye Disease Study**.

Researchers at the University of California, San Diego School of Medicine have discovered a genetic interaction that may be integral to the development and progression of **glaucoma**. The findings, published online in *Molecular Cell*, suggest that some **variants of a gene known as SIX6** boost expression of another gene, p16INK4a, which in turn **accelerates senescence and death of retinal ganglion cells**. Investigators hope this could one day lead to a new therapeutic approach for primary open-angle glaucoma.

Lesions Linked with Retinal Non-Perfusion

New research highlights the importance of the peripheral retina in diabetic retinopathy progression.

By Rebecca Hepp, Senior Associate Editor

After discovering, earlier this year, that the presence of peripheral lesions increased the risk of rapid diabetic retinopathy progression, researchers from the Joslin Diabetes Center's Beetham Eye Institute further discovered that these lesions also closely correlate with retinal non-perfusion caused by loss of small blood vessels or capillaries.

This follow up study, published recently in *Ophthalmology*, used ultra-widefield (UWF) imaging to examine the eyes of 37 patients with diabetes and varying levels of retinopathy. They found that the areas of non-perfusion, identified by UWF retinal angiography, matched up closely with the peripheral lesions identified with normal UWF imaging.

The researchers hope this discovery could one day allow clinicians to use peripheral lesions to estimate the extent and location of non-perfusion damage, as well as the risk of disease progression, without having to use UWF angiography in every case.

A related trial by the Diabetic Retinopathy Clinical Research Network is now underway, and if it confirms this study's findings, the Joslin researchers suspect the diabetic eye disease grading system will be updated to reflect these risk factors and imaging approaches, Lloyd Paul Aiello, MD, PhD, director of the Beetham Institute, professor of ophthalmology at Harvard Medical School and senior author on the paper, said in a press release.

Photo: Paul A. Chous, OD.

The study findings mechanistically tie predominantly peripheral lesions to the development of proliferative diabetic retinopathy, Dr. Chous says.

"The bottom line is that we cannot solely focus on the posterior pole in diabetes, but must pay close attention to the peripheral retina because abnormalities there substantially increase patient risk of blinding eye disease," says Paul A. Chous, OD, who has a private practice in Tacoma, Wash.

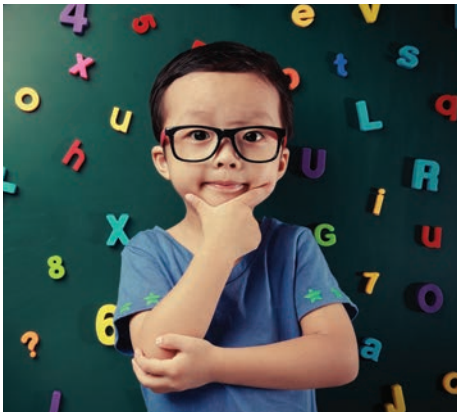
Silva PS, Dela Cruz AJ, Ledesma MG, et al. Diabetic retinopathy severity and peripheral lesions are associated with nonperfusion on ultrawide field angiography. Ophthalmology. 2015 Sep:pii:S0161-6420(15)00773-3. [Epub ahead of print].

Reading, Genes Increase Risk of Myopia in Children

Researchers at the Columbia University Medical Center have found that an infrequently occurring variation in the APLP2 gene causes susceptibility to myopia in children who read for more than 60 minutes per day.

The study, published in *PLOS Genetics*, suggests that those carrying an alternate form of the APLP2 gene are more likely to develop the condition, and that the condition develops between the ages of eight and 15. Importantly, the cohort study found that those who read for more than an hour were five times more likely to suffer from myopia.

Using data collected from 13,988 pediatric subjects, researchers evaluated the effect of the APLP2 phenotype on refractive error over an average of 15.5 years. Refractive error was evaluated by autorefraction every three



years between the ages of approximately seven and 15. Statistical analysis predicted that those who carried the gene and self-identified as having read for lengthier periods of time would be more myopic than the group who self-identified as having read less. According to Andre Tkatchenko, MD, PhD, first author of the study, this is the first evidence showing the relationship between genetics and behavioral factors in causing a change in refractive error.

Hypothetically, reducing the gene in the eye during childhood would be an effective treatment, Dr. Tkatchenko suggests in a press release. However, engaging in environmental behaviors favorable to reducing myopia during the ages of elementary and middle school is the only practical measure for ensuring the refractive health of the eye.

Jeffrey J. Walline, PhD, OD, associate dean of research at the Ohio State University, agrees. "I recommend that parents encourage outdoor activities to decrease the likelihood of myopia onset," he says, adding that the best evidence for slowing myopia progression comes from soft bifocal or corneal reshaping CL wear.

Tkatchenko AV, Tkatchenko TV, Guggenheim JA, et al. APLP2 regulates refractive error and myopia development in mice and humans. PLoS Genet. 2015 Aug;(11)8. [Epub].

Retina Offers Insights into Schizophrenia Pathophysiology

A meta-analysis, published in *Schizophrenia Research: Cognition*, suggests that retinal changes may precede the behavioral onset of schizophrenia.

Researchers from Rutgers University and Mount Sinai's New York Eye and Ear Infirmary reviewed the current evidence on visual disturbances in schizophrenia and concluded that if further research links certain retinal changes to the progression of schizophrenia, doctors could better diagnose and manage schizophrenia in high-risk patients who have not yet presented with the behavioral component of the disease. For example, reduced b-wave amplitude in electroretinography (ERG) readings were found in high-risk pediatric patients who did not display behavioral symptoms of schizophrenia.

For patients who are already being treated for the disease, the state of the retina would be an indicator of the need for additional or new medications to prevent the

(continued on pg. 8)

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The Retina in Schizophrenia

(continued from pg. 6)
onset of psychotic episodes. Differences have been noted in A-wave flash ERG readings before and after hospitalizations for acute bouts of psychosis; reduction in A-wave amplitude existed before hospitalization and treatment for a psychotic episode, but not after.

"Measurement of retinal changes may help doctors, in the future, adjust schizophrenia treatment for each patient," study co-author Richard B. Rosen, MD, says in a press release.

Another important retinal feature noted in the meta-study was retinal nerve fiber layer (RNFL) thinning, which may have diagnostic implications for ODs.

"Some, but not all, studies indicate thinning of the retinal nerve fiber layer in schizophrenia. Evidence from other neuropsychiatric populations suggests that thinning parallels loss of brain volume, cognitive decline and illness progression in these conditions," says Steven Silverstein, PhD, the study's first author. "It is not yet known the extent to which retinal nerve fiber layer thinning in schizophrenia parallels any such changes; however, if it is later found that RNFL thinning does predict these changes, this would have several implications," Dr. Silverstein says.

First, he recommends an OCT exam. Second, if the exam indicates RNFL thinning, order a more expensive and time-consuming structural brain scan, as well as neuropsychological testing and functional assessment.

"If these tests indicate brain volume loss, cognitive decline or both, this would suggest a need for cognition-enhancing medication and for cognitive remediation, which has been shown to improve cognition and slow progressive grey matter loss in younger schizophrenia patients," Dr. Silverstein says.

Silverstein SM, Rosen R. Schizophrenia and the eye. Schizophrenia Research: Cognition. 2015 June;2(2):46-55.

Australian Vision Therapy Study Makes a Media Splash

Making its way into mainstream consciousness recently is a topic that's been hotly debated in the eye care industry for years: vision training. Now, a new report on its effects, published in the journal *Current Biology*, has the optometry community talking once again. The report suggests that the physiological blind spot, a zone of functional blindness all sighted people have, "can be shrunk through training to distinguish direction signals at the blind spot periphery."

The study, which comes from the University of Queensland in Australia, reports "training on 20 successive weekdays improved sensitivity to both direction and color, suggesting a generalizable benefit."

Using a computer and an eye patch, the researchers showed 10 patients a waveform in the visual field of their blind spot day after day. After the 20 days in a row, the participants' blind spots shrunk by about 10%, the *New York Times* reports.

"Through what the authors are calling perceptual learning, they were able to demonstrate an impact on the functional size of the blind spot," says Marc Taub, OD, chief of Vision Therapy and Rehabilitation at The Eye Center at Southern College of Optometry, who was not involved in the research. "In this study, perceptual learning refers to the distinguishing of direction signals at the periphery of the blind spot."

Although it's a small, controlled study, it demonstrates the concept of neural plasticity, he says.

"This has implications in the world of traditional vision therapy for conditions such as amblyopia, strabismus and the visual consequences of acquired brain injury," Dr. Taub says.

The researchers consider their study the first step toward a possible future treatment for vision-threatening diseases such as macular degeneration. ■

Miller PA, Wallis G, Bex PJ, Arnold DH. Reducing the size of the human physiological blind spot through training. Current Biology. 2015 August;25(17):pR747–R748.

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SCHEDULE

7:00am - 7:55am	Registration & Breakfast
7:55am - 8:00am	Welcome and Introduction
8:00am - 10:00am	Cornea/Refractive Section I
10:00am - 11:00am	Cornea/Refractive Section II
11:00am - 11:15am	Break
11:15am - 12:15pm	Cornea/Refractive Section III
12:15pm - 1:15pm	Lunch
1:15pm - 3:15pm	Cataract Section I
3:15pm - 3:30pm	Break
3:30pm - 4:30pm	Cataract Section II
4:30pm - 5:30pm	Drugs, Devices, and Treatments Section I

- The Optometric Council on Refractive Technology will sponsor it's 12th annual education symposium, bringing together the most notable experts in the field of cornea, cataract, and refractive technology to discuss evolving clinical innovations and management of ocular surface disease and other anterior segment complications
- The meeting is held the day before the American Academy of Optometry meeting in New Orleans, LA. This interactive meeting encourages questions, comments, and audience participation with panel discussions.
- Up to eight hours of COPE CE* will be awarded to attendees. Registration fee includes a continental breakfast and gourmet lunch.
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For more information, contact Clark Chang, OD at cchang@vision-institute.com
or Chris Freeman, OD at jcfreeopt@gmail.com or visit www.ocrt.org

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*Encuity's *Treatment Answers*, based on frequency of Dry Eye product recommendations. Jan 2013 – Dec 2013. APC02PV15

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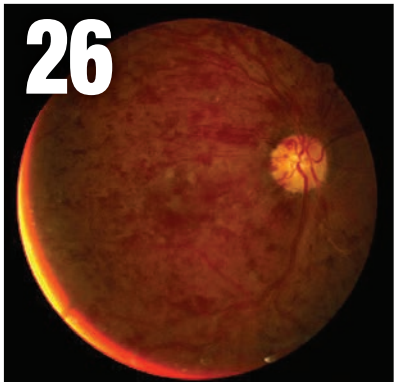

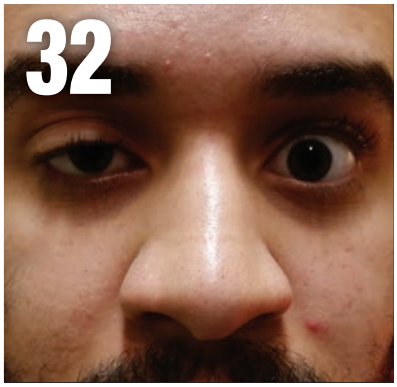

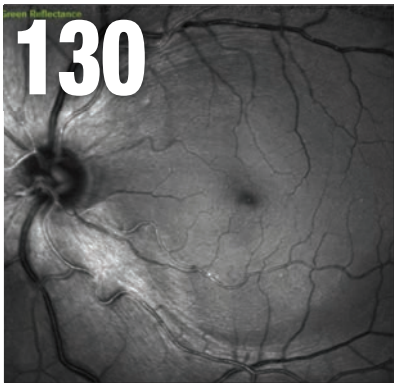

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REVIEW OF OPTOMETRY®

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ZeaVision's comprehensive EyePromise portfolio is an ideal choice for medically-focused eye care professionals.

As an innovator ZeaVision has developed a comprehensive portfolio of evidence-based ocular nutrition products to fight vision loss and preserve healthy vision.

Dennis Gierhart, PhD and co-founder of ZeaVision, began researching the diet-derived carotenoid, zeaxanthin, in the early 1990's upon learning that zeaxanthin was found naturally in very high concentrations within the macula. Dr. Gierhart hypothesized that zeaxanthin must be critical to eye health given its unique biological position and location. Dr. Gierhart then developed the first commercially available zeaxanthin and demonstrated one could increase serum and macular levels with supplementation.

The scientific community has since come to appreciate the benefits of both zeaxanthin and lutein (macular pigments) as important diet-derived antioxidants and photo-protectants. It is now understood that sub-optimal levels of macular pigment protection can affect one's risk of developing Age-related Macular Degeneration (AMD). Scientific research has also demonstrated that one's density of macular pigment can affect visual function. Dense macular pigment can decrease photophobia, shorten glare recovery time, improve contrast sensitivity, color vision, and increase visual processing speed.

COMPREHENSIVE EYEPROMISE PRODUCT LINE

Since the company's inception, the EyePromise product portfolio has expanded to serve patients' nutritional needs. With more than 50-million doctor-recommended doses consumed and counting, the product line now includes eye vitamins for AMD, diabetes vision support, dry eye, and optimizing visual performance.

EyePromise brands are evidence based and underpinned by extensive clinical research validating their performance.

Numerous clinical research studies have been conducted with EyePromise brands by leading eye care professionals and published in peer-reviewed journals. Please contact ZeaVision to obtain copies of the:

- Ocular Nutrition Impact on Tear Film Study (ONIT)
- Diabetes Visual Function Supplement Study (DiVFuSS)
- Double-Blind, Placebo-Controlled Study on the Effects of Lutein and Zeaxanthin on Neural Processing Speed and Efficiency
- Preliminary Results in Macular Pigment Optical Density Associated with and without Zeaxanthin and Lutein Supplementation

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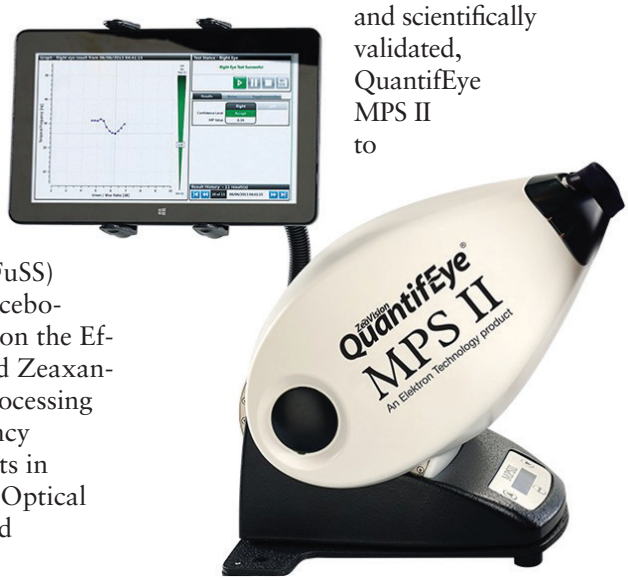
The natural ingredients within each EyePromise formula are of the highest quality. EyePromise macular health formulas contain dietary zeaxanthin derived from a unique natural botanical source; orange paprika peppers grown in the USA. EyePromise macular health formulas are also supported by a money-back guarantee to increase MPOD. The EyePromise Dry Eye formula, EZ Tears, is also supported by a money-back guarantee to improve dry eye symptoms within 30 days. Both guarantees are unparalleled in the eye vitamin industry.

QUANTIFEYE MPS II INSTRUMENTATION

Zeavision provides eye care practitioners the market leading and scientifically validated, QuantifEye MPS II to



The QuantifEye MPS II allows eye care professionals to assess a key risk factor for AMD.

measure macular pigment optical density (MPOD) in a fast, simple, and non-invasive manner.

The QuantifEye MPS II instrument allows eye care professionals to assess a key risk- factor for Age-related Macular Degeneration, as well as monitor supplement compliance and macular protection progress. The instrument also helps the practitioner identify patients who would best benefit from EyePromise supplementation to improve visual function.

With more than five million eyes accurately measured, the QuantifEye MPS II instrument:

- Is scientifically validated in numerous clinical studies
- Is accurate and repeatable
- Is fast and easy to operate
- Creates a significant revenue stream for the eye care practice

Eye care professionals are provided a 60-day, free evaluation of the instrument in their practice, including on-site staff training by a highly skilled ZeaVision Implementation Specialist.

PATIENT AND STAFF EDUCATIONAL MATERIALS - THE EYE CARE SUPPORT PROGRAM™ (ESP)

ZeaVision's unique EyeCare Support Program was designed to assist eye care professionals in the successful implementation of ocular nutrition practices. With the support of its clinical advisors,

EyePromise offers a full line of products to serve patients with varying dietary needs.

ZeaVision has also developed an ample portfolio of ocular nutritional education materials to support eye care professionals and patients. These materials are available in multiple formats including print, video, and audio. Many of the tools reside in a unique, on-line education portal available to ZeaVision customers free of charge. The program includes:

- Staff, doctor, and patient education as well as training materials
- A comprehensive patient compliance program
- Practice marketing support materials
- Staff to support in-practice training

THE EYE-Q INTELLIGENT AMD RISK MANAGEMENT PROGRAM®

The combination of EyePromise, QuantifEye MPS II, and the EyeCare Support Program is marketed as the Eye-Q Intelligent AMD Risk Management Program. This turn-key program is unparalleled, providing all of the products as well as support materials required to successfully implement and run a successful ocular nutrition component in an eye care practice.

EYEPROMISE REWARDS PROGRAM

ZeaVision also rewards EyePromise customers with a unique, points based incentive program that enables eye care professionals to earn valuable rewards. The many reward options include product discounts, gift cards, EyePromise apparel and staff luncheons. Program points are earned on EyePromise purchases, education program participation, and use of ZeaVision education tools.

Doctors interested in EyePromise, QuantifEye MPS II, The Eyecare Support Program, or the EyePromise Rewards Program should contact ZeaVision to learn more about this exciting industry-leading program via the information below.

ZeaVision Customer Support:
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Letters to the **Editor**

Glaucoma Care Belongs in Optometry's Wheelhouse

Glaucoma is now an unavoidable subject for ODs—who, today, shoulder the burden of 85% of all comprehensive eye care services in the United States, putting us at the forefront of glaucoma detection.[1-3]

With the country's population aging and the expected increase in rates of glaucoma in the next decade, it is important we embrace this role.[4,5]

More than two million prescriptions for prostaglandin analog eye drops are written by optometrists, proving that the profession is involved in managing these patients—and studies show that allowing optometrists to prescribe medication has a positive impact on patient outcomes.[6,7]

I believe it is time for optometry to become the pillar in the management of glaucoma. With studies such as the OHTS, AGIS, EMGT and CIGTS providing guidelines, glaucoma special interest groups and continuing education, never before have doctors seeking guidance had so much access to knowledge.[8-16]

With the expected strain that the aging population will place on eye care and the expected increase in glaucoma prevalence, optometry may have no choice but to get involved.

Our technology has improved too. We can now employ objective and repeatable tests, such as evoked potentials like ERGs and VEPs, ocular imaging techniques such as tomography and standardized methods to test field loss.

Today, we have a much clearer "north" to our clinical compass as we seek to manage this condition. Additionally, with the expected strain that the aging population will place on eye care and the expected increase in glaucoma prevalence, optometry may have no choice but to get involved in glaucoma management. We have the tools and the knowledge, now let's get to work.

—Agustin Gonzalez, OD
Richardson, Texas

1.Gonzalez A, Lakhani R, Bennett N, De Paz C. A twelve quarter quantitative analysis of ophthalmic medication prescribing by optometry in the United States. Clinical Optometry. 2014;6:5-10.
2. Gonzalez A. Quantitative analysis of ophthalmic drug prescription writing in optometry. Presented at AAO San Francisco. 2010.
3. The American Optometric Association. An action-oriented analysis of the state of the optometric profession. 2013.
4. World Health Organization. Glaucoma is second leading cause of blindness globally. www.who.int/bulletin/volumes/82/11/feature1104/en/. Accessed Feb 28, 2015.
5. Tielsch J, Sommer A, Katz J, et al. Racial variations in the prevalence of primary open-angle glaucoma: the Baltimore eye survey. JAMA. 1991;266(3):369-74.
6. Gonzalez A. A 3 year qualitative analysis of glaucoma ophthalmic prescription writing by optometrist. Presented at AAO Denver. 2012.
7. Azuara-Blanco A, Burr J, Thomas R, et al. The accuracy of accredited glaucoma optometrists in the diagnosis and treatment recommendation for glaucoma. Br J Ophthalmol. 2007;91(12):1639-43.
8. Kass M, Heuer D, Higginbotham E, 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.
9. Musch D, Lichter P, Guire K, et al. The collaborative initial glaucoma treatment study—study design, methods, and baseline characteristics of enrolled patients. Ophthalmology. 1999;106:653-62.
10. Lichter P, Musch D, Gillespie B, et al; the CIGTS Study Group. Interim clinical outcomes in the collaborative initial glaucoma treatment study comparing initial treatment randomized to medications or surgery. Ophthalmology. 2001;108:1943-53.
11. Heijl A, Leske M, Bengtsson B, et al. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002 Oct;120(10):1268-79.
12. Grødum K, Heijl A, Bengtsson B. Risk of glaucoma in ocular hypertension with and without pseudoexfoliation. Ophthalmology. 2005 Mar;112(3):386-90.
13. Anderson D. Collaborative normal tension glaucoma study. Curr Opin Ophthalmol. 2003;14(2):86-90.
14. Ederer F, Gaasterland D, Sullivan E. The Advanced Glaucoma Intervention Study (AGIS): 1. Study design and methods and baseline characteristics of study patients. Control Clin Trials. 1994;15(4):299-325.
15. Gedde S, Schiffman J, Feuer W, et al. Three-year follow-up of the tube versus trabeculectomy study. Am J Ophthalmol. 2009;148(5):670-84.
16. Minckler D, Francis B, Hodapp E, et al. Aqueous shunts in glaucoma: a report by the American Academy of Ophthalmology. Ophthalmology. 2008;115(6):1089-98.

Sight **Gags** By Scott Lee, OD

For the 75% of dry eye patients worldwide with evaporative dry eye (MGD) symptoms[1]...

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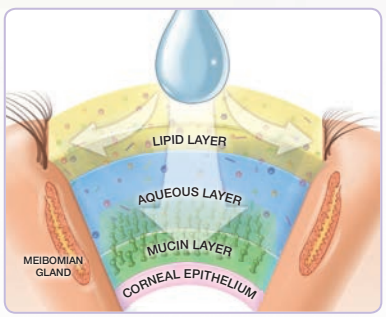

SYSTANE® BALANCE Lubricant Eye Drops forms a protective matrix that is designed to replenish the lipid layer for long-lasting relief from the symptoms associated with evaporative dry eye (MGD). This unique formulation is designed to work on all 3 layers of the tear film, specifically increasing LLT. This helps create a protective environment for the ocular surface.[2]

SYSTANE® Brand products are formulated for the temporary relief of burning and irritation due to dryness of the eye.

References: 1. Akpek EK, Smith RA. Overview of age-related ocular conditions. *Am J Manag Care.* 2013;19 (5 suppl):S67-S75. **2.** Korb DR, Blackie CA, Meadows DL, Christensen M, Tudor M. Evaluation of extended tear stability by two emulsion based artificial tears. Poster presented at: 6th International Conference on the Tear Film and Ocular Surface: Basic Science and Clinical Relevance; September 22-25, 2010; Florence, Italy.

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Out of Step

Health tracking technology holds great promise, but the abuse potential is just as great. **By Jack Persico, Editor-in-Chief**

It sounds almost like a headline from *The Onion*: "Swiss Insurance Company Will Charge Higher Premiums for Lazy People." But, no, that's from *Popular Science* (reporting, ironically, on developments likely to be considered neither popular nor science). Posted online in mid-September, the article recounts a story from Swiss news website *The Local* about a big insurer there called CSS that's looking into new data-driven ways to control costs. Its recent test program, called MyStep, uses fitness trackers to record the daily step count of CSS customers. Those who hit the company's target of 10,000 steps per day qualify for discounts on their insurance premiums.

There's nothing wrong with that, in principle. Insurance discounts for healthy lifestyle choices have been routine for years. It's a great way to align incentives with desirable outcomes. But this particular company is already hoping it can be mandatory rather than voluntary. The results of the test "should reveal whether and how insurance companies can introduce an appropriate offer tailored to customers' needs," Volker Schmidt of CSS told *The Local*, in a deft bit of corporate-speak. It's not hard to see what he's driving at: higher rates. "Given the increased cost of healthcare," he said, "we will inevitably have to promote individual responsibility in order to strengthen solidarity between insured people." Err, solidarity?

Such a program would likely benefit able-bodied people. But what about those with asthma or COPD who can't meet the mandatory daily step requirement? Or those lacking the resources to buy a pricey fitness tracker? Or someone whose job or family demands are such that daily exercise at that level and of that type just doesn't fit? I fail to see the solidarity any of that would create. Just the opposite, probably.

With a final flourish of tone-deaf language, Mr. Schmidt concluded, "Eventually we will be implanted with a nano-chip which will constantly monitor us and transmit the data to a control centre." What could possibly go wrong?

Health tracking technology offers abundant opportunities to improve the world. Apple recently showed off an app that will continuously monitor fetal heart rates for pregnant women who wear an Apple Watch. Data sent to the ob-gyn will reveal potential problems immediately. Novartis and Google's "smart" contact lens to continuously monitor blood glucose levels was just greenlighted for human trials. But no one, regardless of political persuasion, wants to be coerced into having an implanted chip send personal health data *to an insurer*. A doctor, maybe. Oneself, sure. But an actuary at Cigna or CMS? No, thank you.

This month, as you begin to adopt the new ICD-10 coding system—mandatorily, of course—that will increase the amount of health data collected by orders of magnitude, consider the cons as well as the pros of so-called "big data" efforts. They can harm just as easily as they help. ■

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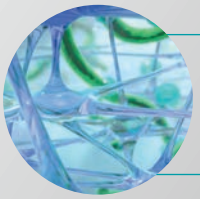

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Learning the Texas Lingo

Things are a little different around here, but I think I'm gonna like it.

By Montgomery Vickers, OD

Do you recall your first real job, way back in the day? I worked at Montgomery Supermarket for $1.00 per hour. I was 15 years old, and they paid me in cash every week. I was a stock boy, meaning I had to run up and down steps carrying cans of beans and such to put on the shelves. My immediate supervisors were 20-year-old brothers, Harlan and Gary, pronounced, by them, as "Hah-lahn and Gah-ree." They spoke some kind of Appalachian mountain dialect. The only thing I remember that they ever said was "bedeer bedarr." I said "OK" and carried down more cans of beans and such.

I quit every day, but Mr. Montgomery called Mom and she assured him, every day, that I would be back in the morning—and I was.

It didn't take too much of this to convince me to be my own boss one day. And that's how it all turned out, as I practiced private practice optometry for 35 years—right up until July 22, 2015.

Now I am beginning my second stint as an employee right here in the Big D: Dallas, Texas. This is my first week in an all-medical eye care practice, and I am lovin' it.

I love the practice, the specialist, the staffers and the grub we ate at lunch today.

Happy birthday, Toni, and thanks for the food, Heather!

I have already learned that there are many differences between optometry in St. Albans, W.Va. and optometry in Dallas, Texas. Here are just a few:

In West Virgina it's …	In Texas it's …
Muscae volitantes	Brisket
Bilateral cataracts	Bilateral Cadillacs
Scleral buckle	Silver buckle
Pterygium	Scorpion
Tea tree oil	Oil. Just oil.
Sampaolesi's	Tamales
Three other ODs in town	300 ODs in my building
Will he show up for his appt.?	Willie Nelson
Bifocals	Buy boots
Glaucoma	Guacamole
Schlemm's canal	Rio Grande
Try to succeed	Try to secede
Prokera	Pro football
Corneal abrasion	Chap chafe
Branch vein occlusion	Ranch vein occlusion
Donder's table	Texas fried pies

As you can see, there are a lot of things different out here in the Lone Star State. But ...

My Dad wore cowboy hats. At Christmas I always asked for a cowboy gun and holster. Now, Dad's gone to the back 40 in the sky, and I wear his hats. I also still want the six-shooter. Heck, down here I could strap it on and wear it. It's OK! This is just a leftover of the long history of the independent soul of Texas. I'm starting my journey with my new Texan OD buddies and the search for tamale nirvana, but can't find a good ol' Mountain State pepperoni roll anywhere. Maybe at a Big 12 tailgate. See you there. ■

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Neovascular Glaucoma Stages

Depending on the presentation, this diagnosis may be cause for urgent concern.

By Richard Mangan, OD

Neovascular glaucoma (NVG) is a secondary glaucoma that is considered an advanced complication of ischemic retinal vascular disease. NVG accounts for 3.9% of all glaucoma cases.[1] Most commonly related to proliferative diabetic retinopathy, central retinal vein occlusion (RVO) or ocular ischemic syndrome, retinal ischemia triggers a cascade of events that lead to aberrant blood vessel growth over the anterior iris and into the anterior chamber angle, also referred to as rubeosis iridis. IOP is adversely affected when fibrovascular tissue forms in the iridocorneal angle, impeding trabecular meshwork outflow. When left unchecked, this fibrous tissue leads to peripheral anterior synechiae and secondary angle closure.[1]

While patients with NVG can present without symptoms, they are more likely to report any combination of symptoms, including: redness, pain, light sensitivity, headache and decreased vision.[1] In cases of secondary angle closure, headaches become more intense and may be associated with nausea and vomiting.[2]

Etiology

Iris neovascularization at initial presentation is considered an indicator of poor visual prognosis. Entrance acuities can range from 20/40 to no light perception, with most presenting at 20/200 vision or worse.[3,4] More than 95% of such eyes develop visual acuity of counting fingers or worse within one year.[5] Despite the guarded prognosis, NVG is considered an ocular urgency because the potential for complete loss of vision, intractable pain and globe enucleation increases with delay in management (*Table 1*).[6]

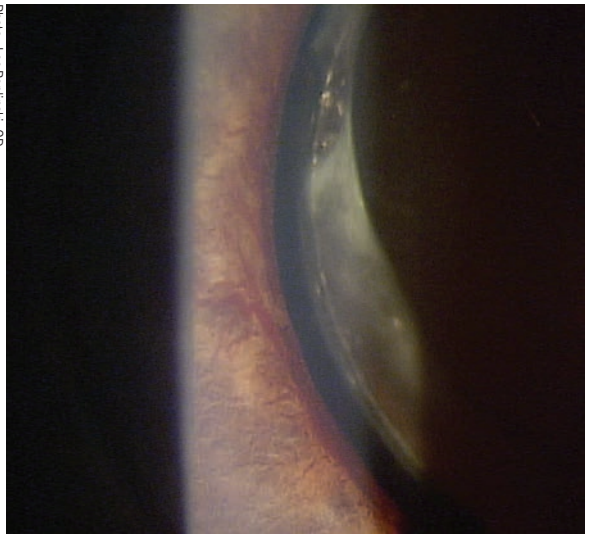

Photo: Lee Peplinski, OD

Fig. 1. Slit lamp image of rubeosis iridies after CRVO.

When confronted with a patient who has neovascular glaucoma, a careful and thorough case history may be helpful in determining the underlying etiology. Patients with a history of poorly controlled Type 1 diabetes, with or without proliferative diabetes retinopathy, may certainly have diabetic NVG (*Table 2*). However, diabetic patients often have other health problems such as hypertension and hyperlipidemia.[7] A patient who has these and other risk factors (*Tables 2 and 3*) and reports sudden, profound vision loss 60 to 90 days prior my have an ischemic CRVO.

Table 1. Three Stages of NVG

Stage	1	2	3
	Rubsosis iridis	Secondary open-angle glaucoma	Secondary angle closure glaucoma
Symptoms	None to mild, redness, pain, photophobia	Redness, pain, photophobia, mild headache	Acute severe pain, headache, photophobia, nausea or vomitting
Pupils	Poorly reactive	Poorly reactive	Distorted, fixed, mid-dilated. May include eversion of pupillary margin
SLE	Neovascular tufts at pupillary margin NVI (irregular, non-radial vessels)	Positive NVI	Conjunctival injection (w/ ciliary flush), positive NVI +/- hyphema with corneal edema, aqueous flare
Tonometry	Normal IOP	Elevated IOP	Significantly elevated IOP (50+mm Hg)
Gonioscopy	NVA (Can occur without NVI)	Positive NVA, fibrovascular membrane formation	NVA + fibrovascular membrane formation, synechial angle closure

For allergic conjunctivitis[1]

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IMPORTANT RISK INFORMATION

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

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

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

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

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

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

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

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

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

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

Revised: 10/2012

FULL PRESCRIBING INFORMATION: CONTENTS*

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

FULL PRESCRIBING INFORMATION

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

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

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

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

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

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

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

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

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

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

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

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

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

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

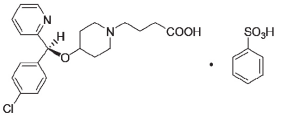
8.3 Nursing Mothers
Following a single 3 mg/kg oral dose of radiolabeled bepotastine besilate to nursing rats 11 days after delivery, the maximum concentration of radioactivity in milk was 0.40 mcg-eq/mL 1 hour after administration; at 48 hours after administration the concentration was below detection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

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

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

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

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



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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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Osaka, Japan 541-0046

US/BEP/13/0028 4/13

In cases when no retinal ischemia is evident, ask about a history of carotid artery disease on the same side as the eye with NVG, as this is a risk factor for ocular ischemic syndrome due to carotid insufficiency.

If unsure, consider ordering a carotid duplex ultrasound.[6] Additional ancillary testing and serology may be useful in staging the disease and isolating the cause of the retinal hypoxia and secondary NVG (*Table 4*).

Treatment

Management of NVG can be challenging and complex. It is often best comanaged in a team approach with subspecialists in both retina and glaucoma.

Whom you will call first will depend on disease severity based on your clinical assessment.

The mainstay of treatment is ischemia and IOP control. Patients who present with redness, pain, moderately elevated IOP (Stage 1 or 2 NVG) or some combination of those, will likely benefit from the initiation of topical medical therapy, such as IOP-lowering agents (e.g., b-blockers, alpha-agonists, CAIs), topical steroids and cycloplegics.

Remember, the use of prostaglandin analogues in lowering IOP in NVG patients is controversial, as they are pro-inflammatory.[8]

Regardless of disease stage, consult a retinal specialist for consideration of pan-retinal photocoagulation (PRP) with or without anti-VEGF treatment.

According to the Diabetic Retinopathy Study (DRS), reducing the amount of viable retina via PRP has been shown to inhibit, reduce and reverse neovascularization of the anterior segment.[9]

Using a spot size of 500µm to 800µm, up to 2,000 laser burns are applied to the outer retina using one of three methods: a slit-lamp delivery system, an indirect laser or an endolaser, if a vitrectomy is warranted.

Anti-VEGF drugs such as Avastin (Genentech), Macugen (Gilead Sciences), Eylea (Regeneron), and Lucentis (Genentech) block angiogenic factors that promote new vessel proliferation.[10]

Several favorable reports exist regarding intravitreal and, to a lesser extent, intracameral administration of anti-VEGF agents in patients with NVG.[11] While occasionally used in isolation, when visibility of the posterior segment is poor (i.e., hemorrhage), the trend is for the concomitant use of these drugs with PRP.

Surgical Options

In some cases, this combined treatment along with topical medical therapy is enough to control patients' intraocular pressure. However, history shows that approximately 80% of patients with NVG will inevitably require surgical intervention.[6,8] Therefore, a glaucoma specialist should also be consulted when confronted with NVG.

Glaucoma surgery may be indicated for refractory stage 2 cases in eyes with remaining functional vision. Ideally, surgery is performed three to four weeks after PRP, so the eye has had enough time to quiet down before being subjected to surgery.

The use of anti-VEGF drugs prior to trabeculectomy (with or without an anti-fibrotic agent such as mitomycin C or 5-FU) or valve implant surgery, can also improve outcomes.[12]

With that said, most surgeons prefer tube shunt surgery to trabeculectomy because outcomes are less affected by inflammation. This is especially true in emergent cases (i.e., IOP of 65mm Hg) when there is no time to pre-treat the inflammation with PRP.

According to one study, regardless of the type of tube shunt, success rates are comparable.[13] Unfortunately, when comparing surgical outcomes (sustained IOP reduction) between NVG patients and other forms of glaucoma, NVG patients continue to exhibit the worst overall results.[14]

When there is no visual potential, treatment is mainly focused on pain control. Sometimes, this can be accomplished medically with 1%

Table 2. Incidence of NVG

IDDM[1,2]

- 5% chance in NPDR
- 45% to 65% in untreated PDR
- IDDM patients have 8% to 24% chance of NVD, NVE or NVI

CRVO

- 20% of all CRVOs are ischemic
- NVG occurs in 45% of ischemic CRVOs
- 8% to 10% of ALL CRVO patients

1. American Optometric Association. Optometric Clinical Practice Guide: Care of the Patient with Diabetes Mellitus. Available at: www.aoa.org/documents/CPG-3.pdf accessed: September 18, 2015.
2. Diabetes Control and Complications Trial (DCCT). Update. DCCT Research Group. Diabetes Care. 1990 Apr;13(4):427-33.

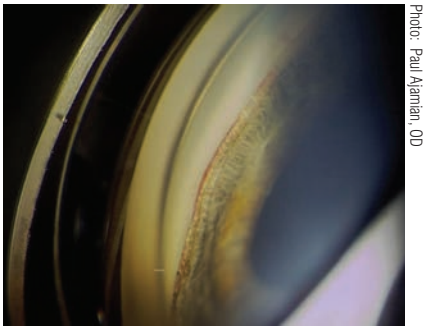

Photo: Paul Ajamian, OD

Slit lamp image shows rubeosis iridis.

atropine, topical steroids and IOP lowering eye drops.

In cases of corneal decompensation, bandage contact lenses may be used. If medical therapy fails to provide relief, consider recommending cyclodestructive procedures such as cyclocryotherapy or cyclophotocoagulation.

Retrobulbar alcohol injection is indicated after medical and surgical treatments have failed and in cases where the patient does not want the eye enucleated.

Overall Health

Lastly, neovascular glaucoma is associated with a high mortality rate.[1] These patients are generally of poor health; consequently for the comanaging optometrist, communication with their primary care providers is vital. Their physicians can likely provide a more detailed general health history, while providing insight into their likelihood of following a prescribed treatment plan.

Based on your conversation, a complete medical work-up may be warranted, including the aforementioned lab studies.

The management of neovascular glaucoma is certainly challenging, and the diagnosis carries a poor prognosis. While new treatments (i.e., topical anti-VEGF therapy, pigment epithelium-derived factor and new treatment protocols (i.e., RISE & RIDE trials) are being developed, we need to remain diligent in educating patients on the importance of proper diet, regular exercise, compliance with prescribed treatments and keeping scheduled doctor's appointments in the fight against proliferative eye disease.

The best chance of deferring vision loss secondary to neovascular glaucoma is undoubtedly to prevent it from developing in the first place. ■

Table 3. CRVO Risk Factors

More Common	
Age	90% occur in patients age 55 or older
Hypertension	Present in 73% of cases
Hyperlipidaemia	Present in 35% of cases
DM	Present in 10% of cases
Contraceptive pills	
High IOP	
Smoking	
Less Common	
Behçet syndrome	
Sarcoidosis	
Wegener granulomatosis	
Myeloproliferative disorders	
Chronic renal failure	
Cushing syndrome	
Hypothyroidism	
Orbital disease	
Dehydration	

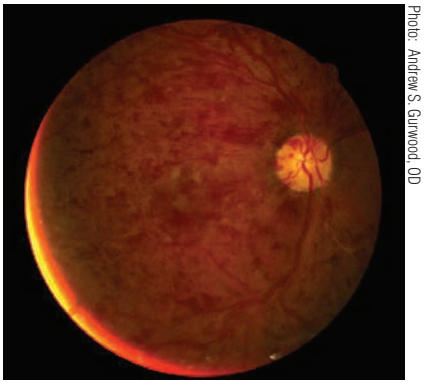

Photo: Andrew S. Gurwood, OD

Fundus image displays central retinal artery occlusion.

1. Shazly T, Latina M. Neovascular glaucoma: etiology, diagnosis and prognosis. Semin Ophthalmol. 2009 Mar-Apr;24(2):113-21.
2. Yazdani S, Hendi K,. Intravitreal bevacizumab (Avastin) injection for neovascular glaucoma. J Glaucoma. 2007 Aug;16(5):437-9.
3. Kuang T, Ling Liu C, Chou C, Hsu W. Clinical experience in the management of neovascular glaucoma. J Chin Med Assoc. 2004 Mar;67(3):131-5.
4. Tatsumi T, Yamamoto S, Uehara J, et al. Panretinal photocoagulation with simultaneous cryoretinopexy or intravitreal bevacizumab for neovascular glaucoma. Graefes Arch Clin Exp Ophthalmol. 2013 May;251(5):1355-60.
5. Sivalingam A, Brown GC, Magargal LE (1991) The ocular ischaemic syndrome. Visual prognosis and the effect of treatment. Int Ophthalmol 15:15–20.
6. Sivak-Callcott J, O'Day D, Gass D, Tsai J. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. Ophthalmology. 2001 Oct;108(10):1767-76.
7. Davis J, Chung R, Juarez D. Prevalence of Comorbid Conditions with Aging Among Patients with Diabetes and Cardiovascular Disease. Hawaii Med J. 2011 Oct;70(10):209–13.
8. The Diabetic Retinopathy Study Research Group. Indications for photocoagulation treatment of diabetic retinopathy: Diabetic Retinopathy Study Report no. 14. Int Ophthalmol Clin 1987;27:239-53.
9. Olmos L, Lee R. Medical and surgical treatment of neovascular glaucoma. Int Ophthalmol Clin. 2011 Summer; 51(3):27-36.
10. Lüke J, Nassar K, Lüke M, Grisanti S. Ranibizumab as adjuvant in the treatment of rubeosis iridis and neovascular glaucoma-results from a prospective interventional case series. Graefes Arch Clin Exp Ophthalmol. 2013 Oct. 251(10):2403-13.
11. Martinez-Carpio PA, Bonafonte-Marquez E, Heredia-Garcia CD, Bonafonte-Royo S. [Efficacy and safety of intravitreal injection of bevacizumab in the treatment of neovascular glaucoma: systematic review]. Arch Soc Esp Oftalmol. 2008 Oct. 83(10):579-88.
12. Luke J, Luke M, Grisanti S. [Antiangiogenic treatment for neovascular glaucoma and after filtering surgery]. Ophthalmologe. 2009 May. 106(5):407-12
13. Hong CH, Arosemena A, Zurakowski D, Ayyala RS. Glaucoma drainage devices: A systematic literature review and current controversies. Surv Ophthalmol 2005;50:1:48-60.
14. Schwartz, KS, Lee RK, Gedde SJ. Glaucoma drainage implants: a critical comparison of types. Curr Opin Opthalmol. 2006;17:181-9.

Table 4. Preliminary Testing to Determine NVG Etiology

Ancillary Testing:	Anterior segment OCT, NFL OCT, fluorescein angiography, ERG, B-Scan ultrasound, carotid auscultation and duplex ultrasound Blood pressure evaluation
Initial Lab Work:	CBC w/ Diff, platelets, ESR, CRP, FBG, A1c, ANA, lipid panel, total cholesterol panel

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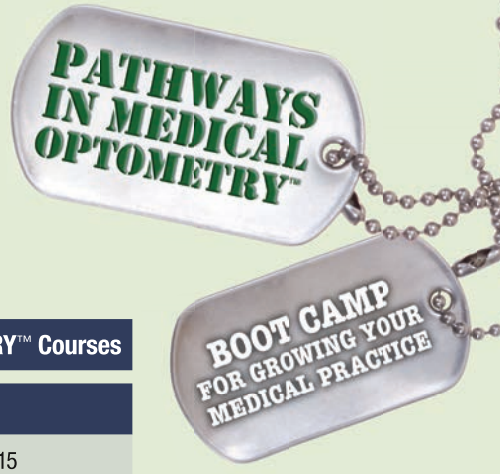

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Drag and Droop

What's causing this patient's ptosis and diplopia? It's time to think systemic.

Edited by Paul C. Ajamian, OD

Q A 21-year-old presented to my office stating that his right lid was "swollen" and he was seeing double at times. Where do I start with the workup?

A "I would first investigate for an infectious/infiltrative process of the eye with the lid swelling," says Leonard Messner, OD.

Orbital cellulitis, orbital pseudotumor and other infiltrative processes commonly produce eyelid edema with restriction of ocular motility and frequently affect the optic nerve to produce visual dysfunction, Dr. Messner says.

Of course, there may be more to the story. "If the involved lid is more of a ptotic vs. swollen lid, this changes the decision tree to explore the differentials related to ptosis associated with diplopia," Dr. Messner says. "If the ptosis and motility deficits are ipsilateral, investigate for isolated or combined cranial neuropathy, such as oculomotor palsy or cavernous sinus syndrome, respectively."

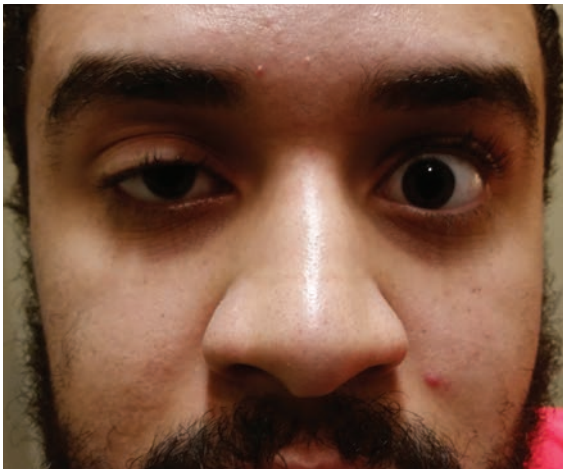
If the ptosis is associated with a contralateral motility deficit, this points strongly to myasthenia gravis (MG). "If the motility and eyelid dysfunctions do not obey the rules of neuroanatomy and vary over the course of the day, think MG," says Dr. Messner.

Diagnostic Tools

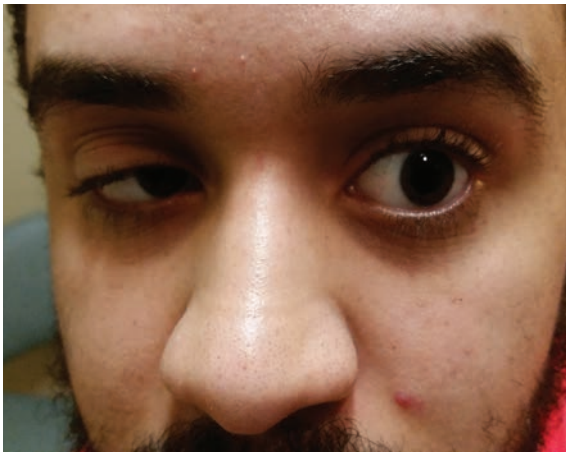
If you suspect neuromuscular disease, use your diagnostic toolbox to elucidate its exact etiology. For example, "Osher's 'peek sign' is performed by asking the patient to gently close their eyelids as if they are sleeping," says Dr. Messner. "Since concomitant orbicularis weakness is common with MG, it's often easy to manually pry the lids apart."

Ptosis and diplopia may not always occur together in MG. "Ptosis alone may be the first clinical presentation of ocular MG," Dr. Messner says. "So, check all patients with ptosis for orbicularis weakness."

You can also use Cogan's lid twitch. "I ask the patient to look in a slightly downward position for 10 to 20 seconds, then redirect their gaze to primary position. With the redirection, the upper lid will typically overshoot or twitch before coming down to its normal position," Dr. Messner explains.



Acquired ptosis in one eye and a motility restriction in the other should be considered myasthenia gravis until proven otherwise.



An ice pack is also an effective MG diagnostic tool. Dr. Messner uses crushed ice in a surgical glove applied to the eye with closed lids for several minutes. Improvement of the ptosis is highly suggestive of MG. He also takes facial photos before and after to check for any signs of improvement.

Historically, 50% to 80% of ocular MG cases convert to generalized MG, with later-in-life diagnosis as a risk factor for conversion, Dr. Messner says, with recent research showing a conversion rate of 21%.[1] He is careful to note that ocular myasthenia gravis is not distinct from generalized myasthenia gravis.

"It is incorrect to think of ocular myasthenia and generalized myasthenia as distinct disease processes; rather, ocular MG represents an early form of the disease," notes Dr. Messner.

It is important to question patients as to the presence of other bulbar signs (difficulty chewing, swallowing and breathing), as these are also common with MG, yet are often overlooked or misdiagnosed, Dr. Messner says.

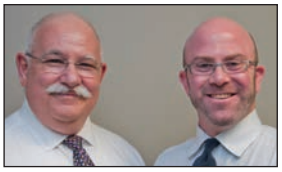
"Many patients complain of difficulty swallowing, with the build-up of phlegm in their throat owing to paralysis of their laryngeal muscles," Dr. Messner says. He has also seen patients with breathing problems initially attributed to sinus disease. "If I suspect an individual of ocular MG, I order acetylcholine antibody titers along with a chest CT to rule out thymoma, which is present in about 15% of the MG cohort."

Treatment

The initial goal is to effectively treat the underlying disease process. Since anticholinesterase agents do not treat its autoimmune etiology, early treatment with prednisone is the evolving trend, according to Dr. Messner. Immunosuppresant therapy can reduce the rate of conversion to 10.5%.[1]

"While pharmacotherapy is much more effective than in the past, many individuals will continue to have problems with persistent ptosis and diplopia," says Dr. Messner. Optometric care may include ptosis crutches or eyelid taping. For diplopia during the acute and subacute stages, Dr. Messner suggests unilateral occlusion and prism therapy once stable. ■

1. Nagia L, Lemos J, Abusamra K, et al. Prognosis of ocular myasthenia gravis. Ophthalmol. 2015 April 16. [Epub].



Child's Play—Or Is It?

A seemingly simple diagnosis may overshadow common independent comorbidities that also require management. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

Sometimes, a condition that warrants our attention may also distract us from our obligation to consider the patients' total ocular and visual status. Often, the identification of an ocular disease stops the diagnostic process short of fully identifying all of the patient's needs, such as low vision management, prism or vision therapy. The presentation of deficiencies in visual acuity can sometimes be attributed to the effects of ocular disease as well as factors that fall within the scope of routine optometric practice. As such, optometrists must complete a thorough optometric evaluation notwithstanding the presence of ocular disease. This is especially important in the context of a case where the ocular disease limits visual acuity.

Surgical Shortcomings

A seven-year-old male was referred to us with a presentation of long-standing bilateral impairment of visual acuity, lateral movement of the head to the left side and increased frequency of squinting and closing the right eye. The patient had a history of esotropia and nystagmus at one year of age, which was addressed surgically in 2008 and was said to have resolved, subsequent to patching therapy that was ultimately unsuccessful. He was deficient in balance and depth perception, which was amplified in dark conditions. The patient's medical and developmental histories were overall unremarkable.

The patient demonstrating the head turn found upon first presentation. He had to be told which direction his head used to turn as he did not remember.

The patient's mother observed that he had trouble walking up and down stairs, and on occasion preferred to crawl down them. He had difficulty distinguishing the borders of objects from a distance. She also indicated that he was having trouble with reading. Although the patient said that he did not have trouble seeing written material in his textbooks, his mother noted that he held reading material very close to his face. He also needed to sit in the front of the classroom and often walked up to the board in order to see the text. His mother observed that his movements appeared uncoordinated. He was in standard education classes and was underperforming, she said. His school was providing materials in large print, but was not being forthcoming with other compensatory strategies.

His entering corrected visual acuity was 20/80 at distance (OU). At near with both eyes together he saw 20/40 with a hyperopic astigmatic correction and flat-top bifocal (OD +4.25-3.00 x165, OD +4.00-4.00 x 180 OS, +2.75 add OU).

All chair skills were within normal limits except for the presence of nystagmus, which was seen when we performed eye movement testing was during which he showed significant fixation loss. Gross motor eye movement testing showed poor pursuit saccades. No improvement was noted in visual acuity with trial frame refraction. Phoroptor testing, cover test and NPC were not attempted secondary to the presence of nystagmus, although there was no gross deviation present by observation. The nystagmus was found to oscillate least when the patient had his head turned to the left. Anterior segment and indicated posterior segment evaluations were within normal limits.

Based on the previous primary care and current vision therapy examinations, the patient was diagnosed with decreased vision secondary to congenital nystagmus and oculomotor dysfunction. A three-pronged approach to treat the patient was put into effect.

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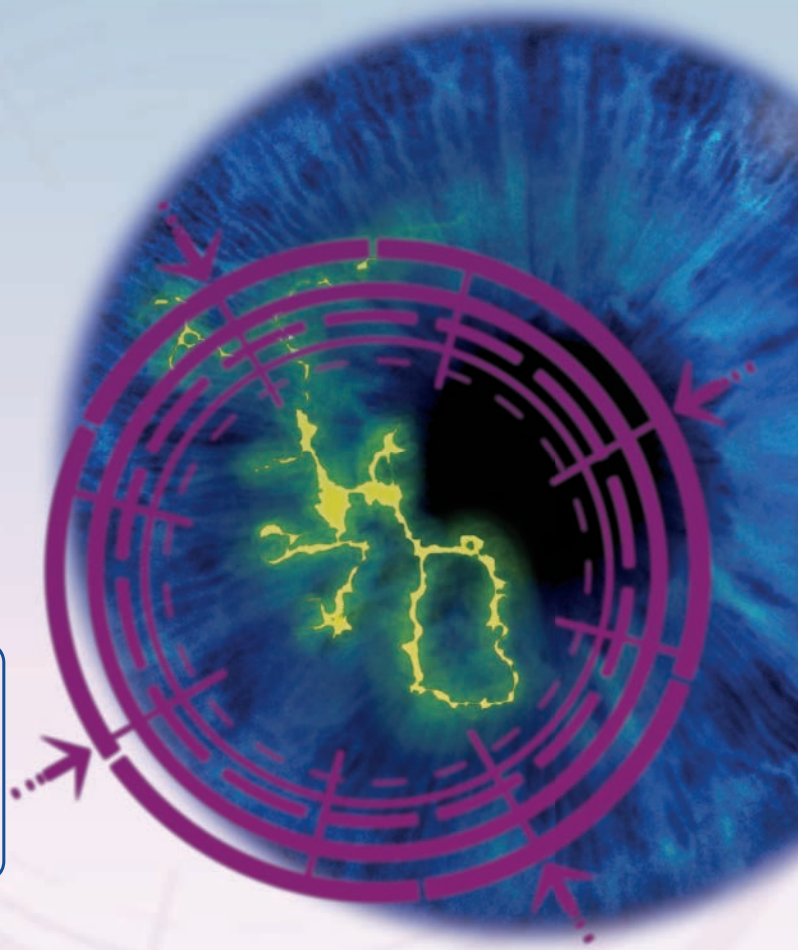


- Inactive in healthy corneal cells[1]
- Up to 77% of dendritic ulcers resolved at Day 7[2,3,*]

*As demonstrated in a phase 3 open-label, randomized, controlled, multicenter clinical trial (N=164) in which patients with herpetic keratitis received either ZIRGAN® or acyclovir ophthalmic ointment 3%, administered 5 times daily until healing of ulcer and then 3 times daily for 1 week. Clinical resolution (healed ulcers) at day 7 was achieved in 77% (55/71) of patients treated with ZIRGAN® versus 72% (48/67) treated with acyclovir (difference, 5.8%; 95% CI, -9.6%-18.3%). ZIRGAN® was noninferior to acyclovir in patients with dendritic ulcers.

Indication

ZIRGAN® (ganciclovir ophthalmic gel) 0.15% is a topical ophthalmic antiviral that is indicated for the treatment of acute herpetic keratitis (dendritic ulcers).

Important Safety Information about ZIRGAN®

- ZIRGAN® is indicated for topical ophthalmic use only.
- Patients should not wear contact lenses if they have signs or symptoms of herpetic keratitis or during the course of therapy with ZIRGAN®.
- Most common adverse reactions reported in patients were blurred vision (60%), eye irritation (20%), punctate keratitis (5%), and conjunctival hyperemia (5%).
- Safety and efficacy in pediatric patients below the age of 2 years have not been established.

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

References: 1. Foster CS. Ganciclovir gel—a new topical treatment for herpetic keratitis. *US Ophthalmic Rev.* 2008;3(1):52-56. **2.** ZIRGAN Prescribing Information, April 2014. **3.** Croxtall JD. Ganciclovir Ophthalmic Gel 0.15% in Acute Herpetic Keratitis (Dendritic Ulcers). *Drugs*. 2011;71(5):603-610.

BAUSCH + LOMB

Zirgan®
(ganciclovir ophthalmic gel) 0.15%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

Zirgan ganciclovir ophthalmic gel 0.15%

Initial U.S. Approval: 1989

1 INDICATIONS AND USAGE

ZIRGAN (ganciclovir ophthalmic gel) 0.15% is indicated for the treatment of acute herpetic keratitis (dendritic ulcers).

2 DOSAGE AND ADMINISTRATION

The recommended dosing regimen for ZIRGAN is 1 drop in the affected eye 5 times per day (approximately every 3 hours while awake) until the corneal ulcer heals, and then 1 drop 3 times per day for 7 days.

3 DOSAGE FORMS AND STRENGTHS

ZIRGAN contains 0.15% of ganciclovir in a sterile preserved topical ophthalmic gel.

4 CONTRAINDICATIONS

None.

5 WARNINGS AND PRECAUTIONS

5.1 Topical Ophthalmic Use Only

ZIRGAN is indicated for topical ophthalmic use only.

5.2 Avoidance of Contact Lenses

Patients should not wear contact lenses if they have signs or symptoms of herpetic keratitis or during the course of therapy with ZIRGAN.

6 ADVERSE REACTIONS

Most common adverse reactions reported in patients were blurred vision (60%), eye irritation (20%), punctate keratitis (5%), and conjunctival hyperemia (5%).

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy: Teratogenic Effects

Pregnancy Category C: Ganciclovir has been shown to be embryotoxic in rabbits and mice following intravenous administration and teratogenic in rabbits. Fetal resorptions were present in at least 85% of rabbits and mice administered 60 mg/kg/day and 108 mg/kg/day (approximately 10,000x and 17,000x the human ocular dose of 6.25 mcg/kg/day), respectively, assuming complete absorption. Effects observed in rabbits included: fetal growth retardation, embryolethality, teratogenicity, and/or maternal toxicity. Teratogenic changes included cleft palate, anophthalmia/microphthalmia, aplastic organs (kidney and pancreas), hydrocephaly, and brachygnathia. In mice, effects observed were maternal/fetal toxicity and embryolethality. Daily intravenous doses of 90 mg/kg/day (14,000x the human ocular dose) administered to female mice prior to mating, during gestation, and during lactation caused hypoplasia of the testes and seminal vesicles in the month-old male offspring, as well as pathologic changes in the nonglandular region of the stomach (see Carcinogenesis, Mutagenesis, and Impairment of Fertility).

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

8.3 Nursing Mothers

It is not known whether topical ophthalmic ganciclovir administration could result in sufficient systemic absorption to produce detectable quantities in breast milk. Caution should be exercised when ZIRGAN is administered to nursing mothers.

8.4 Pediatric Use

Safety and efficacy in pediatric patients below the age of 2 years have not been established.

8.5 Geriatric Use

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

12 CLINICAL PHARMACOLOGY

12.1 Mechanism of Action

ZIRGAN (ganciclovir ophthalmic gel) 0.15% contains the active ingredient, ganciclovir, which is a guanosine derivative that, upon phosphorylation, inhibits DNA replication by herpes simplex viruses (HSV). Ganciclovir is transformed by viral and cellular thymidine kinases (TK) to ganciclovir triphosphate, which works as an antiviral agent by inhibiting the synthesis of viral DNA in 2 ways: competitive inhibition of viral DNA-polymerase and direct incorporation into viral primer strand DNA, resulting in DNA chain termination and prevention of replication.

12.3 Pharmacokinetics

The estimated maximum daily dose of ganciclovir administered as 1 drop, 5 times per day is 0.375 mg. Compared to maintenance doses of systemically administered ganciclovir of 900 mg (oral valganciclovir) and 5 mg/kg (IV ganciclovir), the ophthalmically administered daily dose is approximately 0.04% and 0.1% of the oral dose and IV doses, respectively, thus minimal systemic exposure is expected.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, and Impairment of Fertilty

Ganciclovir was carcinogenic in the mouse at oral doses of 20 and 1,000 mg/kg/day (approximately 3,000x and 160,000x the human ocular dose of 6.25 mcg/kg/day, assuming complete absorption). At the dose of 1,000 mg/kg/day there was a significant increase in the incidence of tumors of the preputial gland in males, forestomach (nonglandular mucosa) in males and females, and reproductive tissues (ovaries, uterus, mammary gland, clitoral gland, and vagina) and liver in females. At the dose of 20 mg/kg/day, a slightly increased incidence of tumors was noted in the preputial and harderian glands in males, forestomach in males and females, and liver in females. No carcinogenic effect was observed in mice administered ganciclovir at 1 mg/kg/day (160x the human ocular dose). Except for histocytic sarcoma of the liver, ganciclovir-induced tumors were generally of epithelial or vascular origin. Although the preputial and clitoral glands, forestomach and harderian glands of mice do not have human counterparts, ganciclovir should be considered a potential carcinogen in humans. Ganciclovir increased mutations in mouse lymphoma cells and DNA damage in human lymphocytes in vitro at concentrations between 50 to 500 and 250 to 2,000 mcg/mL, respectively.

In the mouse micronucleus assay, ganciclovir was clastogenic at doses of 150 and 500 mg/kg (IV) (24,000x to 80,000x human ocular dose) but not 50 mg/kg (8,000x human ocular dose). Ganciclovir was not mutagenic in the Ames Salmonella assay at concentrations of 500 to 5,000 mcg/mL. Ganciclovir caused decreased mating behavior, decreased fertility, and an increased incidence of embryolethality in female mice following intravenous doses of 90 mg/kg/day (approximately 14,000x the human ocular dose of 6.25 mcg/kg/day). Ganciclovir caused decreased fertility in male mice and hypospermatogenesis in mice and dogs following daily oral or intravenous administration of doses ranging from 0.2 to 10 mg/kg (30x to 1,600x the human ocular dose).

14 CLINICAL STUDIES

In one open-label, randomized, controlled, multicenter clinical trial which enrolled 164 patients with herpetic keratitis, ZIRGAN was non-inferior to acyclovir ophthalmic ointment, 3% in patients with dendritic ulcers. Clinical resolution (healed ulcers) at Day 7 was achieved in 77% (55/71) for ZIRGAN versus 72% (48/67) for acyclovir 3% (difference 5.8%, 95% CI - 9.6%-18.3%). In three randomized, single-masked, controlled, multicenter clinical trials which enrolled 213 total patients, ZIRGAN was non-inferior to acyclovir ophthalmic ointment 3% in patients with dendritic ulcers. Clinical resolution at Day 7 was achieved in 72% (41/57) for ZIRGAN versus 69% (34/49) for acyclovir (difference 2.5%, 95% CI - 15.6%-20.9%).

17 PATIENT COUNSELING INFORMATION

This product is sterile when packaged. Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the gel. If pain develops, or if redness, itching, or inflammation becomes aggravated, the patient should be advised to consult a physician. Patients should be advised not to wear contact lenses when using ZIRGAN.

Revised: April 2014

Bausch & Lomb Incorporated
Tampa, FL 33637

US/ZGN/15/0005
Based on 9224702 (flat)-9224802 (folded)

1. Low Vision Treatment

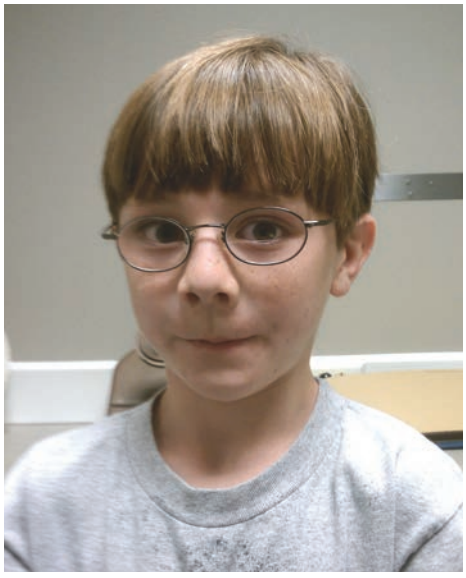
The visual acuity loss was managed with traditional low vision devices.

For distance, Max TV glasses (Eschenbach)—a 2x magnification system—were worn during school and for other distance activities. In office, visual acuity with the Max TV glasses was measured at 20/40 OU. The downside to using these glasses is that the patient had to remove his habitual prescription, but the acuity gain made this a moot point. When these were placed on the child's face, he smiled and said that he had never seen so well.

For near vision, a 4x dome magnifier was used for all activities. In office, visual acuity with the dome magnifier was measured at 20/15 OU. This portable device allowed him comfort knowing that it would always be with him and could fit into his backpack or pocket. Additionally, a 2x bar magnifier with light was used for near activities involving reading, so that the patient can see the entire line at once. Patients with low vision often benefit from increased lighting. In this case, the contrast on the copied work was extremely poor, so the patient was excited.

2. Yoked Prism

A 4Δ yoked prism (prism with the bases in the same direction and of equal power) based left, was used to reduce the amount of head turn, essentially driving the patient into the null point.[1,2] Yoked prism shifts the image in the same direction in each eye without impacting the image quality. For this child, reducing the amount of time that he was turning his head was crucial to avoiding issues with posture and neck/spinal issues in the future. The amount was determined by trial and error and, once determined,



The patient with glasses, showing the lack of head turn.

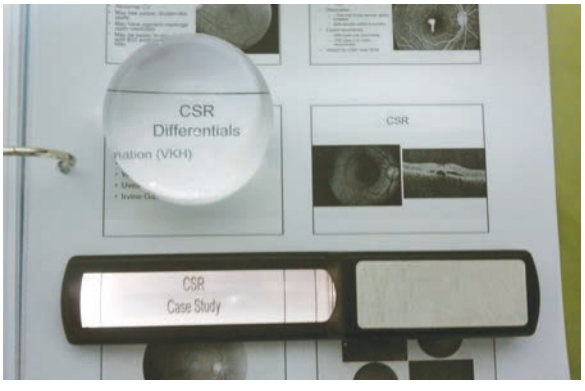
was trialed for two weeks using Fresnel prism. While the impact was immediately witnessed in the office, adaptation was a concern. Upon evaluation several weeks later, his mother indicated that the turn was still significantly reduced so the decision was made to grind in the prism.

3. Oculomotor-based Vision Therapy

Therapy was implemented to improve eye movements and the coordination of the visual motor system. Since the child was performing poorly on visually dependent activities, the therapy program encouraged better quality and more efficient visual input. Reading tutoring was a future consideration but vision therapy would set the stage for future success. Since many of the complaints were visual-motor based, the therapy program stressed gross motor visual skills at first, before moving to fine motor and reading eye movements. The therapy moved from larger to smaller targets and moved in sequence from monocular, bi-ocular to binocular skills. Fixation skills were addressed, then pursuits and finally saccades. Activities included but were not limited to Wayne Saccadic Fixator (an electronic board with lights that appear in set patterns and provides tactile and auditory feedback, Wayne Engineering), pegboard (stationary and rotating), chalkboard racetrack and stick in straw, starting with marker in paper towel roll.

This child's visual acuity and efficiency issues, along with potential neck pain, might have been overlooked or disregarded. This case shows that blending several optometric disciplines allows practitioners to use all of the tools at their disposal to treat the visual and systemic signs and symptoms intrinsic to each of our patients. ■

1. Cotter SA. Clinical Uses of Prism: A Spectrum of Applications. St. Louis: Mosby, 289.
2. Fischer V, Mahaphon T. Use of Yoked Prism in the Management of Nystagmus in Spina Bifida. Optometry 2006;77(6):275-6.



4x dome and 2x bar magnifiers aided near vision tasks.

Comanagement Essentials

It is important to keep up with the legalities of pre- and post-cataract surgery care.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Comanagement is a non-financial arrangement between a physician performing surgery and a comanaging physician who provides care to the patient for some portion of the global follow-up period. Comanagement is available for any procedure with a global period of 10 days or greater, and the rules remain the same irrespective of the length of the global period.

Comanagement of any surgery begins with the formal transfer of care from the surgeon to the comanaging physician—typically to the physician who originally referred the patient for a surgical evaluation. However, a referral cannot be based upon the requirement that the surgeon refer the patient back to the referring physician. In a comanagement situation, the patient chooses the comanaging physician, so be sure to discuss the arrangement with your patient before the initial surgical evaluation. Above all, the patient's wellbeing is the most important factor to consider.

Each physician has certain protocols to follow. Have a clear agreement in place with the surgeon to establish the guidelines for communication, timely reports back to the surgeon and when the patient will be seen again after surgery. The surgeon should provide information on the surgery claim filed, so you can use the correct information for the postoperative care claim.

Let's look at coding for traditional monofocal IOL implantation.

Pre-Cataract Surgery

In most cases, a comprehensive eye examination and a single diagnostic ultrasound A-scan to determine the appropriate pseudophakic power of the intraocular lens are sufficient (*Table 1*). For patients with a dense cataract, an ultrasound B-scan may be used.

Where the only diagnosis is cataract(s), Medicare does not routinely cover testing other than one comprehensive eye examination and an A-scan or, if medically justified, a B-scan. Claims for additional tests are denied as not necessary unless there is an additional diagnosis and the medical necessity for the additional tests is fully documented.

Transfer of Care

The global surgery fee schedule allowance includes preoperative evaluation and management services rendered the day of or the day before surgery, the surgical procedure and the postoperative care services within the defined postoperative period. Postoperative care may be rendered by an ophthalmologist, optometrist or providers licensed to render such services. A transfer of care occurs when the referring physician transfers the responsibility for the patient's complete care to a receiving physician outside of their group practice at the time of referral, and the receiving physician documents approval of care in advance. Each provider must agree and document the transfer of care in the medical record. The agreement must be in the form of a letter or written as a notation in the discharge summary, hospital records or ambulatory surgical center records.

The appropriate CPT-4 modifiers must be added to the surgical procedure code:

- -54 surgical care only
- -55 postoperative management only
- -79 unrelated procedure or service by the same physician during the postoperative period

The claim for surgical care only and postoperative care only must identify the same surgical

Table 1. Diagnosis: H25.13 Age-Related Nuclear Cataract, Bilateral (OD/MD)

	Dates of Service		Place of Service	Type of Service	Procedures, Services, Supplies (Explain Unusual Circumstances)	Diagnosis Code	Charges	Days or Units
	From MM/DD/YY	To MM/DD/YY			CPT-HCPCS -Modifier			
1	2/25/2015		11		92004	A	$149.58	1
2	2/25/2015		11		92015	B	$20.03	1
3	2/26/2015		11		76519-26-50	A	$63.16	2
4								
5								
6								

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Comanagement Coding

Example: Billing for 1st Eye

Dr. Jones performs procedure code 66984 on March 1st and cares for the patient through March 2nd. Dr. Smith assumes responsibility for the patient on March 3rd for the remainder of the global period.

Dr. Jones' claim contains:

- 03/01/2015 66984 -54
- 03/01/2015 66984 -55 assumed 03/02/2015, relinquished 03/02/2015

Dr. Smith's claim contains:

- 03/01/2015 66984 -55 assumed 03/03/2015, relinquished 05/30/2015

Diagnosis: H25.12 Age-Related Nuclear Cataract, Left Eye

		Dates of Service		Place of Service	Type of Service	Procedures, Services, Supplies (Explain Unusual Circumstances)	Diagnosis Code	Charges	Days or Units
		From MM/DD/YY	To MM/DD/YY			CPT-HCPCS -Modifier			
Surgeon	1	3/1/2015		11		66984-54-LT	A	XXX.XX	1
	2	3/1/2015		11		66984-55-LT	A	XXX.XX	1
Comanaging Physician	1	3/1/2015		11		66984-55-LT	A	XXX.XX	1

Billing for 2nd Eye

Dr. Jones performs procedure code 66984 on the 2nd eye on May 1st and cares for the patient through May 2nd. Dr. Smith assumes responsibility for the patient on May 3rd for the remainder of the global period.

Dr. Jones' claim contains:

- 05/01/2015 66984 -79 -54
- 05/01/2015 66984 -79 -55 assumed 05/02/2015, relinquished 05/02/2015

Dr. Smith's claim contains:

- 05/01/2015 66984 -79 -55 assumed 05/03/2015, relinquished 07/30/2015

Diagnosis: H25.11 Age-Related Nuclear Cataract, Right Eye

		Dates of Service		Place of Service	Type of Service	Procedures, Services, Supplies (Explain Unusual Circumstances)	Diagnosis Code	Charges	Days or Units
		From MM/DD/YY	To MM/DD/YY			CPT-HCPCS -Modifier			
Surgeon	1	5/1/2015		11		66984-79-54-RT	A	XXX.XX	1
	2	5/1/2015		11		66984-79-55-RT	A	XXX.XX	1
Comanaging Physician	1	5/1/2015		11		66984-79-55-RT	A	XXX.XX	1

date of service and procedure code. For claims where physicians share postoperative care, the assumed and relinquished dates of care must be indicated in Item 19 of the CMS-1500 claim form, or electronic media claim equivalent. When more than one physician bills for the postoperative care, the percentage is apportioned based on the number of days each physician was responsible for the patient's care. The maximum percentage for postoperative care for 66984 is 20%, and the length of the associated global period is 90 days.

The diagnosis for cataract is the most appropriate for clinicians to use for postoperative care. In the ICD-10 system, practitioners should consider categories H25.xxxx, H26.xxxx, H28.xxxx and Q12.0.

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The Postoperative Period: Always 90 Days?

Ninety days is the most common global period for procedures such as cataract surgery. But it can vary depending on the procedure. For example, punctal occlusion has a global period of 10 days. The optometrist responsible for the postoperative period provides all care during this designated time, whether it's 90 days or 10, without billing extra fees.

However, if a patient develops a new medical condition unrelated to the surgery during the postoperative period, that care is not considered part of the postoperative care. This can be billed separately using the -24 modifier for office visits or the -79 modifier for new surgical procedures (such as a foreign body removal).

If a complication of surgery occurs, the care is considered part of the postoperative period and cannot be billed separately. Other procedures such as an OCT would be billable.

The Centers for Medicare and Medicaid Services (CMS) calculates the reimbursement fee for comanagement, generally, at 20% of the total allowed fee for the surgery if care is provided for the entire global period; however, this can vary based on procedure. Otherwise, the reimbursement fee is prorated based on the number of days care is actually performed during the postoperative period. Some private insurers and Medicare Advantage plans may use a different calculation, so check before providing postoperative care.

Farewell to Global Periods

In 2014 CMS proposed the elimination of global periods because the Office of the Inspector General identified a number of surgical procedures that include more visits in the global period than are actually being furnished, thus creating greater costs to the system than are medically necessary. To address this, beginning in 2017, CMS has proposed the inclusion of all services provided on the day of surgery and to pay separately for visits and services furnished after the day of the procedure. So, comanagement isn't ending, but the single global payment and the 20% value assessment is. In 2017 the comanaging physician will provide only the postoperative visits that are medically necessary and bill for them individually.

Comanagement is great opportunity, allowing us to provide great care to our patients beyond our current scope of practice—not to mention collaborate with peers and colleagues. ■

Send questions and comments to ROcodingconnection@gmail.com.

21st Annual Surgery Report

Principles and Protocols of CATARACT COMANAGEMENT

It's our responsibility to care for our patients from diagnosis through the postoperative recovery process. Here's what you need to know.

By Audrey Lu, OD, Paul J. Gruosso, OD, Joseph A. Miller, OD, and Vanessa M. Santos-Nevarez, OD

Cataract patients—and their need for care—are both on the rise, especially as the baby boomer generation approaches retirement age. The 2000 United States census data estimated that roughly 20.5 million Americans older than 40 years of age had a cataract. By 2020, that number is expected to rise 46.8%, to 30.1 million.[1] Plus, the growing diversity and complexity of IOL options adds to the patient's need for guidance, a task particularly well-suited to optometrists.

As primary eye care providers, we are often the first to recognize, diagnose and educate the patient regarding the presence of a cataract. Leading the comanagement effort for these patients is an essential part of our practice modality as well. We establish enduring relationships with our patients and become particularly acquainted with their medical and social histories.

Patients will look to us for guidance from the moment we inform them of the presence of a cataract to the final postoperative follow-up.

This article reviews the basic principles and protocols of caring for this perpetually expanding patient population.

Initial Evaluation

By age 60, most individuals develop some degree of cataract. Our role is to evaluate and diagnose visually significant cataracts and determine the most appropriate treatment plan. A comprehensive examination may include, but is not limited to, elements listed in *Table 1*.

Contrast sensitivity, a glare test or both may be of assistance in quantifying the functional disability that may be far worse than visual acuity measurements indicate.[2]

Potential acuity testing is valuable in determining how much the cataract is contributing to the patient's visual loss. A poor best-corrected visual acuity and a poor potential acuity may reveal that the cataract may not be the sole culprit for the patient's decreased vision and symptoms. Further investigation for other possible causes (e.g., retinal disease or amblyopia) is necessary.

Table 1. Ocular Examination of Cataract Patients[2]

Minimal testing	Recommended ancillary testing
Visual acuity	Contrast sensitivity
Pupillary evaluation	Glare testing
Ocular motility	Potential acuity testing
Visual field screening (confrontations, etc.)	
Refraction	
Intraocular pressure measurement	
Biomicroscopy (undilated and dilated)	
Fundus evaluation	

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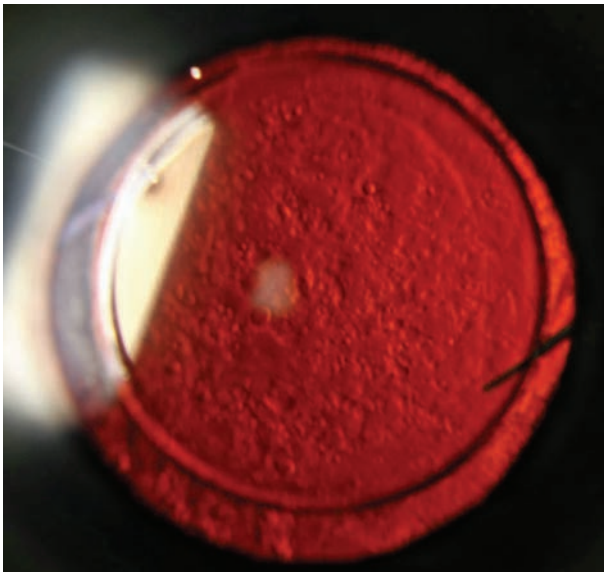
Table 2. Grading of Three Common Types of Cataracts

Cataract Type	Grade 1	Grade 2	Grade 3	Grade 4
Nuclear	Mild	Moderate	Pronounced	Severe
Cortical	Obscures 10% of intrapupillary space	Obscures 10% to 15% of intrapupillary space	Obscures 50% to 90% of intrapupillary space	Obscures more than 90% of intrapupillary space
Posterior Subcapsular	Obscures 3% of the area of the posterior capsule	Obscures 30% of the area of the posterior capsule	Obscures 50% of the area of the posterior capsule	Obscures more than 50% of the area of the posterior capsule

Photo: Aaron Bronner, OD.



Typical posterior capsule opacity, a complication of cataract surgery, as seen with retroillumination.

Diagnosis

Cataracts are frequently graded by visual examination and then assigned numerical values to indicate severity. Alternate grading systems, such as the Lens Opacity Classification Systems (LOCS, LOCSII and LOCSII) use cross-sectional slit lamp photographs as references to aid in grading. The recently introduced LOCSIII system contains a sequence of six reference photos for three types of cataract.[3] Due to the limited availability of reference photos in the everyday clinical setting, clinicians commonly use a modified four-point grading system derived from the LOCSII (*Table 2*).[2-3]

For finer incremental steps, clinicians can add a '+' sign to designate any cataract that falls between grade levels.

Basis for Treatment

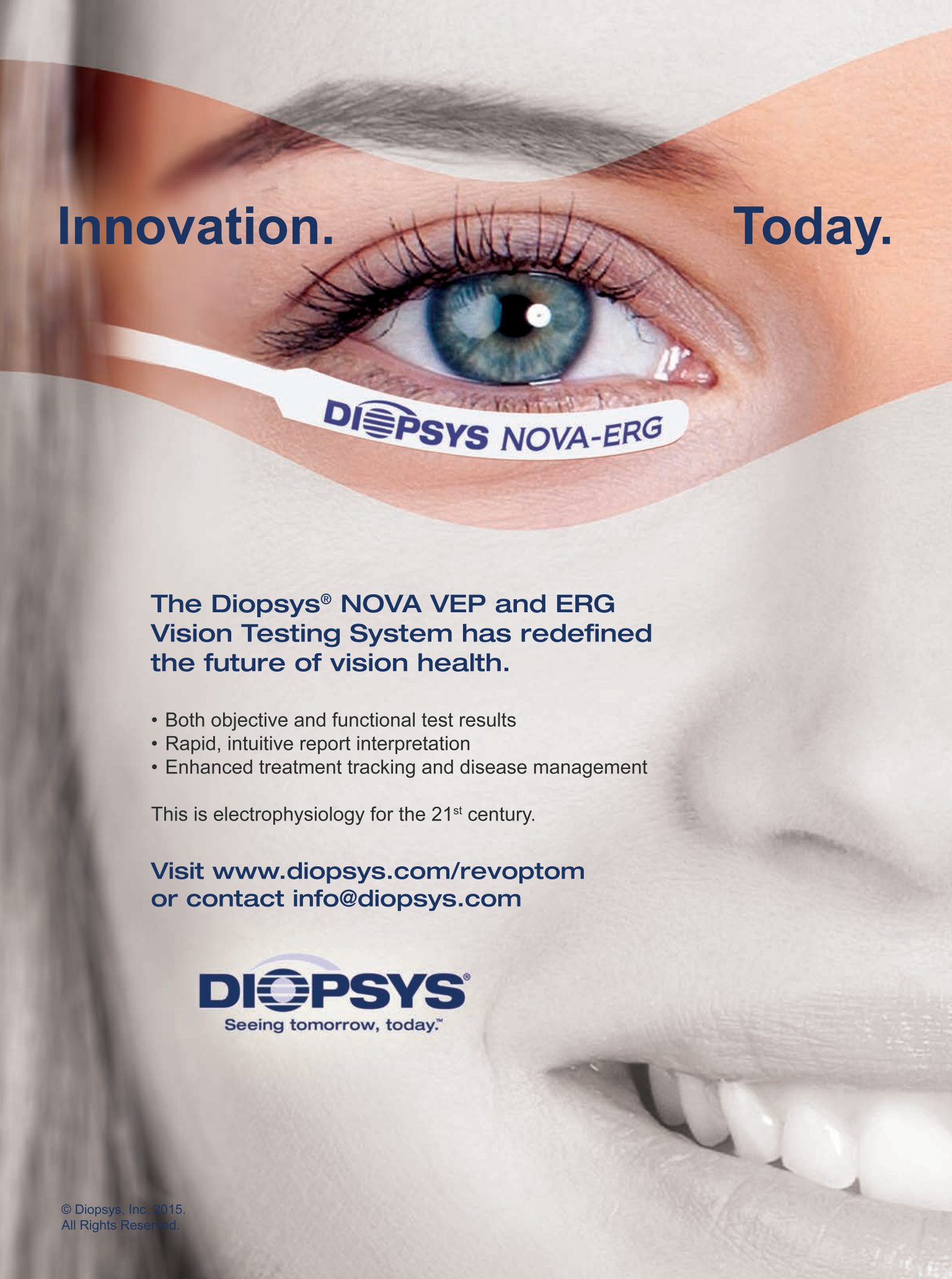
Not all visually significant cataracts require surgical treatment.[2] Therefore, patients with cataracts can be divided into two groups: surgical and nonsurgical. The basis of treatment depends on the extent of the patient's visual disability, symptoms and visual needs. Common symptoms of incipient cataracts include visual blur, reduced contrast sensitivity, perceived color changes and sensitivity to light, glare or both.

Non-surgical Patients

Some patients may appear to be developing cataracts, but are not yet candidates for surgery. The initial treatment for symptomatic cataracts may include updating a patient's prescription (spectacles, contact lenses or both). Patients may also benefit from wearing a wide-brimmed hat and sunglasses with specific tints or filters to increase ultraviolet (UV) protection and reduce glare disability. Tints that selectively filter shorter wavelengths may increase image contrast by reducing light scattering and lens fluorescence. Pink, grey or green filters can decrease the amount of light entering the eye to improve light scatter. Other special lenses, such as the Corning CPF filter 511 and 527, may also help in contrast enhancement and glare reduction.[5]

In addition, advising these patients on appropriate illumination control and lighting sources for reading may give them the tools they need to improve their functional visual ability.[2]

Follow these patients at four- to 12-month intervals to determine whether further vision loss or functional disabilities develop. Such progression may require an updated refraction or consideration for cataract surgery at that time. To ensure patients are aware of their condition's prognosis, it is helpful to educate them on the natural course



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of cataract formation, as well the signs and symptoms associated with progression. Proper patient education is necessary to ensure patients understand the limitations of corrective lenses, which can decrease chair time from refraction checks.

Surgical Patients

Poor visual acuity testing results—or subjective interference in daily function—indicate that it is time for cataract surgery. It should be stressed that functional complaints of daily living are the most important criteria, more so than visual acuity. Ideally, a cataract is ready for surgery if it meets the "Goldilocks criteria," meaning it has to be "just right." Cataracts that cause almost no preoperative decline in visual acuity are at risk for worse postoperative visual acuity.[6,7] Inversely, a hypermature cataract increases the risk of intraoperative complications.[6,7]

Today, the Goldilocks cataract is considered one that has reduced visual acuity to the level that it interferes with the patient's lifestyle and everyday activities, and satisfactory functional vision cannot be obtained with spectacles, contact lenses or other optical aids.

The American Optometric Association (AOA) guidelines divide surgically indicated cataract patients into two groups:

(1) visual acuity of 20/40 or better

(2) visual acuity of 20/50 or worse

The American Academy of Ophthalmology (AAO) guidelines designate a patient as a surgical candidate when visual function no longer meets the patient's needs.[8] There is no specific acuity requirement. Eye care providers must pay extra attention to sort out the 20/40 vision or better surgically indicated group due to the less obvious visual acuity deterioration. These patients often have complaints of decreased vision during specific tasks, complaints of monocular diplopia, large refractive difference between the eyes or a combination of all three. All of these symptoms require further investigation to confirm that the cataract is the primary etiology for the complaints and whether surgery would be beneficial.

Individuals with visually significant cataracts who elect to defer surgery should be advised of the dangers of reduced vision, including increased risk of falls and possibly decreased driving ability. If reduced vision is significant, patients should be educated on whether or not they meet their state's legal requirements for driving. Low vision evaluation would be an option in cases where the cataract progresses but the patient continues to defer surgery or if surgery is not a viable option due to comorbidities.

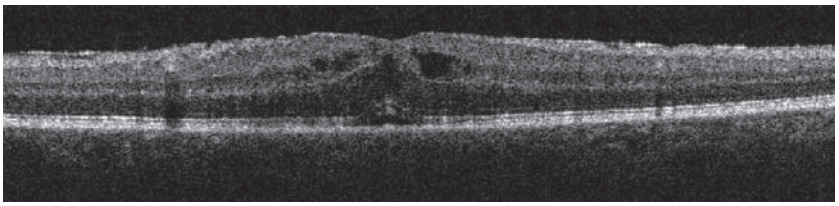

Photo: Aaron Bronner, OD.

Postoperative CME appearing three weeks after surgery. Central macular thickness of 430µm. Patient had self discontinued topical steroid 10 days after surgery.

Table 3. Coexisting Conditions That Contraindicate Cataract Surgery

- Active proliferative diabetic retinopathy
- Rubeosis iridis and/or neovascular glaucoma
- Microphthalmos
- Buphthalmos

Coexisting Conditions and Contraindications

As primary eye care providers, we become closely acquainted with our patients' histories. It is our responsibility to remain attentive to any coexisting ocular conditions (e.g., dry eye, recurrent anterior uveitis, pseudoexfoliation syndrome, glaucoma) and to ensure those conditions are stable prior to referring a patient for surgical consultation.

Patients with conditions that may result in pupillary rigidity or zonular weakness or both, such as pseudoexfoliation syndrome, or have history of ocular trauma should be informed about the higher risk of surgical complications.[8,9] It is also helpful to know the patient's current list of medications, because some potentially increase the risk of surgical complications. For example, Flomax (tamsulosin, Boehringer Ingelheim) has been known to cause intraoperative floppy iris syndrome (IFIS).[8]

Risk of developing pseudophakic cystoid macular edema increases in patients with diabetes mellitus, recurrent anterior uveitis, epiretinal membrane, vitreomacular traction, retinal vein occlusion or a combination of any of these.[10] While these factors are not absolute contraindications, these patients should be counseled regarding possible surgical risks and complications.

Generally, surgical intervention is contraindicated when it will not significantly improve visual function due to the presence of coexisting ocular disease or if the patient is unfit for surgery due to an underlying medical condition.

Table 3 is a list of conditions that

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contraindicate surgery.[10,11] However, under special circumstances, cataract surgery may be necessary to improve visualization of the posterior segment for treatment of coexisting posterior segment disease, even if only minimal visual acuity improvement is expected.

The Postoperative Comanagement Protocol

The postoperative follow-up schedule for a typical cataract patient is generally advocated at one day, one week, one month and two months subsequent to the surgical extraction of the cataract.[4] Conventionally, the surgeon will see the patient the day after the surgery, and the optometrist assumes postoperative care at the one-week or one-month visit. This may vary depending on the comanagement relationship with the surgeon.

In addition, postoperative medications—such as topical antibiotic, steroids and NSAIDs—are given to control postoperative inflammation and prevent infection. Exact medications and dosage vary by surgeon. Here is a summary of the general postoperative guidelines:[2,4]

First postoperative visit:

- Occurs 24 to 36 hours after surgery.
- Examination includes: unaided and aided visual acuity (with pinhole); IOP; slit-lamp evaluation; fundus exam with evidence of especially poor vision or retinal disease.
- Fully instruct patients on their postoperative drop regimen (topical antibiotic, steroids and NSAIDs).
- Review with patient the signs and symptoms of possible postoperative complications that may require emergent care.

Second postoperative visit:

- Seven to 14 days after surgery.
- Examination includes: unaided and aided visual acuity (with pinhole); IOP; slit-lamp evaluation; fundus examination with evidence of especially poor vision or retinal disease.
- If eye is quiescent, consider discontinuing antibiotic drops and starting steroid and NSAID taper (a typical taper schedule is decreasing dosage frequency by one less daily drop each week).
- If inflammation is persistent, topical steroid and NSAID use should be increased accordingly. Once inflammation resolves, begin taper.

Third postoperative visit:

- Three to four weeks after surgery.
- Examination is analogous to previous visit.
- Consider prescribing spectacles if the eye is quiet and stable.

Fourth postoperative visit:

- Six to eight weeks after surgery.
- Examination is analogous to previous, with a dilated fundus examination.
- Perform a refraction test for a final postoperative prescription, if one has not been done earlier.
- Discuss the benefits and importance of UV protection, especially for patients with IOL implants.

Postoperative Complications

The most imperative part of our postoperative care is to determine whether findings and symptoms are typical or anomalous at the postoperative stage. For example, mild cells in the anterior chamber and/or increased IOP are common a few days following surgery and most often resolve on their own with time. However, we must remain vigilant in distinguishing signs of complications that could be detrimental if not promptly diagnosed and treated. Possible postoperative complications are listed by time of typical occurrence in *Table 4*.[2,3,12-14] Here is a brief overview of some postoperative complications:[4,15,16]

Acute Endophthalmitis

Causes: Intraocular inflammation due to direct microbial invasion.

Signs/symptoms: Decreased vision, pain, photophobia, redness, corneal edema, anterior chamber reaction, hypopyon and vitritis.

Management: Immediate referral back to surgeon. Typical treatment is topical fortified vancomycin and tobramycin Q1H for 24 to 48 hours, topical Pred Forte 1% Q1H and atropine 1% TID-QID. More complicated cases may require intravitreal vancomycin, ceftazidime, dexamethasone or pars plana vitrectomy.

Table 4. Onset and Incidences of Potential Postoperative Cataract Complications

Complications	Postoperative Onset	Incidence
Toxic anterior segment syndrome	12 hrs to 24 hours	0.1% to 2%
Anterior uveitis	24 hrs to 14 days	~0.20%
Ocular hypertension	24 hrs to 14 days	0.3% to 10%
Acute endophthalmitis	72 hrs to weeks	0.02% to 0.5%
Pseudophakic cystoid macular edema	one to four months	3% to 5%
Posterior capsule opacity	Months to years	20% to 40%

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Toxic Anterior Segment Syndrome

Causes: Sterile acute postoperative inflammation due to a noninfectious substance entering the anterior segment and inducing toxic damage to the intraocular tissues.

Signs/symptoms: Decreased vision, redness, "limbus-to-limbus" corneal edema, marked anterior chamber reaction and possible hyphema.

Management: This condition usually responds well to topical steroid (prednisolone acetate 1% every one to two hours). Patients must be followed closely to ensure inflammation does not worsen.

Ocular Hypertension

Causes: Increased IOP induced by steroids, IOL irritation or synechial angle closure.

Management: Treatment depends on the severity of the condition. The elevated IOP can be treated with topical aqueous suppressants as long as such agents are not contraindicated. If there is minimal inflammation, the steroid can be quickly tapered. If there is mild inflammation, consider changing to a mild steroid or topical NSAID.

Dropless Cataract Surgery

A recent development may make the traditional postoperative routine simpler for both practitioners and patients. So-called 'dropless' cataract surgery includes the delivery of formulated medications at the end of the surgery by transzonular technique to provide sustained therapeutic protection against endophthalmitis and postoperative cystoid macular edema. The intention is to improve compliance and convenience, as well as reduce postoperative drop cost.[1,2] However, questions of pharmacokinetics compared with that of topical daily drops and concerns of steroid response are still under investigation.

Although dropless cataract surgery has yet to make its way to mainstream management, if the surgeon you refer to performs it, be prepared to discuss the risks and benefits with the patient prior to surgery. The patient must be cautioned about the potential initial blur 24 to 48 hours after surgery due to the medication possibly obstructing the visual axis. At any time, if significant inflammation is present postoperatively, additional topical anti-inflammatory medications may be used. Additionally, patients should be made aware that this medication delivery method may not be covered by insurance.

The postoperative management and follow-up timeline remains the same.

1. Steinert RF. Cataract Surgery. 3rd ed. Philadelphia: WB Saunders; 2004:589-92.
2. Heier JS, Topping TM, Baumann W, et al. Ketorolac versus prednisolone versus combination therapy in the treatment of acute pseudophakic cystoid macular edema. Ophthalmology. 2000;107:2034-9.

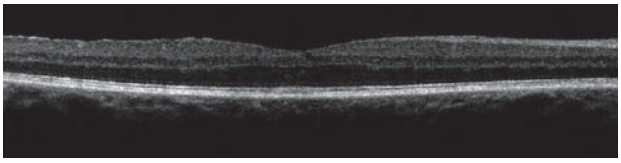

Photo: Aaron Bronner, OD

CME resolving with macular thickness of 300µm after one month of a topical steroid and NSAID.

Pseudophakic Cystoid Macular Edema

Causes: Vessel leakage caused by surgically induced insults, inflammation or both.

Signs/symptoms: Decreased vision, metamorphopsia, scotoma and thickening with or without small intraretinal cysts in the foveal region.

Management: Currently no standardized protocol exists for the prophylaxis and management of pseudophakic CME because of a lack of prospective randomized clinical trials. Therapeutic interventions are based on the proposed pathogenesis of edema, mainly inflammation and vitreous traction.

Posterior Capsule Opacity

Causes: Epithelial cell proliferation and migration, collagen deposition and lens fiber generation.

Signs/symptoms: Decreased vision, opacification of the posterior capsule in the form of fibrosis or pearls.

Management: If functional vision loss is reported, consider Nd:YAG capsulotomy.

Education

Despite the good prognosis, cataract patients may still feel apprehensive when we mention the presence of cataracts and the possible surgical intervention. Therefore, as the patient's primary eye care provider, we are responsible for educating our patients on the natural course of cataracts and determining the treatment option that best fits the individual patient.

As optometrists we have received extensive training and fought hard for the opportunity to care for our patients from initial cataract diagnosis through their final postoperative exam. Surgeons often prefer that the referring optometrist manage the postoperative care, as it increases the time they have available for surgeries. Patients usually prefer to continue care with their optometrist because of their established relationship and the convenience of being seen at the optometrist's office, which is usually closer and offers more flexible scheduling.

For us, it can be financially beneficial to see our patients for postop visits, as we are entitled to a portion of the global fees collected by the ophthalmologist.

If you are currently referring your cataract patients to a practice that does not allow you to do the postop care, we encourage you to express your interest in it. Explain that our scope of practice and training has significantly expanded over the years, allowing you to deliver this highly specialized care.

Our responsibility is to make sure our patient fully understands the process and feels comfortable from the preoperative discussion to the postoperative recovering process. Working closely with the surgeon will help ensure the best possible outcome. ■

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Dr. Santos-Nevarez is a graduate of Salus University Pennsylvania College of Optometry. She completed a Primary Care residency at C.W. Young VA Medical Center, where she is an attending optometrist.

1. Congdon N, Vingerling JR, Klein BE, et al. Prevalence of cataract and pseudophakia/aphakia among adults in the United States. Arch Ophthalmol. 2004;122:487-94.
2. Murrill CA, Stanfield DL, VanBrocklin MD. Optometric Clinical Practice Guideline: Care of the Adult Patient with Cataract. St.Louis, MO: American Optometric Association; 1995.
3. Davison JA, Chylack LT Jr. Clinical application of the Lens Opacities Classification System III in the performance of phacoemulsification. J Cataract Refract Surg. 2003;138-45.
4. Ehlers JP, Shah CP. The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. Philadelphia: Lippincott Williams & Wilkins; 2008.
5. Takeshita B, Wing V, Gallarini L. Corning CPF filters for the preoperative cataract patient. J Am Optom Assoc. 1988;59:793-7.
6. Lundström M, Goh PP, Henry Y, et al. The changing pattern of cataract surgery indications. Ophthalmology. 2015;122.1:31-8.
7. Lundström M, Barry P, Henry Y, et al. Visual outcome of cataract surgery: study from the European Registry of Quality Outcomes for Cataract and Refractive Surgery. J Cataract Refract Surg. 2013;39:673-9.
8. American Academy of Ophthalmology. Cataract in the Adult Eye. Cataract and Anterior Segment Preferred Practice Pattern (PPP) Panel. Accessed June 2, 2015. www.aao.org/preferred-practice-pattern/cataract-in-adult-eye-ppp--october-2011.
9. Kuchle M, Viestenz A, Martus P, et al. Anterior chamber depth and complications during cataract surgery in eyes with pseudoexfoliation syndrome. Am J Ophthalmol. 2000;129:281-5.
10. Noble B, Hayward M. Contraindications to intraocular lens implantation. In: Percival P, ed. Color Atlas of Lens Implantation. St. Louis: Mosby-Year Book; 1991:147.
11. Berontsky DA, Stark WJ, McCartney DL, et al. Changing indications for intraocular lenses: guidelines (legal and ethical) for cataract surgery. In: Caldwell DR, ed. Cataracts: Transactions of the New Orleans Academy of Ophthalmology. New York: Raven Press; 1988:1-8.
12. Javitt JC, Vitale S, Canner JK, et al. National outcomes of cataract extraction: endophthalmitis following inpatient surgery. Arch Ophthalmol. 1991;109:1085-9.
13. Henderson BA, Kim JY, Ament CS, et al. Clinical pseudophakic cystoid macular edema. Risk factors for development and duration after treatment. J Cataract Refract Surg. 2007;33(9):1550-8.
14. Hellinger WC, Hasan SA, Bacalis LP, et al. Outbreak of toxic anterior segment syndrome following cataract surgery associated with impurities in autoclave steam moisture. Infect Control Hosp Epidemiol. 2006 Mar;27(3):294-8.
15. Steinert RF. Cataract Surgery. 3rd ed. Philadelphia: WB Saunders; 2004:589-92.
16. Heier JS, Topping TM, Baumann W, et al. Ketorolac versus prednisolone versus combination therapy in the treatment of acute pseudophakic cystoid macular edema. Ophthalmology. 2000;107:2034-9.

21st Annual Surgery Report

RETINAL CONSIDERATIONS

Prior to Cataract Surgery

Referring patients for cataract removal is common, but when a posterior segment pathology interferes, it must be handled delicately. By Jay M. Haynie, OD

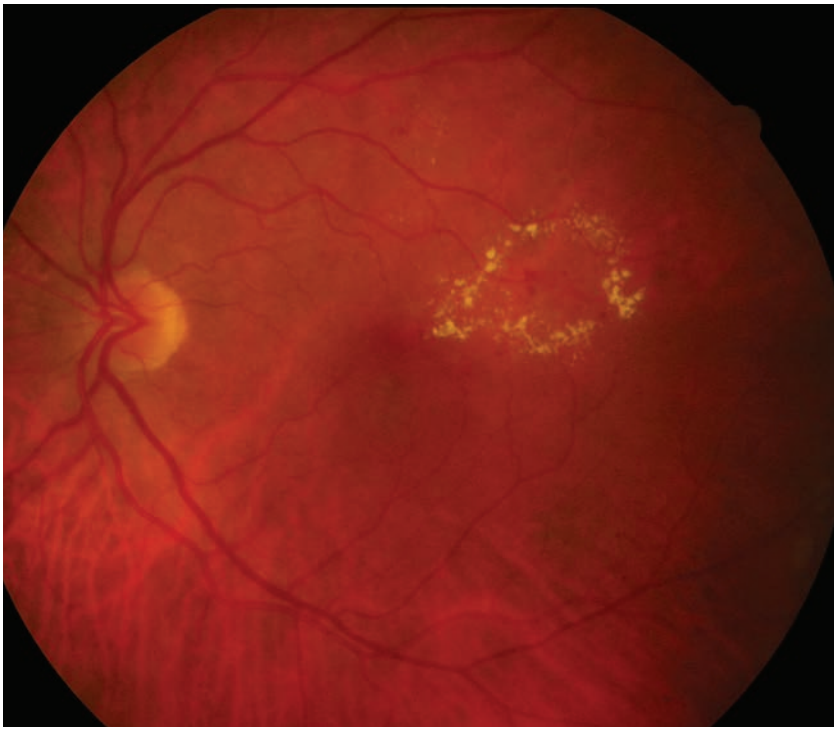
Despite how commonplace cataract surgery has become, postoperative challenges may still occur, particularly when pre-existing retinal pathology is involved.

Multiple retinal conditions may impact cataract surgery outcomes, including disorders such as:

- Diabetic macular edema (DME)
- Retinal vein occlusion (RVO)
- Uveitis
- Epiretinal membrane formation

In addition, peripheral retinal lesions may also have bearing on complications. Peripheral findings that may be relevant to postoperative outcomes include lattice degeneration, retinal breaks, operculated holes, cystic retinal tufts and the whole family of pathologies related to vitreous traction.

Today, imaging tools allow a visual assessment of the posterior pole to a greater degree than ever before. Using this technology, we've been able to increase our understanding of the possible relationship between cataract surgery and some retinal pathologies.



This patient has non-proliferative diabetic retinopathy with clinically significant macular edema.

This article will outline the more common conditions associated with a higher risk of postoperative complications of cataract surgery.

Epiretinal Membranes

Epiretinal membranes are common in potential cataract surgery patients.[1] The Blue Mountains Eye

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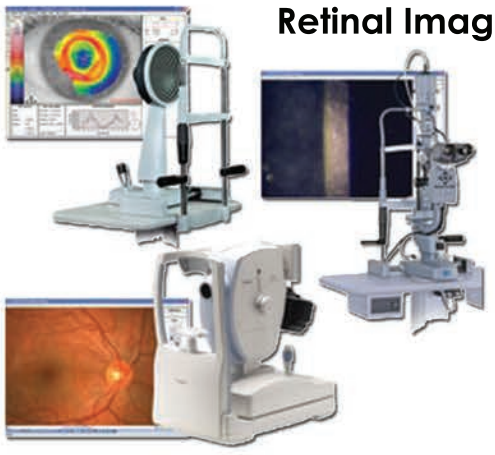

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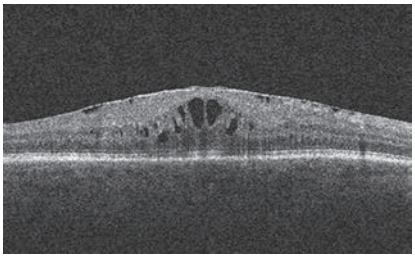

OCT reveals epiretinal membrane with cystoid macular edema.

Study shows the prevalence of an epiretinal membrane varies—estmating that 7% of patients older than 49 years of age will have an epiretinal membrane, 31% of which are bilateral.[1] The incidence increases with age, as follows: [1]

- younger than 60.....1.7%
- between 60 and 69..... 7.2%
- between 70 and 79.....11.6%
- 80 and older..... 9.3%

Some membranes are easily seen clinically whereas others are not. Spectral-domain OCT (SD-OCT) has allowed clinicians to visualize the anterior retina and the vitreoretinal interface with much greater detail than fundus photography and, in some instances, better than the clinical exam.

Cystoid macular edema (CME) is one complication of epiretinal membrane.[2] It is important to identify macular edema when considering a patient for cataract surgery. Because the risk of post-cataract CME is increased in patients with an epiretinal membrane, any macular edema should be treated to establish some level of stability prior to clearing a patient for surgery.[2] Although treatment of macular edema secondary to epiretinal membrane may not improve visual symptoms, it may reduce the risk of postoperative exacerbation.[2]

Retinal Vein Occlusions

Retinal vein occlusion (RVO) is the second most common retinal vasculature disease after diabetic retinopathy.[3] When considering cataract surgery in a patient with a history of RVO, be sure the patient is completely free of retinal ischemia and check whether any macular edema is present. Fluorescein angiography (FA) is the standard diagnostic test for identifying capillary drop out and retinal ischemia. It should be performed on patients with an RVO, especially if the RVO is associated with central vision loss, disc edema, multiple hemorrhages, cotton-wool spots or macular edema.

Using SD-OCT, clinicians can evaluate the degree of macular edema through retinal contour maps, follow the response of treatment and adjust accordingly.

The goal of treatment is to suppress the edema; however, many patients with a retinal vein occlusion will require months of treatment. Intravitreal agents Lucentis (ranibizumab, Genentech), Eylea (aflibercept, Regeneron) and Ozurdex (dexamethasone, Allergan) are currently FDA approved for the treatment of macular edema secondary to a retinal vein occlusion. Avastin (bevacizumab, Genentech), although not FDA approved for use in the eye, is still a common first-line agent.

When considering cataract surgery in a patient with a history of a retinal vein occulusion, it is best to wait until the macular edema has resolved and remains stable for two to three months without intervention, unless the cataract precludes visualization of the posterior pole. Investigators have demonstrated that the risk of postsurgical cystoid macular edema in a patient with a history of a RVO is up to 30 times higher, and the risk persisted even in eyes without preoperative macular edema.[2]

Age-related Macular Degeneration

AMD remains the leading cause of legal blindness in patients older than 65, despite efforts to identify these individuals early in the course of the disease.[4] Cataracts are the leading cause of reversible blindness worldwide and, ultimately, removal of a cataract will improve visual function whether or not the patient has AMD.[4]

The ongoing debate is whether uncomplicated cataract surgery can contribute to AMD progression. Some studies demonstrate that cataract surgery can improve visual function in the AMD patient in the short-term, but others associate cataract surgery as a risk factor for wet AMD in the long-term.[5-7] As a result of conflicting studies and the inability to predict which patients are at high risk, practitioners are left to make a clinical judgment based on the limited data available.

The major protective filter to near ultraviolet radiation (300nm to 400nm) is provided by the crystalline lens, which becomes cloudy with cataract formation. Investigators theorize that this protects the retina against blue light in the visible spectrum and that a cataract may actually be somewhat protective against the development of AMD, making removal of the cataract a potential risk factor for AMD development.[8] One study evaluated progression of AMD after unilateral cataract surgery in patients with bilateral symmetric findings of AMD. Exudative AMD developed in 19% of eyes that underwent cataract surgery compared with 4% of unoperated eyes.[9] For this reason, investigators suggest reserving cataract surgery in AMD patients for those with reduced visual function. They also suggest close follow up of such

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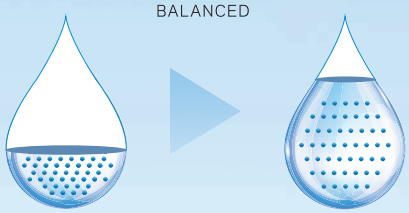

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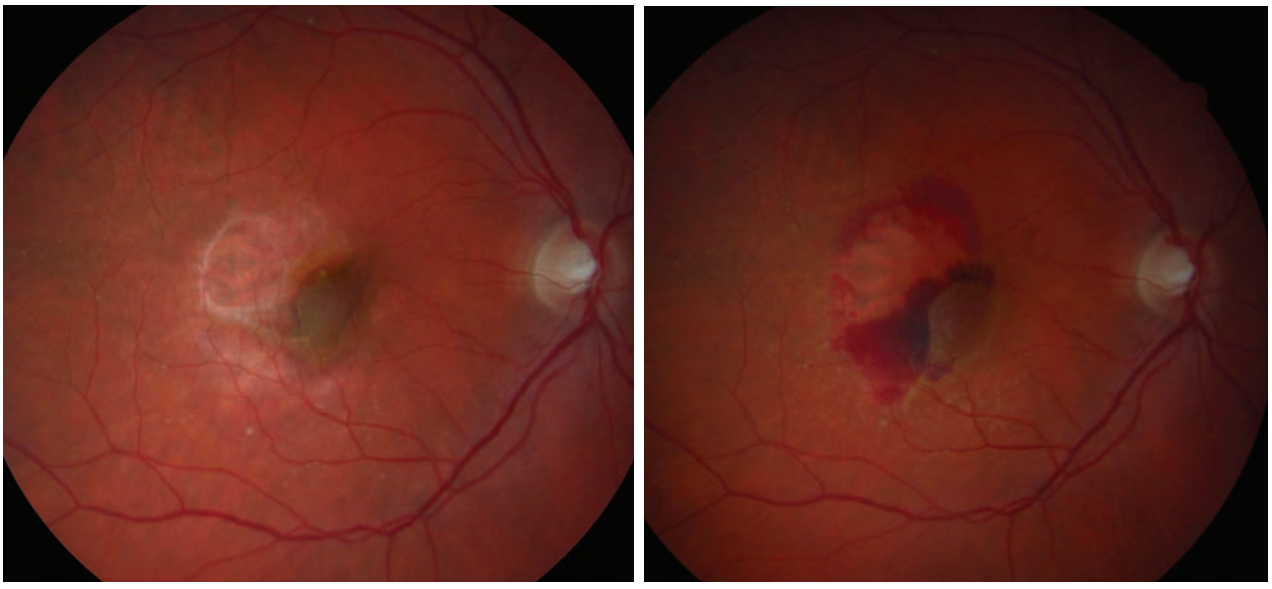

At left, a fundus image of wet AMD post-injections and pre-cataract surgery. At right, wet AMD eight weeks post-cataract surgery with recurrent activity.

eyes.[6] A conflicting report concluded that cataract surgery did not increase the risk of developing exudative AMD.[4]

In contrast to potential exacerbation of AMD, we can look at the other side of the discussion and consider a gain in visual function by removing the cataract. Several studies conclude that by removing the cataract in an AMD patient, both visual function and quality of life were significantly improved.[5,6]

When considering cataract surgery in a patient with AMD, begin with an assessment of visual function objectively (e.g., snellen acuity, potential acuity, amsler grid testing, dark adaptation) as well as subjectively (e.g., night glare symptoms, light/dark adaptation complaints, or metamorphopsia). Then determine if the cataract itself is contributing to impaired visual function enough to consider the assessed risk of AMD progression.

Regarding patients with dry AMD, use an OCT to evaluate the integrity of the RPE, the size of drusen or pigment epithelial detachment present, and to rule out any cystoid macular edema or subretinal fluid that may not be clinically visible. Counsel patients with high-risk characteristics—such as soft, confluent drusen or the presence of a pigment epithelial detachment—on the increased risk of progression and follow them closely for the first 12 months postoperatively.

When considering cataract surgery in a patient with prior wet AMD, you may suspect the risk of progression exceeds the risk of non-exudative AMD. However, the literature shows otherwise; it does not necessarily imply a causal link, and the timing of cataract removal may be of even greater consequence when dealing with patients who have a history of exudative AMD.[10] Patients with exudative AMD are treated with anti-VEGF compounds monthly for an initial three months, and the retreatment is based on a PRN dosing vs. a treat-and-extend protocol. Although no concrete recommendation for the timing of cataract surgery exists, in my clinical experience it is commonplace to defer cataract surgery until the patient has not required intervention for a 90-day period. As with dry AMD, patients should be followed more closely during the postoperative period compared with a person without AMD. You can follow them using serial OCT images, but, in the event they develop CME or subretinal fluid, promptly provide a referral to a retina specialist.

With AMD, recommendations for cataract removal remain unclear, and oftentimes it becomes a personal decision made by the patient after you have discussed with them the risk factors associated with surgery and progression of AMD. Some features of exudative AMD—including an occult choroidal neovascularization (CNV), which is an under-recognized feature of AMD—may have few clinical findings. These findings include hemorrhage, edema or lipid seen with ophthalmoscopy, especially through a moderate to dense cataract. For these patients, SD-OCT is particularly helpful to

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References: 1. Data on file. 2. A total of 324 eye care practitioners asked Clear Care patients to use PeroxiClear for at least 7 days resulting in 1,447 responses to an online survey. The results of these survey questions are accurate at a 95% confidence level to + or – 2% points.

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identify cystoid macular edema or subretinal fluid that would be consistent with wet AMD, and treatment would be advised prior to removal of a cataract. Data from the AREDS study concluded there is a five-year progression risk of 12% to 50% in eyes with intermediate AMD, depending on pigment changes in addition to bilateral large-sized drusen.[11] In addition, efforts are currently being made to determine high-risk patients, based on SD-OCT biomarkers.

Diabetic Macular Edema

More than 300 million people worldwide are affected by diabetes and related complications, including diabetic retinopathy and DME, which are the leading cause of visual impairment in working-aged Americans.[12] Diabetes also increases the probability of developing a cataract and may contribute to an increased risk of reduced visual outcomes after cataract surgery.[12,13] The preoperative work-up of a patient with diabetic retinopathy, DME or both should include careful examination of the posterior segment with or without fluorescein angiography. Consider SD-OCT imaging for all patients with reduced acuity not consistent with the degree of lens opacity. It should also be applied for all diabetic patients with microaneurysms or clinically significant macular edema, based on the established criteria defined by the ETDRS. This criteria includes:

1. Retinal thickening at or within 500µm of the foveal center.
2. Hard exudate at or within 500µm of the center of the fovea with adjacent retinal thickening.
3. A zone of retinal thickening one disc area in size within one disc area of the center of the fovea.

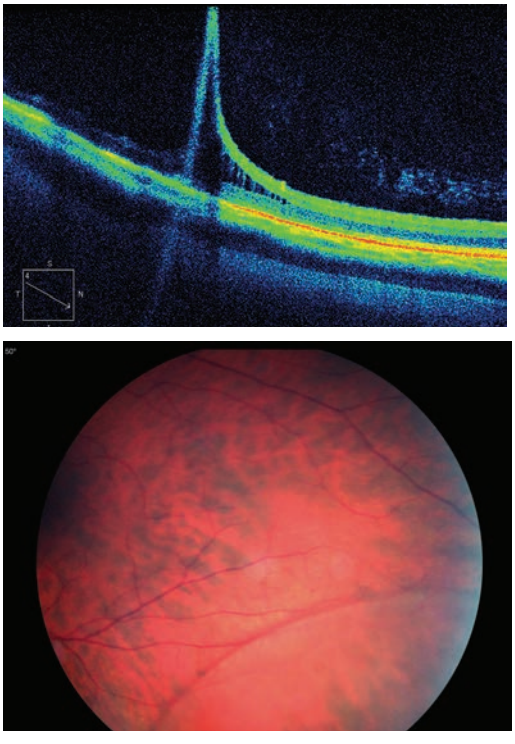
One study demonstrated the incidence of developing center-involved macular edema in patients with no DME prior to cataract surgery was 0% at 16 weeks postoperatively. Eyes with a history of diabetic macular edema had different incidences of center-involved macular edema post-cataract surgery, depending on history of treatment. In patients with no history of treatment for DME, the incidence was 4%, whereas patients with a history of DME treatment had an incidence of 21% of developing center-involved edema.[14]

Consider SD-OCT imaging in all patients with diabetic retinopathy, and certainly those with macular edema, prior to cataract surgery to determine risk of complications.[14] If center-involved edema is detected, refer the patient to a retina specialist. Postoperative care for the diabetic patient may require additional visits and, for those with a history of DME, serial SD-OCT images.

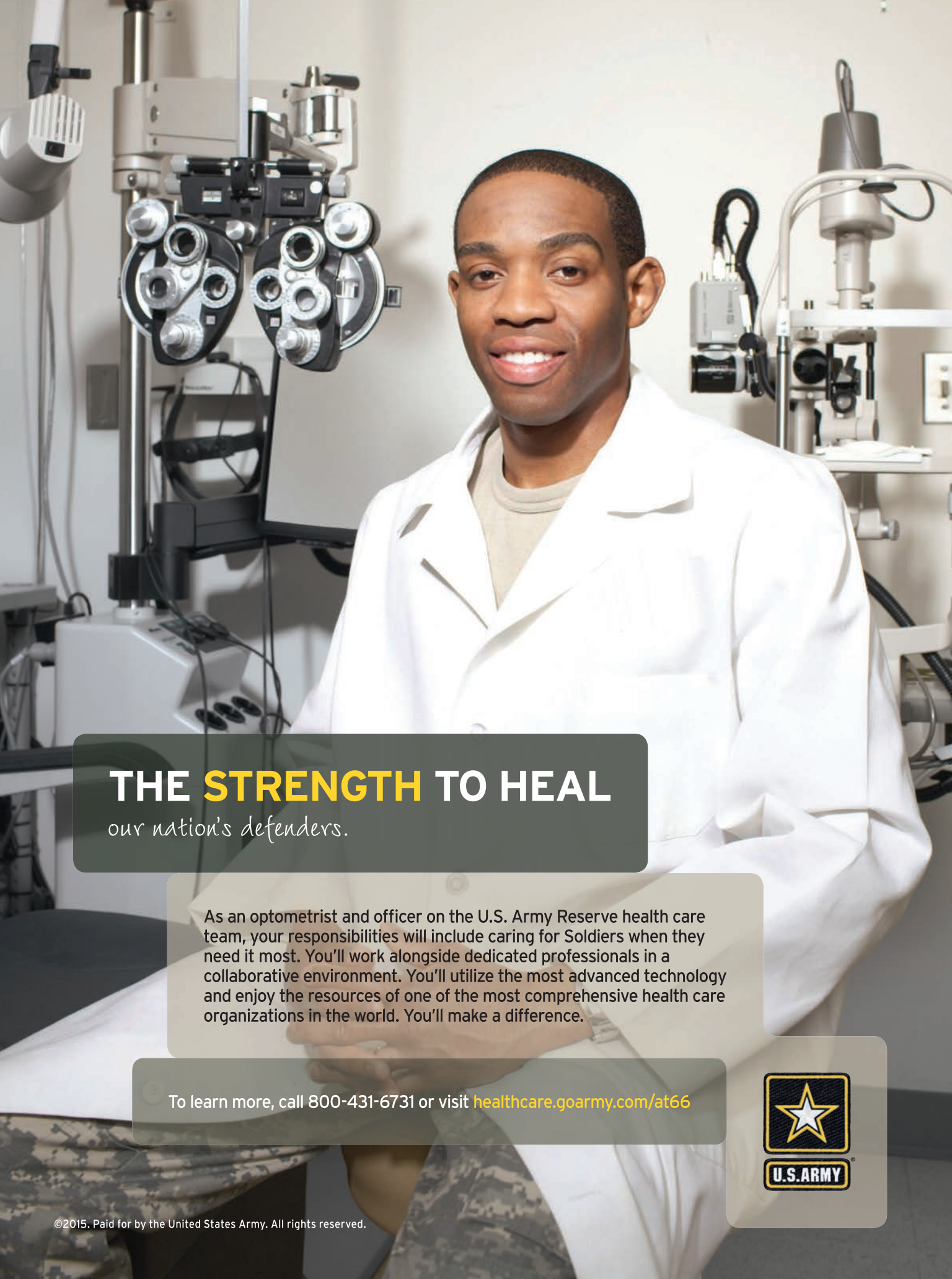
Peripheral Retinal Degenerations

Lattice degeneration is present in 7% to 8% of the general population, and of those cases up to 45% are bilateral.[15] Lattice degeneration is associated with an increased risk of a retinal detachment—the incidence of which is about 1% in a person's lifetime.[16] When a patient with lattice degeneration is considered for cataract surgery, one management option includes prophylactic treatment. Given the low incidence of retinal detachment, it is unnecessary to treat all patients with lattice degeneration prophylactically. Atrophic holes associated with lattice degeneration increases the risk of retinal detachment to approximately 2% if asymptomatic prophylactic treatment has not been recommended.[17] However, a consideration for treatment is made if the patient has symptoms of vitreous traction (e.g., flashing lights), the fellow eye has a history of retinal detachment or if the patient has a positive family history of a retinal detachment.

Cystic retinal tufts are chalky white in color, round or oval in shape and primarily composed of glial tissue. Their incidence in autopsy cases is 5%, with 20% of those presenting bilaterally.[18] Despite being associated with 6.5% to 10% of non-traumatic retinal detachments, the risk of a retinal



OCT scans allow practitioners to see changes in the peripheral retina, such as the retinoschisis in the top image. Below, a fundus image of bullous retinoschisis.



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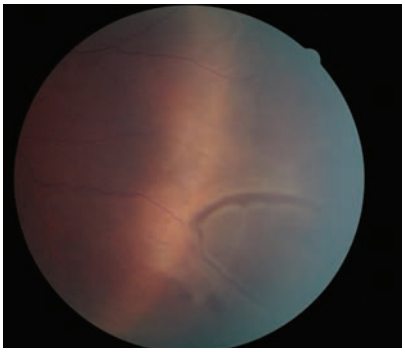
detachment in cystic retinal tuft patients is quite low and estimated to be only 0.28%.[18] Prophylactic treatment of cystic retinal tufts is not necessary in cataract patients.

Symptomatic retinal breaks are associated with photopsia or an increase in floater symptoms. In a patient with a symptomatic posterior vitreous detachment (PVD), the risk of a retinal break is 10% to 15% and, if vitreous hemorrhage is present, the risk increases to 70%.[19,20] Acute symptomatic horseshoe tears may progress to a retinal detachment in 30% to 50% of cases, and prophylactic treatment with laser photocoagulation or cryotherapy can reduce the risk of progression down to 5%.[21,22] In contrast to horseshoe tears, symptomatic operculated breaks do not progress to a retinal detachment unless residual vitreous traction is seen on adjacent retinal tissue. Although this can be difficult to assess clinically, examination with scleral depression is useful. Treatment for operculated tears in the absence of intact vitreous traction is not advised prophylactically.

The peripheral retina is typically examined with an indirect ophthalmoscope as part of the evaluation prior to cataract surgery. Scleral depression may be part of such examination. While challenging to perform in some cases, scleral depression may provide additional information in that retinal findings may be viewed dynamically.

When evaluating a cataract patient who has peripheral retinal degeneration or retinal breaks, symptomatic or not, it is always a good idea to get surgical clearance from a retinal specialist.

Advances in imaging technology have also created ways to assess the peripheral retina before and after cataract surgery. Widefield photography and SD-OCT imaging allow a practitioner to identify, document, and follow retinal pathology.



Horseshoe tears, such as this one, may progress to a retinal detachment.

The OCT image on page 60 documents the presence of a peripheral retinoschisis in a phakic patient. The character of the retinal layer separation is clear, and it will be helpful to know this if the patient is found to have any change after cataract surgery.

While the peripheral retina may be studied with these imaging methods, other areas of the posterior pole, especially the macula, may be evaluated as well. OCT scanning in particular has greatly expanded our understanding of the macula in its relation to disease. This has allowed for our increased ability to evaluate the macula in patients before and after cataract surgery.

Fortunately, in most instances patients who undergo cataract surgery have satisfactory results. While it is true that eye care practitioners need to be mindful of retinal conditions that may complicate cataract surgery, it is also worth noting the solid benefits that most achieve. With this in mind, ODs can move forward with the tools and knowledge that best serve those who need cataract surgery—even retina patients. ■

Dr. Haynie is the executive clinical director at Retina & Macula Specialists, with office locations in Tacoma, Renton, Olympia and Kennewick, Wash. He is a member of Thrombogenics' advisory board.

1. Mitchell P, Smith W, Chey T, Wang JJ, Chang A. Prevelence and associations of epiretinal membranes. The Blue Mountains Eye Study, Australia. Ophthalmology. 1997 June;104(6):1033-40.
2. Henderson B, Kim J, Ament C, et al. Clinical pseudophakic cystoid macular edema-Risk factors for development and duration after treatment. J Cataract Refractive Surgery. 2007;33:1550-8.
3. Farah SE. The impact of cataract surgery on preexisiting retinal disease. EyeNet Magazine. 2010 September.
4. Arevelo J, Garcia R, Sanchez J. The AMD/Cataract Surgery Connection. Review of Ophthalmology. 2005 December.
5. Shuttleworth GN, Luhishi EA, Harrad RA. Do patients with age related maculopathy and cataract benefit from cataract surgery? Br J Ophthalmology. 1998;82:611-6.
6. Armbrecht A, Findlay C, Aspinall P, et al. Cataract surgery in patients with age-related macular degeneration: one-year outcomes. J Cataract Refractive Surgery. 2003;29:686-93.
7. Wang J, Klein R, Smith W, et al. Cataract surgery and the 5-year incidence of late stage age-related maculopathy: pooled findings from the Beaver Dam and Blue Mountains eye studies. Ophthalmology. 2003;110:1960-7.
8. Smith B, Belani S, Ho A. Light energy, cataract surgery, and progression of age-related macular degeneration. Curr Opin Ophthalmol. 2005;16:166-9.
9. Pollack A, Marcovich A, Bukelman A, Oliver M. Age-related macular degeneration after extracapsular cataract extraction with intraocular lens implantation. Ophthalmology. 1996;103:1546-54.
10. Freeman EE, Munoz B, West SK, et al. Is there an association between cataract surgery and age-related macular degeneration? Data from three population-based studies. Am J Ophthalmol. 2003;135:849-56.
11. Age-Related Eye Disease Study Research Group. A randomized, placebo-controlled, clinical trial of high-dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and vision loss: AREDS Report No. 8. Arch Ophthalmology. 2001;119(10);1417-36.
12. Klein B, Klein R, Moss S. Incidence of cataract surgery in the Wisconsin Epidemiolgic Study of Diabetic Retinopathy. Am J Ophthalmol. 1995;119(3):295-300.
13. Klein B, Klein R, Moss S. Prevelence of cataracts in a population-based study of patients with diabetes mellitus. Ophalmology. 1985;92(9):1191-6.
14. Baker C, Almukhtar T, Bressler N, et al. Macular edema after cataract surgery in eyes without pre-operative central-involved diabetic macular edema. JAMA Ophthalmology. 2013 July;131(7):870-9.
15. Foss R, Simons K. Vitreous in lattice degeneration of retina. Ophthalmology. 1984;91:452-7.
16. Wilkes S, Beard C, Kurland L, et al. The incidence of retinal detachment in Rochester, Minnesota, 1970-1978. Am J Ophthalmology. 1982;94:670-3.
17. Byer NE. Long-term natural history of lattice degeneration of the retina. Ophthalmology. 1989;96:1396-401.
18. Byer N. Cystic retinal tufts and their relationship to retinal detachment. Arch Ophthalmology. 1981;99:1788-90.
19. Linder B. Acute posterior vitreous detachment and its retinal complications. Acta Ophthalmology. 1966;87:1-108.
20. Benson WE. Retinal detachment: Diagnosis and Management, 2nd ed. Philadelphia: Harper & Row Publishers Inc, 1988: 4.
21. Davis MD. Natural history of retinla breaks without detachment. Arch Ophthalmology. 1974;92:183-94.
22. Colyear BH Jr, Pischel DK. Clinical tears in the retinal without detachment. Am J Ophthalmology. 1956;41:773-92.

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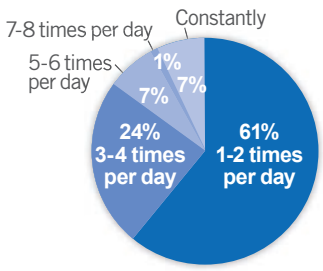

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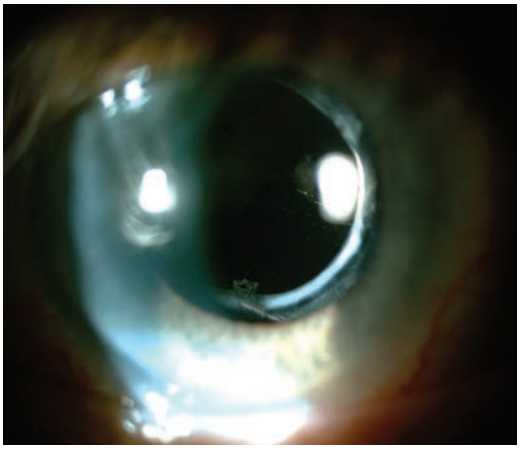
21st Annual Surgery Report

Can't Get No Satisfaction: Post-Cataract Surgery Dysphotopsias

Although they are little, they can cause a lot of trouble for cataract patients. Here's what you need to know. **By Gleb Sukhovolskiy, OD**

Today's cataract surgery is extremely successful at improving patients' vision and quality of life. Yet, a small percentage of patients remain dissatisfied after the procedure, even if it results in 20/20 visual acuity. One study found the primary cause of post-surgical dissatisfaction in a normal pseudophakic population was dysphotopsias—virtually unknown two decades ago.[1]



Introduction of the acrylic intraocular lens material helped to significantly reduce the rate of posterior capsular opacification after cataract surgery.

Dysphotopsias are still only vaguely familiar to most optometrists; however, with their incidence on the rise, it is important for us to be able to comanage dysphotopsia cases effectively with surgeons when intervention is necessary.

The term *dysphotopsia* is used to describe a variety of visual symptoms that result from light reflecting off the intraocular lens (IOL) onto the retina.[2] Dysphotopsias are generally divided into two categories: positive and negative. Positive visual changes involve symptoms of bright artifacts, while negative dysphotopsias are perceived as shadows or dark areas in the visual field.[3] Patients may report glare, starbursts, halos or shadows when describing their visual symptoms.

It's difficult to estimate the prevalence of dysphotopsias. Studies report a range from as low as 1.5% to as high as 67% for positive dysphotopsias, with most data showing more moderate numbers of 12% to 35%).[4-6] Negative dysphotopsias are less prevalent and are thought to occur in only 0.5% to 2.4% of patients.[5,7] In the vast majority of cases, the symptoms subside several weeks after surgery due to the process of neuroadaptation. Neuroadaptation can occur in response to an unwanted monocular or binocular visual disturbance.[8] Inherent neural plasticity of the brain helps the visual cortex negate the effect of an undesirable pattern.[8,9] This may explain why long-term significant effects are only observed in a small percentage of the pseudophakic population.

IOL Evolution

To understand the increase in the incidence of positive dysphotopsias, it is important to consider the evolution of IOLs. The first commonly accepted intraocular lens material was polymethyl methacrylate (PMMA), which usually coincided with a rounded-edge lens design. Advantages of PMMA material

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included low cost and durability inside the eye.[10] PMMA lenses cause little or no dysphotopsias, which is supported by the fact that dysphotopsias were virtually unknown when PMMA was the IOL material of choice.[2] These lenses are rarely used today due to the material's inability to fold, requiring a large incision during surgery, and a high rate of posterior capsular opacification (PCO) due to edge design.[11]

Because larger incisions increase the risk of infection and iris complications, and researchers found that the rounded-edge design is a prominent risk factor for PCO, flexible acrylic and silicone materials with vertical, sharp-edged designs were introduced, with great success. Because rising evidence indicates that IOLs with sharp edges result in lower rates of PCO than those with round edges, sharp-edged lens designs are now used more frequently than round-edged designs.[12]

Acrylic lens material redefined cataract surgery with its ability to fold and fit through small incisions, while also greatly reducing the rate of PCO.[13,14] Because of these advantages, acrylic IOLs are the most commonly implanted lenses in the United States today. However, acrylic lens materials has created a subsequent trend of increased incidence of dysphotopsias.[2]

Positive Dysphotopsias

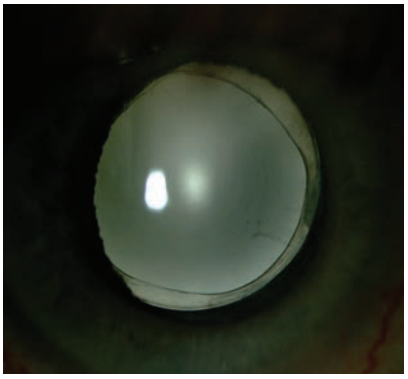
Several studies evaluated the role of IOL material in the formation of positive dysphotopsias. Investigators speculate that positive dysphotopsias are caused by stray light projecting onto the retina, which worsens if that stray light is concentrated in one particular area. One study looked at the light reflected from the surfaces of IOLs and found that when the initial light hits the retina and scatters out of the eye, some of it gets reflected from the posterior surface of the IOL back onto the retina.[15] The researchers found that materials with higher refractive indices help to concentrate a larger amount of light onto a smaller area of retina, resulting in symptoms. Acrylic lenses typically have a higher refractive index than PMMA or silicone lenses, further supporting the researchers' findings.

Another study argues that the increased surface reflectivity of acrylic lenses causes more symptoms compared with silicone or PMMA lenses.[16] The reserchers supported their claim by replacing acrylic IOLs of eight symptomatic patients with silicone or PMMA IOLs, which alleviated symptoms of dysphotopsia. Other case reports have similar findings, but no large-scale studies have been done.[4,16]

In addition to lens material, IOL edge design has also been implicated in the development of positive dysphotopsias.[17] Dysphotopsias were a rare occurrence when round-edged designs were used, but an industry move toward sharp edges resulted in higher incidences of dysphotopsias. Using computer analysis, researchers found that both sharp- and round-edged IOLs produce stray light, but only the sharp-edged design concentrated the rays of stray light into an arc on the retina, causing symptoms.[18] Round-edged designs spread the light more uniformly throughout the retina, minimizing the effect.

Negative Dysphotopsias

Negative dysphotopsia is a much less studied and understood visual complication than positive dysphotopsia. Patients usually complain of a dark shadow in the temporal visual field. It typically manifests after in-the-bag posterior chamber IOL implantation.[19] Numerous theories attempt to identify a cause for negative dysphotopsia; suspects include IOL parameters and optics, corneal incision scars, anterior capsulotomy edge involvement, and distance of IOL from the iris.[7,16,17,20]



It's still unclear if anterior capsular fibrosis increases or reduces negative dysphotopsias. Some clinicians see it as the cause, and use YAG capsulotomy to relieve patients' symptoms. Others believe that increased opacification and translucency of the lens capsule helps to scatter light into the region of the retina in which the shadow is formed.

One of the most promising studies used computer software to simulate negative dysphotopsias in normal eye models.[19] Researchers found that shadows formed in the peripheral retina when light rays underwent changes at the posterior sharp edge of the lens. Computer analysis showed the formation of ring-patterned scotomas, which would be apparent only temporally on a patient's visual field, as the nose blocks the nasal aspect.

The researchers determined that several optical factors are required for the formation of negative dysphotopsias.[19] The most significant of those factors are: a small pupil; a placement of an acrylic lens at a distance of more than 0.06mm but less than 1.23mm behind the pupil; a sharp-edged design; and a



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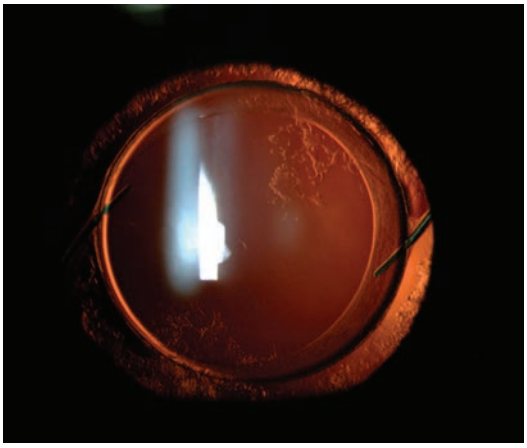
functional nasal retina that extends anterior to the shadow. Secondary factors include a high index of refraction of IOL material and the nasal location of the pupil relative to the eye's optical axis.[19]

Several different treatments to reduce negative dysphotopsia have been attempted. Investigators reported that an IOL exchange with a reduction of iris-IOL distance helped reduce symptoms of negative dysphotopsia.[20] Similar findings were observed in another study, which found that exchanging an in-the-bag IOL for a sulcus-fixated lens resulted in resolution of symptoms in five eyes of five different women.[21] Research also shows that reverse optic capture and secondary piggyback IOL implantation can resolve negative dysphotopsia symptoms.[22]



In-the-bag IOL with mild PCO. Some surgeons improved certain patients' negative dysphotopsias by replacing an in-the-bag IOL with a sulcus-fixated IOL. Negative dysphotopsia symptoms were never eliminated simply by placing a different IOL inside the capsular bag in place of the original.

Several studies report partial or complete resolution of symptoms after YAG laser anterior capsulotomy of the nasal portion of the anterior capsule.[23,24] This evidence disagrees with an earlier study's hypothesis that eventual opacification and translucency of the anterior capsule helps reduce shadow perception by scattering light into that region of the retina.[19] Compared to other studies, this explanation addresses why the incidence of negative dysphotopsias decreases drastically two to three years after surgery. Negative dysphotopsias are still poorly understood, and more studies are needed on this topic.

Management

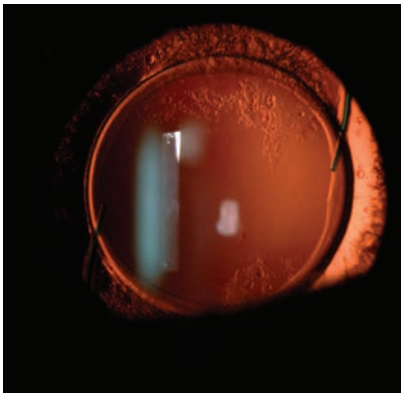
Dysphotopsias are the primary source of patient dissatisfaction after cataract surgery.[1] In most cases they diminish with time, but some patients have severe long-term symptoms.[7] It is important for optometrists to recognize dysphotopsias, as we are integral to patient education and comanagement with a surgeon. Case history should be conducted very carefully, beginning with open-ended questions. Probing for specific symptoms may elicit a positive response by certain patients, even in the absence of significant problems. Hyper-focusing on those problems may make symptoms subjectively more bothersome to patients.

It is vital to understand that cataract surgery should not be regarded as a "cookie-cutter" treatment that can solve all visual problems of all patients. Individual eyes may respond to surgery in a unique way—some of them by developing visually significant dysphotopsias.

There is currently no agreed-upon management strategy for positive dysphotopsia symptoms. Eye care providers should educate patients that in most cases, they subside after the initial postoperative period, and visual disturbances should disappear without further treatment.[7]

If severe symptoms persist after four to six weeks, intraocular lens exchange can be considered; however, it should be the last resort. Timing is important when considering an IOL exchange, and earlier intervention may be easier for the surgeon and involves less risk for the patient. Haptics of certain lenses may become fibrosed or start eroding through the edge of the capsule into the sulcus, even several weeks post-surgery.[25] Intraocular lenses may also develop a strong adherence to the capsule, making it difficult for the surgeon to dissect it from the capsular bag.[26] If IOL exchange is done soon enough after the initial cataract extraction, the surgeon may use the original clear corneal incision.[25] Note that if an intraocular lens exchange is considered, YAG capsulotomy should be avoided, as the open posterior capsule requires a vitrectomy during the IOL exchange procedure.[25]

Some patients with severe negative dysphotopsias have found relief



Intraocular lens exchange may be difficult months or years after the original cataract surgery, as haptics of certain lenses may erode through the edge of the capsule and their manipulation may damage the zonules.

from IOL exchange with reverse optic capture, sulcus fixation and piggyback IOL insertion.[20-22] YAG anterior capsulotomy has also been shown to help resolve symptoms of negative dysphotopsias.[23,24]

Much is still to be studied in this area of cataract surgery, but awareness of these complications is important, as they can cause significant patient dissatisfaction. ■

Dr. Sukhovolskiy is a resident at the Pacific Cataract and Laser Institute in Kennewick, Wash.

1. Kinard K, Jarstad A, Olson R. Correlation of visual quality with satisfaction and function in a normal cohort of pseudophakic patients. J Cataract Refract Surg. 2013;39:590-7.
2. Schwiegerling J. Recent developments in pseudophakic dysphotopsia. Curr Opin Ophthalomol. 2006;17:27-30.
3. Hood CT, Sugar A. Subjective complaints after cataract surgery: common causes and management strategies. Curr Opin Ophthalmol. 2015;26:45-9.
4. Ellis MF. Sharp-edged intraocular lens design as a cause of permanent glare. J Cataract Refract Surg. 2001;27:1061-4.
5. Meacock WR, Spalton DJ, Khan S. The effect of texturing the intraocular lens edge on postoperative glare symptoms: a randomized prospective, double-masked study. Arch Ophthalol. 2002;120:1294-8.
6. Tester R, Pace NL, Samore M, Olson RJ. Dysphotopsia in phakic and pseudophakic patients: incidence and relation to intraocular lens type. J Cataract Refract Surg. 2000;26:810-6.
7. Osher RH. Negative dysphotopsia: long-term study and possible explanation for transient symptoms. J Cataract Refract Surg. 2008;34:1699-1707.
8. Kershner RM. Neuroadaptation and premium IOLs: What does the brain think? Ophthalmology Management. 2011;15(12):51-4.
9. Kent C, Kershner RM, Mainster M, McDonald JE. Multifocal neuroadaptation: Can training help the brain? Rev Ophthalmol. 2010;17(3):24-31.
10. Bellucci R. An introduction to intraocular lenses: material, optics, haptics, design and aberration. Cataract. ESASO Course Series. Basel, Karger, 2013;3:38-55.
11. Auffarth GU, Brezin A, Caporossi A, et al. Comparison of Nd:YAG capsulotomy rates following phacoemulsification of PMMA, silicone, or acrylic intra-ocular lenses in four European countries. Ophthalmic Epidemiol. 2004;11(4):319-29.
12. Cheng J, Wei R, Cai J, et al. Efficacy of different intraocular lens materials and optic edge designs in preventing posterior capsular opacification: a meta-analysis. Am J Opthalmol. 2007;143:428-36.
13. Hollick EJ, Spalton DJ, Ursell PG, et al. The effect of polymethylmethacrylate, silicone, and polyacrylic intraocular lenses on posterior capsular opacification 3 years after cataract surgery. Ophthalmology. 1999;106:49-54.
14. Yan Q, Perdue N, Sage EH. Differential responses of human lens epithelial cells to intraocular lenses in vitro: hydrophobic acrylic versus PMMA or silicone discs. Graefe's Arch Clin Exp Ophthalmol. 2005;243:1253-62.
15. Erie JC, Bandhauer MH, McLaren JW. Analysis of postoperative glare and intraocular lens design. J Cataract Refract Surg. 2001;27:614-21.
16. Davison JA. Positive and negative dysphotopsia in patients with acrylic intraocular lenses. J Cataract Refract Surg. 2000;26:1346-55.
17. Peng Q, Visessook N, Apple DJ, et al. Surgical prevention of posterior capsule opacification. Part 3: Intraocular lens optic barrier effect as a second line of defense. J Cataract Refract Surg. 2000;26:198-213.
18. Holladay JT, Lang A, Portney V. Analysis of edge glare phenomena in intraocular lens edge designs. J Cataract Refract Surg. 1999;25:748-52.
19. Holladay JT, Zhao H, Reisin CR. Negative dysphotopsia: The enigmatic penumbra. J Cataract Refract Surg. 2012;38:1251-65.
20. Vamosi P, Csakany B, Nemeth J. Intraocular lens exchange in patients with negative dysphotopsia symptoms. J Cataract Refract Surg. 2010;36:418-24.
21. Burke TR, Benjamin L. Sulcus-fixated intraocular lense implantation for the management of negative dysphotopsia. J Cataract Refract Surg. 2014;40:1469-72.
22. Masket S, Fram NR. Pseudophakic negative dysphotopsia: Surgical management and new theory of etiology. J Cataract Refract Surg. 2011;37:1199-1207.
23. Cooke DL, Kasko S, Platt LO. Resolution of negative dysphotopsia after laser anterior capsulotomy. J Cataract Refract Surg. 2013;39:1107-9.
24. Folden DV. Neodymium:YAG laser anterior capsulectomy: Surgical option in the management of negative dysphotopsia. J Cataract Refract Surg. 2013;39:1110-5.
25. Grayson, D. The ins and outs of lens explantation. Rev Ophthalmol. 2011;18(10):36.
26. Leysen I, Bartholomeeusen E, Coeckelbergh T, Tassignon MJ. Surgical outcomes of intraocular lens exchange. Five-year study. J Cataract Refract Surg. 2009;35:1013-8.

The Debut of a Pupil Optimized Multifocal Contact Lens

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Michael Ciszek, OD

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Clarke D. Newman, OD, FAAO

Drs. Clayton and Shah are paid consultants for Johnson & Johnson Vision Care, Inc.; Drs. Ciszek, Vincent-Riemer and Lee received compensation for their time in contributing to this article, and Drs. Newman and Barnett for their service on a Johnson & Johnson Vision Care, Inc. Advisory Board.

ACUVUE® Brand Contact Lenses are indicated for vision correction. As with any contact lens, eye problems, including corneal ulcers, can develop. Some wearers may experience mild irritation, itching or discomfort. Lenses should not be prescribed if patients have any eye infection, or experience eye discomfort, excessive tearing, vision changes, redness or other eye problems. Consult the package insert for complete information. Complete information is also available from Johnson & Johnson Vision Care, Inc., by calling 1-800-843-2020 or by visiting acuvueprofessional.com.

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This lens is an excellent option, even for patients who have had difficulty reading with a multifocal contact lens previously."
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Charles Clayton, OD

"This lens is super easy to fit in a wide range of patients, from prior multifocal lens wearers to single-vision or monovision wearers to contact lens neophytes and even some post-LASIK patients. The success rate in my first 35 completed fits, which included all those types of patients, was very high: 76.4% were satisfied with their vision and comfort."
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Presbyopic contact lens wearers are so eager to stay in contact lenses that many are willing to tolerate big tradeoffs. But what if you could offer them great distance and near vision, all-day comfort, and the health and convenience benefits of a daily disposable lens? With 1-DAY ACUVUE® MOIST Brand MULTIFOCAL Contact Lenses, you can.

"If you really listen to patients voice their frustrations, you find that presbyopes' complaints are partly functional – they hate switching back and forth between readers and no readers at work, for example – and partly social. Nobody likes being perceived as old when they can't see a menu. I recently fit a 60-year-old patient in 1-DAY ACUVUE® MOIST Brand MULTIFOCAL and she was absolutely delighted. She told me, "You gave me back my 39-year-old eyes!" Prescribing contact lenses is such a simple thing for us, but it can have a huge impact on our patients."
Arti Shah, OD, FAAO

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

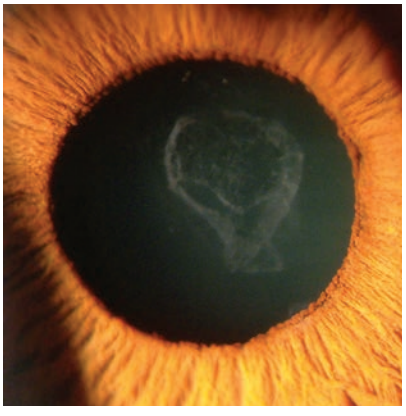
21st Annual Surgery Report

No Kidding Around: Managing PEDIATRIC and EARLY-ONSET Cataract

When a younger-than-expected patient presents with lens opacity, can you identify the cause? **By Marc D. Myers, OD, and Andrew S. Gurwood, OD**

Second only to uncorrected refractive error among causes of visual impairment, cataract is the number one cause of preventable blindness worldwide.[1] Cataract can occur at all ages due to a multitude of precipitating events, but of course age-related cataract comprises the vast majority of cases. Although the scenario is less common, early-onset lens disease can affect functional vision to the point where patients require ophthalmic intervention much earlier in life. These non-age-related changes of the crystalline lens can be classified as *congenital* or *infantile* (present within the first year of life), *juvenile* (within the first decade of life) or *presenile* cataract (present before the age of about 45 years).[3]

While the management approach of surgical extraction and IOL implantation will not differ in principle from that used in age-related cataract, identifying the form and cause may uncover treatable systemic diseases that require attention.



Diabetic cataract — the result of uncontrolled diabetes —located in the region of the anterior lens cortex.

Why So Soon?

Risk factors associated with development of presenile cataracts include gender, race, exposure to UV radiation, smoking, genetic predisposition, diet, body mass index, systemic diseases, medications, ocular trauma and secondary ophthalmic disease.[4-13] While age-related cataracts are an asymmetric phenomenon, cataracts of non-age-related etiologies are often either asymmetric or unilateral.[4-13]

Ocular diagnoses associated with secondary cataract may include: uveitis, glaucoma, retinal detachment, retinal degenerations such as retinitis pigmentosa or gyrate atrophy, persistent hyperplastic primary vitreous (PHPV), aniridia, Reiter's anomaly, sclerocornea, micro-ophthalmia, retrolenticular fibroplasia, Norrie's disease, anterior segment degeneration and necrosis, retinoblastoma and high myopia.[3,12,13] Other causes include general atopy, high myopia, steroid intake, sunlight exposure and diabetes.[6-13] Another risk factor is vitreoretinal surgery of any kind with virtually a 100% chance of lens opacity associated with vitretomy.[15-17]

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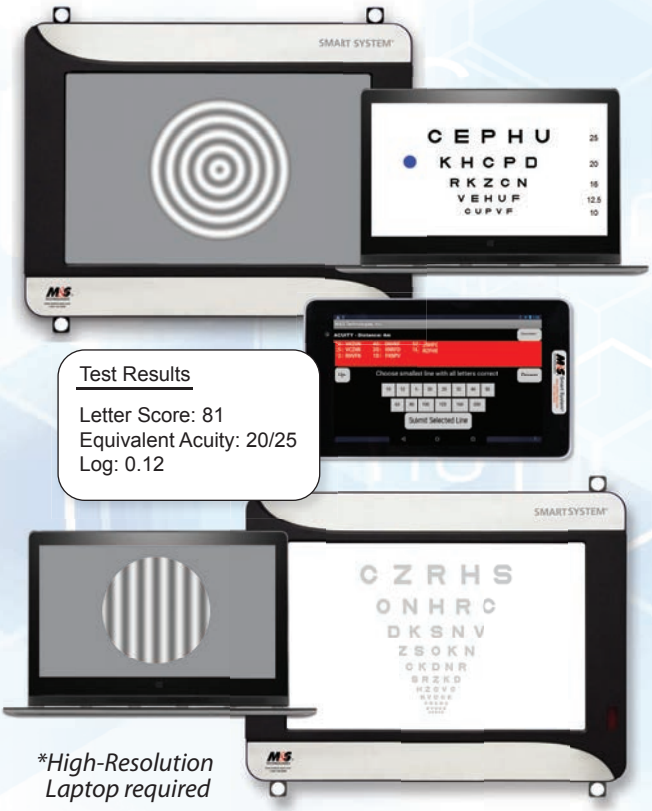

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Bringing Clarity to 'Cataract'

The Meriam Webster Online Dictionary recognizes the word *cataract* to be derived from the Latin word *cataracta*, meaning waterfall.[2] The ophthalmic use of the word connotes a clouding or discoloration of the lens of the eye, with or without clouding of its surrounding transparent membrane, obstructing the passage of light.[2]

Cataracts may result from:

(1) the natural process of aging, as the physiology of the tissue becomes altered by a lifetime of exposure to light (age-related cataracts) and the byproducts of normal physiology,

(2) a result of faulty development (congenital cataract) or

(3) disruption of the precise architecture (congenital, pharmacologic, traumatic and metabolic-induced cataracts).[3]

The amount of visual disability produced is related to the location (central or peripheral, anterior or posterior), the size and the nature (cortical, nuclear, subcapsular) of the opacity, as well as the visual demands of the individual. Symptoms can range from visual dimming to difficulties in low levels of illumination. Noticeable alterations in the colors of things, disability glare and decreased visual acuity both at distance and at near are common.

Many ophthalmic surgical procedures performed on the anterior and posterior segment of the eye result in the formation of cataract. Glaucoma procedures have long been recognized to result in cataractogenesis, which was revealed in the Collaborative Initial Glaucoma Treatment Study (CIGTS).[14]

Posterior segment procedures, including the injection of medications and invasive surgery such as vitrectomy or repair of retinal detachment can be associated with the eventual formation of cataract.[15-17]

Anatomy and Physiology

The anatomical nature and position of the crystalline lens within the eye makes any abnormality in its structure capable of producing a deficiency in its function.[1,2,18-24]

The structure, via an encircling network of zonules that connect the supportive surrounding capsule to the circumferential ciliary muscle and gland, enables the focusing of images onto the retina. Any irregularity in lens tissue architecture will lead to zones of opacity in the lens. Depending on where they are, their density and their linear area, variable symptoms such as glare and loss of acuity occur. The lens is underpinned by specific developmental physiology established prenatally during organogenesis. During development, surface ectoderm associated with the neural retina invaginates to form the lens vesicle.[18,19] Cells in the posterior half of the lens differentiate into primary lens fibers that form the lens fiber core. Those in the anterior half maintain a proliferative state as a monolayer of lens epithelium.[18] Differentiating lens fiber cells elongate and cover the old lens fiber core, resulting in spatially-regulated growth of the lens during development.[18]

As the lens forms, its various central regions are referred to as nuclei. The lens has four naturally occurring nuclei: the embryonic nucleus, the fetal nucleus, the juvenile nucleus and the adult nucleus. The differing regions of the lens meld together, forming two Y-shaped sutures, visible upon biomicroscopy.[3] A "hinged" design permits the structure to change shape by altering its convexity, while maintaining relative clarity and superior optical properties (ciliary body contraction induces slack on the zonules, creating more convexity and increased plus power, while relaxation of the ciliary body tightens the zonules to create less convexity and hence less plus power).[2,18]

Congenital Cataract

Conditions present at birth account for one in 10 childhood blindness cases, and approximately half of congenital cataracts are genetically determined.[8,25] The prevalence of cataract in childhood is estimated between one and six per 10,000.[25] Of patients diagnosed with lens disease in their first year of life, more than 8% had a positive family history of cataract. Of the children with a positive family of history of congenital cataract, 8% presented with unilateral involvement.[25]

Congenital cataracts may be isolated, or can be associated with other developmental abnormalities of the eye.[8] They may also be associated with inherited multi-system disorders.[25] Causes of congenital cataracts include intrauterine infections (rubella, varicella, toxoplasmosis), metabolic disorders, trauma and juvenile ocular inflammatory disease.[25] In many instances, especially in unilateral presentations, the etiology of the cataract is unknown.[25]

In one study evaluating idiopathic cases, 67% of the cases were characterized by a history of prenatal maternal illness. The remaining 22% involved the use of medication during pregnancy.[25] In the United States, routine immunization has rendered cataract from congenital rubella virtually nonexistent.[26]

Most congenital cataracts are inherited as an autosomal dominant trait.[3,4] Much like senile cataracts, childhood cataracts are categorized according to both their appearance and distribution of opacification.[3,4] The opacification nomenclature includes nuclear, lamellar, pulverulent, posterior polar, cerulean (blue dot) and anterior polar opacities.[26]

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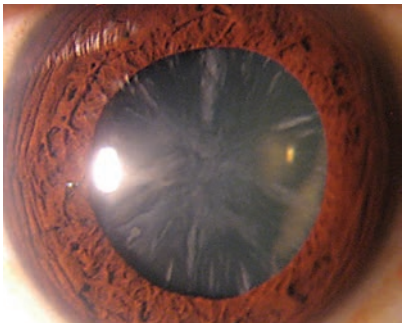
Polar opacities involve either the anterior or posterior pole of the lens (or both, referred to as bipolar opacities), as well as posterior subcapsular cortex opacities. Zonular cataracts include specific lens regions (including nuclear cataracts, which affect the fetal or embryonic nucleus, and lamellar cataracts).[26] Membranous or capsular cataracts can result from resorption of lens proteins after capsular rupture.[26] In membranous cataracts, the early lens was traumatized or experienced severe dysfunction.[26]

Pharmacologic Cataract

The toxic effects of medications on the lens manifest as cloudiness in different anatomical locations, determined by the route of drug administration.[11] For example, equatorial lens changes are common with systemically administered drugs that cross the blood-aqueous barrier.[11] Centrally located anterior lens changes are commonly associated with use of topically administered drugs.[11] Posterior subcapsular and cortical changes occur secondary to the diffusion of noxious substances from the posterior chamber crossing the blood/aqueous barrier.[11] Uveal inflammation and disturbances within the vitreous humor can produce these as well.[11]

We commonly see posterior subcapsular cataracts in patients who have been prescribed oral, topical, inhaled or injectable corticosteroids for long periods of time.[11] Intravitreal injections of triamcinolone used in the treatment of retina pathology is implicated in the development of cataract, particularly posterior subcapsular changes. After a single injection, upwards of 15% to 20% of patients developed symptomatic cataract that warranted extraction. In patients who received multiple injections, 70% developed symptomatic cataract, including posterior subcapsular, nuclear and cortical varieties.[27,28]

Although the exact mechanism of steroid-induced cataract remains unknown, it is likely that glucocorticoid-induced gene transcription events in lens epithelial cells and also other intraocular or systemic cells interact to generate steroid-induced cataracts.[29] The incidence of steroid-induced cataract is related to the dosage and duration of both topical and oral steroid administration.



Stellate cataract of the anterior lens cortex as a result of phenothiazine use.

Steroid cataractogenesis begins in the posterior pole as refractile or multicolored dots that gradually evolve, making it difficult to differentiate from other cataracts. This characteristic can be used to detect the cataract formation early in the course of chronic steroid use. Lens opacities caused by corticosteroids may progress or remain stationary, but rarely regress upon withdrawal of the corticosteroid.[11]

The psychotropic medications chlorpromazine and thioridazine are common phenothiazines involved in cataract formation.[11] Both are used in the treatment of depressive or organic psychoses and schizophrenia.[11] Use of these medications use may lead to the accumulation of fine, white to yellowish-brown granules in the anterior cortex, as well as beneath the anterior capsule and in the area of the pupillary aperture.[11] As the opacity progresses, a stellate pattern of granules develops, becoming a true anterior polar cataract.[11]

Antineoplastic agents used to treat leukemia and other blood dyscrasias are associated with the development of posterior subcapsular lens opacities.[11] These lens changes present as scattered punctate cortical opacities and posterior lens capsule polychromatic sheen. It is thought that cataractogenesis is related to interference with nucleic acid metabolism during mitosis in the cell of the lens epithelium.[11]

Traumatic Cataract

Ocular trauma, resulting in permanent vision loss in 2% to 14% of children, is a leading cause of acquired monocular blindness, and may be the result of penetrating injury, concussive force, electric shock or radiation.[30] Open globe injuries have the most significant association with visually debilitating cataract.[30-35] Anatomically, younger patients exhibit stronger adherence between the posterior lens capsule and anterior vitreous.[30] Furthermore, the tertiary vitreous is anatomically connected to the peripheral retina at the vitreous base. Traction on the vitreous face transmitted to the retina and lens creates risk for both posterior segment disease and cataract formation.[30] In cases of penetrating and concussive force injuries, cataract formation generally occurs in the anatomical region of the lens where the trauma had induced traction on the lens. All regions of the lens have the potential to be associated with traumatic cataract, because any region is subject to trauma.[30-32]

The incidence of cataract resulting from electrocution varies greatly, due to differences in voltage and duration of contact with the current.[33] In

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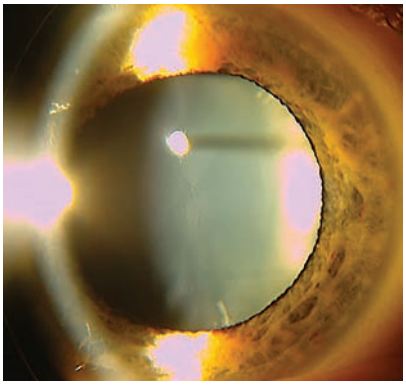
addition, the distance from the site of electrocution to the eye, extent of surface contact and the direction the current took through the body all contribute to the type and amount of cataract development.[33] The onset of cataract development following electrocution may occur within several days, or upwards of 18 months subsequent to the initial injury.[33] Early electrocution lens changes appear as a collection of multiple fine vacuoles below the anterior capsule in the midperiphery of the lens. This may require dilation to detect. Over time, the vacuoles progress to flake-like opacities, coalescing and migrating into the line of vision.[33] Although this type of cataract is often bilateral, the formation of the initial cataract will occur in the eye closest to the site of contact.[33]

Radiation cataracts develop six months to one year following local or systemic exposure. Late-onset cataracts have been reported decades following exposure.[35,36] The most common site of cataract following radiation injury is the posterior subcapsular region of the lens. Opacification is due to radiation damage to the lens epithelium around the equator, which disrupts epithelial DNA, causing abnormal differentiation of the elongating fibers. These cataracts often migrate into the posterior subcapsular area.[34-36]

Metabolic Cataract

Metabolic syndrome (MetS) represents a cluster of metabolic abnormalities involving central obesity, dyslipidemia, hyperglycemia and high blood pressure.[37,38] Studies show an association between MetS and an increased risk of developing cortical, nuclear and posterior subcapsula cataract subtypes.[39-41]

Compared with the general population, patients with diabetes tend to develop cataracts at an earlier age.[42,43] They also have a propensity to progress more quickly. Visual symptoms commonly occur as a result of secondary myopia, with hyperosmotic lens changes secondary to hyperglycemia.[42,43] Often, cataracts noted in young diabetes patients present as diffuse posterior and anterior subcapsular or cortical snowflake opacities.[42,43] Although the pathogenesis of the diabetic cataract is not completely understood, many theories exist.[42-44]



Congenital anterior polar cataract located on the anterior capsular surface.

Poor blood sugar control results in hyperglycemia, which is associated with the conversion of glucose to sorbitol in the polyol pathway. Accumulation of sorbitol in the lens occurs for two main reasons:

(1) Sorbitol is produced faster than it is converted to fructose; an enzyme called sorbitol dehydrogenase is at low ocular concentrations.

(2) The enzymes that compete to turn glucose into sorbitol or fructose don't have the same "strengths" or activities.

(3) Sorbitol is precluded from diffusing through the aqueous/blood barrier due to its classification as a sugar alcohol—its polarity hampers its ability to pass through the lipophilic blood/aqueous barrier.

For these reasons, sorbitol concentration supersedes the concentration of fructose.[42-44] The accumulation of sorbitol creates a hyperosmotic effect (e.g., it changes the tonicity of the ocular tissue relative to its surroundings, drawing in water). Hyperosmotic stress created by sorbitol accumulation induces apoptosis of lens epithelial cells.[42-44]

Central obesity, dyslipidemia and high blood pressure (and medical therapies used to treat these diagnoses) all have an association with the early development of cataract, particularly in cases where the diagnosis is uncontrolled.[42,43] Lens opacification may occur at the level of the cortex, nucleus and posterior subcapsular region, or may present in all three levels simultaneously.[42,43]

'Typical' Cataract Formation

Age-related cataracts are produced when insoluble lens crystallin proteins and other fibrillary materials aggregate within the lens stroma.[19-22] Small molecules known as mini-chaperones reduce protein aggregation by maintaining activity, which reduces the aggregation of proteins by blocking amyloid fibril formation, stabilizing mutant proteins, sequestering metal ions and exhibiting antiapoptotic properties.[19,20] The natural extinction of these chaperone proteins, due to their finite number within the lens, leads to the accumulation of protein and fibril aggregation.[19-22]

Advanced glycation end-products (AGE) have also been implicated in cataract formation.[23,24] The progressive accumulation of AGE in lens tissue mediates irreversible changes in structural proteins, provoking the formation of high molecular weight aggregates that scatter light and impede vision.[24] Further, AGE are responsible for creating aberrant crosslinking of extracellular matrix proteins, disrupting endothelial junctional complexes and creating areas of opacification and clefting.[22-24] In a process involving oxidative radicals, the lens naturally undergoes architectural changes, which produce the precursor opacities to mature cataract.

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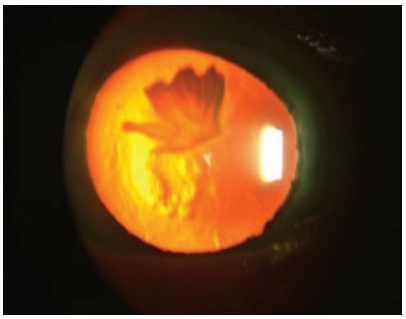

Partial rosette cataract involving the anterior cortex of the lens, imaged using retroillumination.

Treatment

Early detection and management of cataract in young patients avoids amblyopia and provides the best opportunity for optimal visual function throughout life. The evolution of surgical techniques, intraocular lens composition and design, along with a greater understanding of the neural biology of both visual development and early postoperative optical rehabilitation, all play a role in improving outcomes.[46,47]

The anatomy of a child's eye poses a unique challenge for the pediatric cataract surgeon. The young eye has a shorter mean axial length, a steeper cornea, a thin and less rigid sclera and a more elastic lens capsule, putting it at greater risk of developing a severe inflammatory response following surgery.[46,47] Because of the growth of the child's eye, the preoperative biometry for intraocular lens calculation can be quite complex. With this in mind, and because of myopic shift as the eye grows in axial length, the selection of the intraocular lens power must be suited for not only the acute postoperative period, but also for future visual function. Adding to the difficulties, a young child may not cooperate while the clinician attempts to obtain accurate axial length and keratometry readings.[46,47]

Even when considering the above, a recent survey concluded that pediatric ophthalmologists in the United States prefer implanting intraocular lenses at the time of cataract surgery.[46,48] If the decision is made not to implant the lenses, both aphakic spectacles and contact lenses may maximize best corrected visual acuity, reducing the likelihood of the development of amblyopia.

Depending on the age of the child, initial spectacle design may or may not include a bifocal power. Management of residual refractive error with contact lens wear may consist of a soft or rigid gas permeable lens. Lens wear is generally well tolerated and provides flexibility in the lens power as the visual system develops.

Postoperative care may include vision rehabilitation aimed at reducing the potential development of amblyopia. Therapeutic activities consistent with the recommendations of the National Eye Institute's Amblyopia Treatment Study I and II, such as direct occlusive therapy with a patch or atropine drops and organized binocular visual tasks (vision therapy), have been included in the postoperative course of pediatric cataract management.[46,48]

In cases involving young adults rather than infants and adolescents, concerns with respect to the developmental anatomy of the eye are not as great.[46,47] Such patients cooperate during measurements, and the likelihood of permanent degradation of best-corrected visual acuity is minimal. These patients generally have intraocular lenses implanted, with postoperative residual refractive error corrected with either spectacles or contact lenses.[46,47]

Any interruption in the structure, architecture or physiology of the crystalline lens can induce the formation of visually significant opacities capable of impacting function and lifestyle. Patients are vulnerable at any age. While we all will succumb to the age-related processes of opacification, presenile and other early-onset cases typically are accompanied by other health concerns that require care with the appropriate specialist.

Optometrists are uniquely positioned to be the first medical professionals to identify these cases and coordinate care as needed. Early diagnosis and management, thanks to modern materials and techniques, can provide solutions that mitigate amblyopia and provide excellent long-lasting outcomes for our patients. ■

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1. Dobrzynski JM, Kostis JB. Statins and cataracts—a visual insight. Curr Atheroscler Rep. 2015;17(2):477.
2. Merriam Webster. Cataract. www.merriam-webster.com/dictionary/cataract.
3. Hejtmancik JF. Congenital cataracts and their molecular genetics. Semin Cell Dev Biol. 2008;19(2):134-49.
4. Ketibeh M, Eskandari A, Yaseri M, et al. The gender issue in congenital and developmental cataract surgery. J Ophthalmic Vis Res. 2013;8(4):308-13.
5. Prakalapakorn SG, Rasmussen SA, Lambert SR, Honein MA. The national birth defects prevention study. Ophthalmology. 2010;117(8):1500-15.
6. Kleiman NJ. Radiation cataract. Ann ICRP. 2012;41(3-4):80-97.
7. Kar T, Ayata A, Aksoy Y, et al. The effect of chronic smoking on lens density in young adults. Eur J Ophthalmol. 2014;24(5):682-7.
8.Francis PJ, Moore AT. Genetics of childhood cataract. Curr Opin Ophthalmol. 2004;15(1):10-5.
9. Weintraub JM, Willett WC, Rosner B, et al. A prospective study of the relationship between body mass index and cataract extraction among US women and men. Int J Obes Relat Metab Disord. 2002;26(12):1588-95.
10. Salowi MA, Goh PP, Lee MY, et al. The malaysian cataract surgery registry: Profile of patients presenting for cataract surgery. Asia Pac J Ophthalmol (Phila). 2015;4(4):191-6.
11. Li J, Tripathi RC, Tripathi BJ. Drug-induced ocular disorders. Drug Safety. 2008;31(2):127-41.
12. Praveen MR, Shah GD, Vasavada AR, et al. A study to explore the risk factors for the early onset of cataract in India. Eye. 2010;24(6):686-94.
13. Vasudevan M, Premnath G. A prospective observational study to analyze the causes and types of presenile cataract in South Indian patients. J of Evolution of Med and Dent Sci. 2014;53(3):12308-15.

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14. Mochizuki T, Masai I. The lens equator: A platform for molecular machinery that regulates the switch from cell proliferation to differentiation in the vertebrate lens. Dev Growth Differ. 2014;56(5):387-401.
15. Lovicu FJ, McAvoy JW. Growth factor regulation of lens development. Dev Biol. 2005;280(1):1-14.
16. Raju M, Santhoshkumar P, Krishna SK. Alpha-crystallin-derived peptides as therapeutic chaperones. Biochim Biophys Acta. 2015;pii:S0304-4165(15)00172-5.
17. Raju M, Santhoshkumar P, Sharma KK. A-Crystallin-derived mini-chaperone modulates stability and function of cataract causing AG98R-crystallin. PLoS One. 2012;7(9):e44077.
18. Cetinel S, Unsworth L, Montemagno C. Peptide-based treatment strategies for cataract. J Glaucoma. 2014;23(8 Suppl 1):S73-6.
19. Kandarakis SA, Piperi C, Topouzis F, Papavassiliou AG. Emerging role of advanced glycation-end products (AGEs) in the pathobiology of eye diseases. Prog Retin Eye Res. 2014;42:85-102.
20. Nagaraj RH, Linetsky M, Stitt AW. The pathogenic role of maillard reaction in the aging eye. Amino Acids. 2012;42(4):1205-20.
21. Yi J, Yun J, Li ZK, et al. Epidemiology and molecular genetics of congenital cataracts. Int J Ophthalmol. 2011;4(4):422-32.
22. Papania MJ, Wallace GS, Rota PA, et al. Elimination of endemic measles, rubella, and congenital rubella syndrome from the western hemisphere: the US experience. JAMA Pediatr. 2014;168(2):148-55.
23. Jonas JB. Intravitreal trimamcinolone acetonide: a change in paradigm. Ophthalmic Res. 2006;38(4):218-45.
24. Cekic O, Chang S, Tseng JJ. Cataract progression after intravitreal triamcinolone injection. Am J Ophthalmol. 2005;139(6):993-8.
25. Kanthan GL, Wang JJ, Rochtchina E, Mitchell P. Use of antihypertensive medications and topical beta-blockers and the long-term incidence of cataract and cataract surgery. Br J Ophthalmol. 2009;93(9):1210-4.
26. Zhu L, Wu Z, Dong F, et al. Two kinds of ocular trauma score for pediatric traumatic cataract in penetrating eye injuries. Int J Care Injured. 2015;46(11):1828-33.
27. Khokhar S, Gupta S, Agarwal T. Epidemiology and intermediate-term outcomes of open- and closed-globe injuries in traumatic childhood cataract. Eur J Ophthalmol. 2014;24(1):124-30.
28. Lee SI, Song HC. A case of isolated posterior capsule rupture and traumatic cataract caused by blunt ocular trauma. Korean J Ophthalmol. 2001;15(1):140-4.
29. Hashemi H, Jabbarvand M, Mohammadpour M. Bilateral electric cataracts: Clinicopathologic report. J Cataract Refract Surg. 2008;34(8):1409-12.
30. Grewal DS, Jain R, Brar GS, Grewal SPS. Unilateral electric cataract: Scheimpflug imaging and review of the literature. J Cataract Refract Surg. 2007;33(6):1116-9.
31. Mohammadpour M, Movaheden ZE, Jabbarvand M, Hashemi H. Radiation cataract: Clinicopathologic report. J Cataract Refract Surg. 2013;39(2):285-8.
32. Wilde G, Sjöstrand J. A clinical study of radiation cataract formation in adult life following radiation of the lens in early childhood. B J Ophthalmol. 1997;81(2):261-6.
33. Maralani HG, Tai, BC, Wong TY, et al. Metabolic syndrome and risk of age-related cataract over time: An analysis of interval-censored data using a random-effects model. Invest Ophthalmol Vis Sci. 2013;54(1):641-6.
34. Alberti KGMM, Zimmet P, Shaw J. Metabolic syndrome—a new world wide definition. A consensus statement from the international diabetes federation. Diabet Med. 2006;23(3):469-80.
35. Tan JS, Wang JJ, Mitchell P. Influence of diabetes and cardiovascular disease on the long-term incidence of cataract: The Blue Mountains Eyes Study. Ophthalmic Epidemiol. 2008;15(2):317-27.
36. Sabanayagam C, Wang JJ, Mitchell P, et al. Metabolic syndrome components and age-related cataracts: The Singapore Malay Eyes Study. Invest Ophthalmol Vis Sci. 2011;52:2397-404.
37. Bojarskiene F, Cerniauskiene LR, Paunksnis A, Luksiene DI. Association of metabolic syndrome components with cataract. Medicina. 2006;42(1):115-22.
38. Al-Agha A, Ocheltree A, Rashad R, Abdelsalam I. Metabolic cataract in an 8-year-old diabetic boy. Turk J Pediatr. 2012;54(1):83-5.
39. Nabarro JD. Diabetes and United Kingdom: A personal series. Diabetic Med.1991;8(1):59-68.
40. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract—pathogenesis, epidemiology and treatment. J Ophthalmol. 2010 June 17. [Epub].
41. Medsinge A, Nischal KK. Pediatric cataract: Challenges and future directions. Clin Ophthalmol. 2015;9(1):77-90.
42. Wilson ME, Trivedi RH, Morrison DG, et al. Infant aphakia treatment study: Evaluation of cataract morphology and eyes with monocular cataracts. J AAPOS. 2011;15(5):421-6.
43. Wilson ME, Triverdi RH. Choice of intraocular lens for pediatric cataract surgery: Survey of AAPOS members. J Cataract Refract Surg. 2007;33(9):1666-68.
44. Pollreisz A, Schmidt-Erfurth U. Diabetic cataract - pathogenesis, epidemiology and treatment. J Ophthalmol 2010; Epub 2010 Jun 17.
45. Beebe DC, Holekamp NM, Shui YB. Oxidative damage and the prevention of age-related cataracts. Ophthalmic Res. 2010;44(3):155-65.
46. Medsinge A, Nischal KK. Pediatric cataract: Challenges and future directions. Clin Ophthalmol 2015;9(1):77-90.
47. Wilson ME, Trivedi RH, Morrison DG, Lambert SR, Buckley EG, Plager DA, Lynn MJ. Infant Aphakia Treatment Study Group. Infant aphakia treatment study: Evaluation of cataract morphology and eyes with monocular cataracts. J AAPOS. 2011;15(5):421-6.
48. Wilson ME, Triverdi RH. Choice of intraocular lens for pediatric cataract surgery: Survey of AAPOS members. J Cataract Refract Surg. 2007;33(9):1666-68.

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From Ordinary to EXTRAORDINARY:

The Rapid Evolution in Cataract Surgery

By Paul M. Karpecki, OD, FAAO, and Derek Cunningham, OD, FAAO

Release Date: October 15, 2015
Expiration Date: October 31, 2016
Goal Statement: On completion of this educational activity, participants should be able to describe how optometrists can become more involved in the perioperative management of their cataract patients, and detail the considerations and possible complications that cataract patients are subject to during the preoperative course.
Faculty/Editorial Board: Paul M. Karpecki, OD, FAAO, and Derek Cunningham, OD, FAAO
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Cataract is the main cause of blindness in the world.[1] Here in the U.S., three million cataract surgeries are performed each year.[2] This is a relatively modest figure when you consider that, in 2010, roughly 24.4 million Americans had cataracts, according to NEI.[3]

There is no question that the cataract population is growing exponentially. In fact, the number of Americans with the disease is projected to double by 2050—to about 50 million.[3]

Ophthalmologists will most certainly have full surgical schedules. Who then will deliver preoperative and postoperative care for all these patients?

As premium procedures become more commonplace and as outcomes continue to improve, patient expectations will likewise rise. This creates the added burden of extended preoperative counseling and follow-up care, particularly in patients who want better outcomes made possible by multifocal or accommodating IOLs.

In order to responsibly care for these patients, optometrists will need to take a more hands-on approach to cataract comanagement. Patients currently have outstanding options and can enjoy superb outcomes. As cataract surgery evolves and improves, optometrists can likewise help raise the bar in the preoperative and postoperative care of our expanding aging population.

Cataract Assessment

There are several traditional indications for cataract surgery:

- *Trouble seeing street signs*
- *Trouble with night driving/night vision*
- *Trouble recognizing faces*
- *Trouble reading*
- *Trouble seeing the golf ball*
- *Glare in bright sunlight or off car headlights*
- *Haloes around lights at night*
- *Double vision that does not go away upon covering one eye*

These conventional prerequisites are gradually being amended as patients' desire for better vision grows stronger. Likewise, the demographic is changing. Cataract patients don't always fit the "traditional" profile. They want something different—something more.

Younger baby boomers have a mindset that doctors aren't used to.

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Previous generations left all of the decision making up to us. This new, Internet-savvy generation is much more knowledgeable. They act young and want to look young, too. This more youthful lifestyle makes functional vision increasingly important at almost any age.

Although the spotlight may seem focused directly on looking for cataracts early and taking great care to make functional vision outstanding at every age, the nature of the care we provide at each and every regular exam is in no way diminished. In fact, our role as clinicians is more important than ever.

Profile of Today's Seniors

Many of today's seniors and boomers enjoy an active lifestyle. For example:[1-5]

- 82% of seniors 65+ are on the road, driving
- More than 50% of boomers exercise regularly
- 72% of boomers plan to keep working in some capacity after retirement
- 66% of boomers send text messages
- 90% of boomers are on the computer
- 65% of seniors (66+) have a cell phone

1. Data on file, Bausch + Lomb Incorporated. SurgiVision Datalink.
2. Hardy M. Elderly driving statistics. Seniors love to know website http://seniors.lovetoknow.com/Elderly_Driving_Statistics.
3 2010 Del Webb Baby Boomer Survey. November-December, 2009.
4. Sauer T, Moran E, McDonnell J. The State of the Media Democracy: Generational Shifts in Media Consumption and Creation. New York, NY: Deloitte Research; 2009:28.
5. Jones S. Generations Online in 2009. Washington, DC: Pew Research Center; 2009.

The first step in providing better opportunities for cataract patients is to improve disease diagnosis across categories. The glaucoma evaluation, for example, should continue to come first. You don't want to miss glaucoma when you could have done a microincisional glaucoma stent procedure at the time of cataract surgery. Similarly, you don't want to miss ocular surface disease and get a +4.00 diopter surprise post-cataract surgery patient.

Improved Surgical Procedures

From an intraoperative perspective, cataract surgery has improved by leaps and bounds in recent years. In fact, highly skilled surgeons can perform these procedures in as little as five to seven minutes. The primary benefit of abbreviated surgical time is not efficiency; it's safety and typically a quicker recovery. The longer you're in the eye, the greater the complication risk.

The smaller incisions made possible by foldable IOLs and microinjectors are another substantial improvement. Fifteen years ago, 4mm was considered a microincision; a few years later, 3mm became the cutoff. Nowadays, many U.S. surgeons are pushing their clear corneal incisions to 2.3mm to 2.5mm. In Europe, some incisions are as small as sub-2mm.

The ability to correct astigmatism at the time of cataract surgery is another major advance. Although there is still a place for manually performed limbal relaxing incisions (LRIs), the method is highly unpredictable. There are no precise indicators to let the surgeon determine the appropriate arc length and position of LRIs. As a result, surgeons need to be fairly conservative, which often results in undercorrection. Laser cataract surgery, on the other hand, offers highly predictable methods for correcting astigmatism.

Just as femtosecond technology replaced the microkeratome and transformed LASIK, femtosecond lasers are expected to continually improve the predictability of cataract surgery.

The four commonly used lasers on the market are:

- *Catalys (AMO)*
- *LensAR (LensAR, Inc.)*
- *LenSx (Alcon)*
- *Victus (Bausch + Lomb)*

Each laser has its own particular strengths, but all four excel at making cataract surgery more precise by replacing the blade and offering the visualization tools needed to cut any plane at any depth. As such, using a femtosecond system, the surgeon is able to make precise incisions, create a capsulotomy and fragment the lens—all with the aid of a real-time OCT.

The three key advantages of femtosecond that drive the growth of this technology are:

1) The potential for greater safety.[4,5] Potentially fewer capsule tears translates into lower complication rates.[4,5]

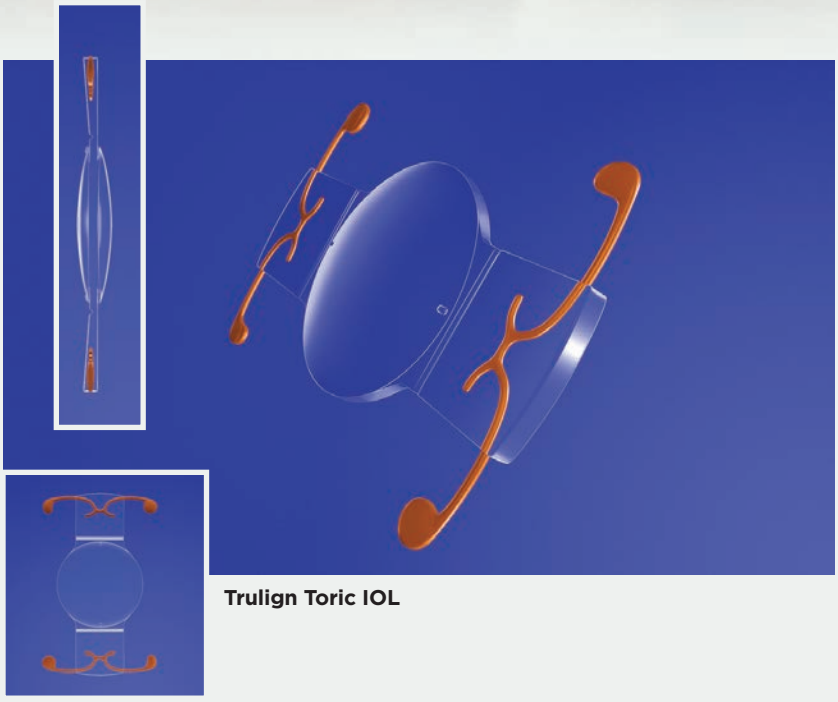
2) Better consistency.[4,6,7] With respect to capsulotomies, femtosecond offers better circularity, accurate diameters and precise centration.[4,6,7]

3) The potential for improved outcomes, particularly concerning optimization of premium products.[4,7] Femtosecond technology allows the surgeon to effectively position IOLs with outstanding predictability.[7] There is less IOL decentration with femtosecond techniques, which is especially important when implanting a toric or a multifocal lens.[4]

Higher IOL Standards

Three of the most commonly implanted presbyopia lenses in the U.S. are the Restor (Alcon), the Tecnis (Abbott Medical Optics) and the Crystalens (Bausch + Lomb). Each features different design characteristics in terms of optics and presbyopia correction.

The AcrySof IQ Restor is a multifocal IOL that has been approved



Trulign Toric IOL

for use in the U.S. since 2005. This lens excels at delivering outstanding near vision. What makes the Restor unique is its apodized diffractive design. The diffractive step widths decrease as you move toward the refractive zone of the lens. Likewise, the diffractive step heights also decrease (apodization), which allows the control of light energy distribution, directing more and more light energy to the distance focal point. This decrease in step heights reduces visual disturbances because, in low-light conditions, as the pupil dilates, more light is distributed to elements in the lens required for clear distance vision. But this also means near vision may not be as crisp with the Restor lens in low light as it is in bright light conditions. The diffractive rings in this lens do not go out to the edge; they stop in the mid-periphery. This makes for outstanding daytime vision but can present some near vision challenges at night.

The Tecnis Multifocal IOL, marketed by Abbott Medical Optics (AMO), has been approved for use in the U.S. since 2009. One difference with the Tecnis compared to the Restor is that its diffractive surface extends all the way out to the periphery, which improves image quality at all distances under any lighting conditions and regardless of pupil size.

The Tecnis is now offered in three different add powers: +4.00, +3.25, and +2.75. This allows for outstanding customization, but to get the greatest benefit and make the best lens selection the doctor must have an intimate understanding of the patient's lifestyle. In this regard, referring and comanaging optometrists' roles are more vital than ever.

As with the Restor lens, the ideal patient for a +4.00 Tecnis is someone who favors near vision-related activities, such as reading or knitting. The +3.25 is designed for patients whose lifestyle demands involve activities at longer reading distances, such as multimedia work. Finally, the +2.75 is intended for patients who spend a lot of time performing intermediate vision activities, such as golfing or grocery shopping. In many cases, surgeons will choose to mix and match these lenses, putting one add in the dominant eye and a different one in the other.

Preparation for Ocular Surgery

To properly prepare for cataract surgery, the following steps must always be followed:

- Optimize the ocular surface
- Normalize the lids
- Prepare the cornea
- Eliminate intraocular inflammation
- Control glaucoma
- Examine the macula
- Evaluate the retinal periphery
- Deliver patient education

The Crystalens, which is based on an entirely different design principle than the Tecnis or the Restor, is the only accommodating IOL approved for use in the U.S. It features a uniform center-to-edge power, so there are fewer concerns about lens decentration.[8] Also, the lens was designed to increase contrast sensitivity.[9] It accommodates by moving forward and backward, and delivers excellent vision across the entire active range.[10] The Crystalens also requires less neuroadaptation than a multifocal and is aberration free.

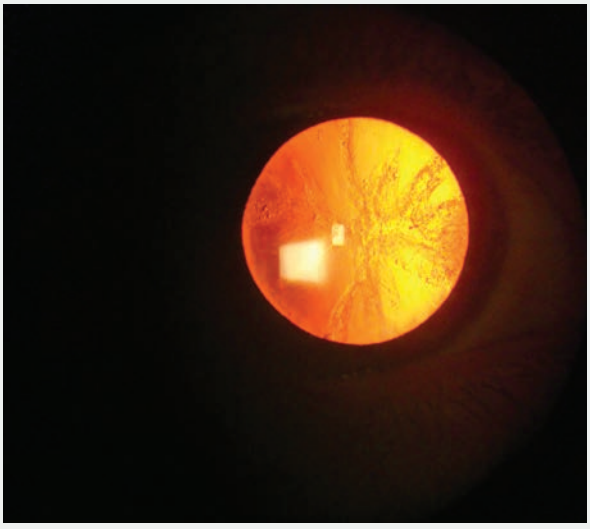
The idea for the Crystalens was born from some of the early research on plate lenses about a decade ago.[11] When the researchers implanted large, flat plate lenses, patients could suddenly see well enough to read again. The reason, they found, was that the ciliary body continues to function our entire lives. Even when it's not attached, it pushes the vitreous forward and allows it to bow out a little bit. This slight movement offers a little bit of near, which could easily be enhanced with a technology like the Crystalens.

The secondary mechanism allowing the Crystalens to function well in presbyopes derives from its aspheric properties. Even if the Crystalens didn't move at all, its asphericity delivers some depth of focus. However, because the Crystalens is dependent on the patient's anatomy, there is

variability from patient to patient. You can't predict how much accommodation a given patient can expect. As such, setting realistic patient expectations is particularly essential with the Crystalens.

It's wise to explain that this lens won't make patients see the way they did when they were 24 years old. It's more likely that post-op vision will mimic what it was like in one's mid-40s or early 50s, with good distance vision and some accommodation.

Photos by: Derek N. Cunningham, OD, FAAO



Cortical cataract

One of the primary benefits of the Crystalens is the outstanding intermediate vision that it provides compared to multifocal IOLs.[10,12] This lens is an ideal choice for patients who desire an active lifestyle.

With respect to corneal astigmatism, if it is at or over 0.75D, you should plan to treat it because it makes a tremendous difference—so much so, in fact, that many surgeons now use a toric IOL in patients with as little as 0.5D of astigmatism.

Until a few years ago, we had to make a choice when selecting an intraocular lens. We could either correct astigmatism or presbyopia; we couldn't do both. The FDA approval in 2013 of a toric version of the Crystalens, called Trulign, changed all of that. Trulign is available in cylinder powers of 1.25D, 2.00D and 2.75D, measured at the IOL plane, and spherical equivalent powers of +4D to +10D in 1D increments and +10.5D to +33D in 0.5D increments. Like the Crystalens, Trulign is also a silicone lens with a 5mm optic and rectangular-hinged flexible haptics with loops made of polyamide.

When presenting this lens to patients, focus your discussion primarily on astigmatism correction and explain that the patient may still need to wear glasses for extended reading or fine print. These patients tend to be extremely pleased after surgery when they discover they can read their watch, cellphone and computer—often without glasses.

Patient Selection

Although many patients educate themselves before they show up in your chair, not all patients understand what cataracts are. Sometimes, they are worried and have concerns about surgery. They don't know what surgeon to go to and they rarely know what procedure is best for them.

As primary care providers, we understand how our patients use their eyes and what their individual needs are. It is our role to relay this information as it affects their decisions about cataract surgery.

The approach we take should be similar to the one we use when presenting contact lens options. Every choice involves compromise, so each decision has to be evaluated in terms of what motivates the patient and what will most effectively improve their quality of life.

Candidates for multifocals and accommodating IOLs should have a visual and functional need for cataract surgery, be motivated not to wear glasses, desire an active lifestyle, qualify for bilateral implants and have realistic expectations.

Poor candidates include patients who expect perfect vision, and those who are not willing to accept potential complications of cataract surgery, including the possibility of glare or halos at night. Patients who demand immediate results are likewise not good candidates for premium procedures.

Preoperative Clinical Management

Complications in cataract surgery usually can be avoided by taking appropriate steps preoperatively (see "Preparation for Ocular Surgery"). Although there are always exceptions, generally speaking, bilateral implantation is preferred and results in superior outcomes. It's also wise, when selecting a premium lens, to ensure that you can achieve good binocularity. If there's a visual disparity, it can have a noticeable effect.

If you can check off these boxes, it's time to start taking a close look at ocular health. You want healthy eyes. If the patient has early macular degeneration or loss of contrast from glaucoma, they're not going to do well with multifocal lenses where they lose some contrast.

Next, move on to surface health. The importance of managing dry eye cannot be overemphasized because it can dramatically affect your calculations. When performing a preoperative evaluation on cataract patients, always perform a thorough ocular surface exam at the slit lamp. (See "Preoperative Ocular Surface Exam"). Another helpful tip when

looking for dry eye: If the topography reveals missing spots, proceed with a dry eye diagnosis.

Some of the newer tools that we now use to decide whether a patient's ocular health is stable enough to allow for good cataract surgery outcomes include the three-minute SPEED questionnaire and osmolarity testing. No single test will prevent patients from falling through the cracks. It's like glaucoma testing—you can't rely on tonometry alone. With dry eye, you can't rely exclusively on symptoms or tear breakup time.

Perioperative Management

This step begins with making sure that the surgical prophylaxis protocol has been followed. Antibiotics as well as NSAIDs should have been used one day prior. In high-risk patients, NSAIDs should be initiated prior. In addition, patients need a five-minute exposure to 5% betadine solution. This is the only proven method to reduce the risk of endophthalmitis.

Anything that happens during the procedure is the responsibility of the surgeon. However, it's critical that you have good dialogue and continuous communication so that the ophthalmologist is aware of the patient's complete history. Send your notes to the surgeon after each visit.

Alert the ophthalmologist to any ocular or systemic medication use. In particular, it is critical that you make sure the surgeon is aware of any history with the drug tamsulosin (Flomax). This alpha-blocker is used to treat benign prostatic hyperplasia (BPH). During surgery, it can cause intraoperative floppy iris syndrome (IFIS). The clinical manifestations of IFIS complicating cataract surgery are poor preoperative pupil dilation, iris billowing and prolapse and progressive intraoperative miosis. In one prospective study, 90% of 167 eyes from patients taking tamsulosin exhibited some degree of IFIS during cataract surgery.[13]

Postoperative Management at Day 1

At the Day 1 post-op visit, confirm medications with the patient and check distance vision only. Do not check near vision at this visit.

Preoperative Ocular Surface Exam

When performing a preoperative evaluation on cataract patients, always look for the following at the slit lamp:

Lids and Lashes
- Blepharitis
- MGD (express the meibomian glands)
- Lid position (ectropion, entropion, dermatochalasis)

Conjunctiva
- Pingueculas
- Blepharoconjunctivitis

Cornea
- Pterygium, EBMD, corneal scars, Salzmann's nodules, etc.
- Tear film analysis
- Endothelial changes (guttatae and Fuchs' dystrophy)
- Previous HSV can reoccur from surgical trauma (pre-treat with oral antivirals)

At the slit lamp, make sure that the wound is secure and the cornea is clear. Note any edema. Some temporary swelling of the cornea may be present due to prolonged phaco time with dense nuclei.

Don't be alarmed if you see some microcystic edema in the cornea. This happens in normal cases when the endothelial cells have been disrupted. It will almost always get better in a few days.

The anterior chamber should be well-formed with about 2+ cell and the IOL should be well-centered in the pupil.

Of course, the primary concern at the Day 1 visit is IOP. However, this is one of the few times when low pressure should be cause for greater concern than high pressure because, to a certain point, the latter is actually helping the incision heal faster.

The body can handle hypertension, but it cannot handle significant hypotension. If you see very low IOP after cataract surgery—for example, 15mm Hg to 20mm Hg below physiological normal—it's an emergency. On the other hand, 15mm Hg above physiological normal is not cause for alarm, unless the patient is diagnosed with advanced glaucoma.

Truly high IOP at Day 1 would be a pressure close to or greater than 40mm Hg. At that level, there is reason to be concerned about the potential to shut off blood vessels in the posterior segment. As such, intervention is wise at this point.

Most cases of elevated pressures at Day 1 can be attributed to retained viscoelastic. To help the patient feel better right away, you can perform corneal decompression—often called "burping the wound." This is not a long-term solution; the pressure will gradually go back up, but not to the same level. It does, however, offer some relief and improves acuity in the short term as the eye slowly normalizes.

If you see a very low pressure, check the anterior chamber and perform a Seidel test. If the anterior chamber is formed and there is no secondary complication from hypotony, you can treat the mild wound leakage conservatively with a bandage contact lens and antibiotics QID, and then follow up in 24 hours. Some doctors are even using ReSure sealant to help in cases where the incision does not appear tight.

Finally, go over all of the instructions with the patient. Review medications and explain that, while

there are no restrictions on physical activities, patients need to avoid swimming, hot tubs and gardening. Remind patients that it is normal for vision to be blurry and eyes to feel a little out of balance. This goes a long way in avoiding buyer's remorse.

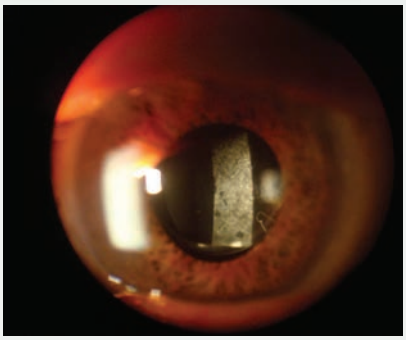
Finally, be sure to fax all of your exam results to the surgeon.

Postoperative Management at Week 1

After one week, the primary concern shifts from IOP to endophthalmitis. Endophthalmitis usually does not occur at Day 1 because it usually is a result of a communicating wound—meaning bacteria got in through the wound orifice. The Betadine that's used during surgery prevents this from happening right away. When endophthalmitis does appear, it's usually only after the Betadine wears off (at about three to five days) and the flora in the eye recolonizes.

If a patient calls with symptoms during the first week, that patient must be seen right away. Ask the patient to grade the pain on a scale of 1 to 10. If they report that it's two or three, it's probably just a little foreign body sensation from the incision. But if they say anything above a five, your very next question should be, "Are you losing vision?" If they say, "yes," it is a surgical emergency in which hours (not days) make a difference.

Because it's always present in ocular flora, *Staphylococcus aureus* is the most common cause of endophthalmitis. More specifically, coagulase-negative MRSA-resistant *Staph*. currently is the most common cause of endophthalmitis. Although no antibiotics are FDA approved for use following cataract surgery, your surgeon's antibiotic selection may be driven by the drug's effectiveness against this particular bug, in which case he or she may prescribe besifloxacin ophthalmic suspension 0.6% (Besivance, Bausch + Lomb) since this particular fluoroquinolone has been shown to be effective against MRSA.[14]



Posterior capsular fibrosis

In addition to the slit lamp exam and a close look for signs of infection (including an increase in anterior chamber cell and flare, or the presence of a hypopyon), the Week 1 post-op exam should include refraction. Check distance vision as well as near vision with good lighting. Perform another IOP check and remember to confirm that the patient is using all medications as directed.

Postoperative Management at Month 1

At the one-month visit, you will again check IOP and obtain a final refraction for distance and near with good lighting.

At the slit lamp, ensure that the cornea is clear and there is no edema. Look for any surface disease such as dry eye or SPK. The anterior chamber should be well-formed with no cell and the IOL should be well centered in pupil. Fully evaluate the posterior capsule. Also, perform a detailed fundus exam, looking closely at the peripheral retina.

Confirm that there is no cystoid macular edema. CME is the most frequent cause of visual decline following uncomplicated cataract surgery.[15] While patients with systemic disease are even more likely to develop it, CME is estimated to occur in 12% of low-risk cataract cases.[16,17]

Risk factors for CME include:

- *Pre-existing ocular inflammation*
- *Diabetic retinopathy*
- *Ocular vascular disease*
- *Cardiovascular disease*
- *Epiretinal/vitreoretinal membrane*

Prophylaxis treatment should be started earlier and extended longer for high-risk patients.[16]

To complicate matters, CME is easy to miss, especially if you don't have access to an OCT. It's particularly complex in multifocal patients because the patient can be seeing 20/25+ and the macula may look fine, yet the patient reports terrible vision. In such cases, if you perform OCT, you will often find that the cause is a very subtle amount of CME.

Mild CME usually will resolve when you add a nonsteroidal anti-inflammatory to the steroid that the patient is already using. Severe cases (e.g., 20/50) need to be referred to retina for possible treatment with anti-VEGF injections, but in cases where it is less than 20/40, you can use an NSAID, such as bromfenac ophthalmic solution 0.07% (Prolensa, Bausch + Lomb) once a day, and schedule follow-up in one week. At that point, if there is improvement, you can stay the course. If not, refer the patient to a retinal specialist.

Because CME usually occurs at four to six weeks, high-risk patients on bromfenac prophylaxis should continue treatment for a minimum of eight weeks.

Postoperative Management at Three Months

At the three-month follow-up, the primary concern is posterior capsular opacification. In addition, look for the following:

- *Ocular surface disease*
- *Cystoid macular edema*
- *Rebound inflammation*

- *Retinal detachment*
- *IOL surprises*
- *Dislocated IOLs*

Always ask the patient if vision is fluctuating. Remember, there is no movement with an IOL—the lens itself doesn't fluctuate. If the vision is not stable, it's almost always due to the ocular surface. In these cases, take a close look at the tear film and meibomian glands.

Improving Cataract Care

As the aging population grows, so too does the need for cataract surgery. While demographics alone dictate the need for optometry to step up, when you add premium products to the equation, the need becomes even greater. Indeed, advanced IOLs and lasers have raised the bar on outcomes. However, expectations have risen as well.

The level of involvement that's required preoperatively and postoperatively in premium cases requires a better understanding of the patient's history and personality. This takes time that busy surgeons often don't have. Optometrists, on the other hand, usually have more than 20 minutes to control the destiny of their patients. We usually have had several years of history upon which to make wise recommendations and can make informed decisions about whether a patient would do better in a multifocal or an accommodative lens. Likewise, our patients trust us to provide quality postoperative care. ■

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Dr. Cunningham is the director of Optometry and Research at Dell Laser Consultants in Austin, Texas. He is also a founding member and chair of the Integrated Ophthalmic Managed Eyecare Delivery Task Force by the American Society of Cataract and Refractive Surgery.

1. Lighthouse International. Prevalence of Vision Impairment. http://www.lighthouse.org/research/statistics-on-vision-impairment/prevalence-of-vision-impairment/ Accessed August 17, 2015.
2. All About Vision. Cataracts. http://www.allaboutvision.com/conditions/cataracts.htm. Accessed August 17, 2015.
3. National Eye Institute. Projections for Cataract. https://nei.nih.gov/eyedata/cataract#5. Accessed August 17, 2015.
4. Friedman NJ, Palanker DV, Schuele G, et al. Femtosecond laser capsulotomy. J Cataract Refract Surg. 2011;37(7):1189-1198.
5. Palanker DV, Blumenkranz MS, Andersen D, et al. Femtosecond laser-assisted cataract surgery with integrated optical coherence tomography. Sci Transl Med. 2010;2(58):58ra85.
6. Reddy KP, Auffarth G. Evaluation of the precision and predictability of anterior capsulotomy using a femtosecond laser. Presented at: the Annual Meeting of the American Academy of Ophthalmology (AAO); October 21-25, 2011; Orlando, FL.
7. Kránitz K, Takacs A, Miháltz K, Kovács I, Knorz MC, Nagy ZZ. Femtosecond laser capsulotomy and manual continuous curvilinear capsulorrhexis parameters and their effects on intraocular lens centration. J Refract Surg. 2011;27(8):558-563.
8. Altmann GE, Nichamin LD, Lane SS, Pepose JS. Optical performance of 3 intraocular lens designs in the presence of decentration. J Cataract Refract Surg. 2005;31(3):574-585.
9. Pepose J. The longevity revolution. Ophthalmol Manage. 2011;Aug(suppl):3-7. [Pepose 2011, p.6A]
10. Ang R. Comparison of 3 presbyopia-correcting IOLs used in cataract surgery. Presented at: XXIX Congress of the European Society of Cataract and Refractive Surgeons (ESCRS); September 17-21, 2011; Vienna, Austria.
11. Nataloni R. Latest accommodating IOL design shows promise. Ocular Surgery News. 2001; March 1.
12. Pepose JS, Qazi MA, Davies J, et al. Visual performance of patients with bilateral vs combination Crystalens, ReZoom, and ReSTOR intraocular lens implants. Am J Ophthalmol. 2007;144(3):347-357.
13. http://www.escrs.org/eurotimes/january2004/images/Hachet_3.jpg on 4/10/11
14. Blondeau JM, Sanfilippo CM, Morris TW, et al. In vitro Antibiotic Susceptibility Profile of Ocular Pathogens - Results from the First ARMOR Canada Surveillance Study. Invest Ophthalmol Vis Sci. 2014 April;55(13): 2840.
15. Mishima H, Masuda K, et al. The putative role of prostaglandins in cystoid macular edema. Prog Clin Res 1989;31:251-264.
16. Samiy N, Foster CS. The role of nonsteroidal antiinflammatory drugs in ocular inflammation. Int Ophthalmol Clin. 1996;36(1):195-206.
17. McColgin AZ, Raizman MB. Efficacy of topical Voltaren in reducing the incidence of post operative cystoid macular edema. Invest Ophthmol Vis Sci. 1999; 40 S289.

QUIZ

1. The primary benefit of a shorter surgical time is:
a) Efficiency
b) A reduction in meds
c) Fewer complications
d) Less patient anxiety

2. In the U.S., what is the current size of a microincision?
a) 4mm
b) 3mm
c) 2.5mm
d) 2mm

3. Femtosecond lasers allow cataract surgeons to:
a) Make precise incisions
b) Create a capsulorhexis
c) Fragment the lens
d) All of the above

4. When used in cataract surgery, which does femtosecond offer?
a) Fewer capsule tears
b) Precise centration
c) Accurate diameters
d) All of the above

5. Which of the following is NOT a multifocal IOL?
a) Restor
b) Tecnis
c) Crystalens
d) None of the above are multifocals

6. Which of the following statements about the AcrySof IQ Restor lens is FALSE?
a) The diffractive step widths increase as you move toward the refractive zone of the lens.
b) It features an apodized diffractive design.
c) The diffractive step widths decrease as you move toward the refractive zone of the lens.
d) The diffractive step heights decrease as you move toward the refractive zone of the lens.

7. The Restor lens features:
a) Near vision that is equally crisp in dim light
b) Diffractive rings that go out to the edge
c) Outstanding near and daytime vision
d) Poor distance vision at night

8. What is the ideal Tecnis add for a patient who spends a lot of time performing intermediate vision activities?
a) +4.00
b) +3.50
c) +3.25
d) +2.75

9. All of the following statements about the Crystalens are true EXCEPT:
a) It is the only accommodating IOL approved for use in the U.S.
b) It requires more neuroadaptation than a multifocal
c) It features a uniform center-to-edge power
d) It is aberration free

10. Which of the following would be an ideal Crystalens patient?
a) A patient who desires an active lifestyle
b) A patient who wants to reclaim the vision they had when they were much younger
c) A patient who favors near vision-related activities
d) None of the above

11. Which of the following lenses correct for astigmatism and presbyopia?
a) Restor
b) Tecnis
c) Trulign
d) Crystalens

QUIZ

12. In cataract surgery patients, the drug tamsulosin (Flomax) causes:
a) Glaucoma
b) Intraoperative floppy iris syndrome
c) Posterior capsular opacification
d) All of the above

13. When comanaging cataract, what is the primary concern at Day 1?
a) IOP
b) CME
c) Endophthalmitis
d) Posterior capsular opacification

14. What test should you NOT perform at Day 1 post-cataract surgery?
a) IOP
b) Distance vision
c) Near vision
d) All of the above should be evaluated

15. Most cases of elevated pressures at Day 1 can be attributed to:
a) Glaucoma
b) Retained viscoelastic
c) Intraoperative errors
d) None of the above

16. Following cataract surgery, corneal decompression can:
a) Lower IOP in the short term
b) Offer some relief
c) Improve acuity
d) All of the above

17. When comanaging cataract, what is the primary concern at Week 1?
a) IOP
b) CME
c) Endophthalmitis
d) Posterior capsular opacification

18. When comanaging cataract, what is the primary concern at Month 1?
a) IOP
b) CME
c) Endophthalmitis
d) Posterior capsular opacification

19. How long should high-risk patients continue bromfenac prophylaxis?
a) 2-4 weeks
b) 4-6 weeks
c) 6-8 weeks
d) A minimum of eight weeks

20. When comanaging cataract, what is the primary concern at three months?
a) IOP
b) CME
c) Endophthalmitis
d) Posterior capsular opacification

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1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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12. Related to your practice needs: ① ② ③ ④ ⑤
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14. Avoided commercial bias/influence: ① ② ③ ④ ⑤
15. How would you rate the overall quality of the material presented? ① ② ③ ④ ⑤
16. Your knowledge of the subject was increased:
◯ Greatly ◯ Somewhat ◯ Little
17. The difficulty of the course was:
◯ Complex ◯ Appropriate ◯ Basic

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21st Annual Surgery Report

Get Taut on Floppy Eyelid Syndrome

This foreboding finding may foretell some more serious conditions.

By Victoria Roan, OD

Floppy eyelid syndrome (FES) is a condition characterized by an elastic-like upper eyelid that is easily pliant and everted with minimal lateral traction (*Figure 1*).[1] The tarsal plate is found to be "rubbery," with loss of the rigidity that normally maintains the integrity of the eyelid.[1] This clinical finding is now linked to several more severe ocular and systemic conditions, most notably keratoconus and obstructive sleep apnea-hypopnea syndrome (OSAHS), which we will discuss in further detail.[1-9]

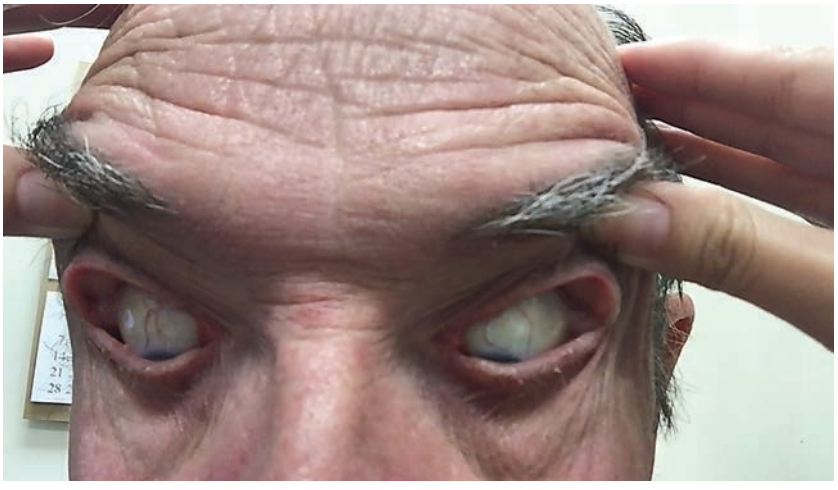

Fig. 1. This patient's elastic, easily pliant eyelids prompt a diagnosis of FES, which is also linked to several severe ocular and systemic diseases.

FES Research

In the study that originally described the condition, a group of 11 men displayed the triad of obesity (BMI>30); lax, easily-malleable upper eyelid; and tarsal papillary conjunctivitis of the upper eyelid palpebral conjunctiva. Their eyelids were found to spontaneously evert during sleep, and as a result of exposure of the upper palpebral conjunctiva to the pillow, these patients developed chronic conjunctivitis. The affected side corresponded to the side that the patient usually slept on, and if bilateral, the patient either slept without a preferred side or slept on their stomaches. Another study found asymmetric involvement in 50% of patients and an equal distribution between either eye in the remainder of patients.[2] Later studies found



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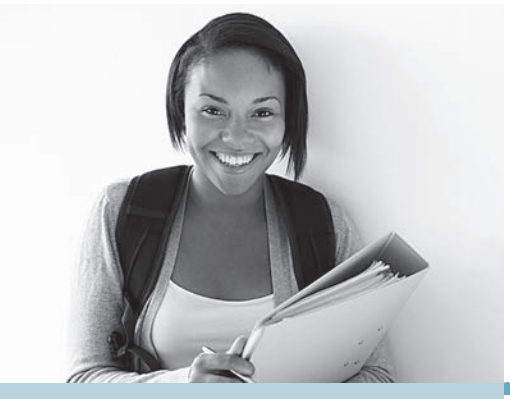

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that up to 37% of cases are female and that not all patients with FES are overweight or obese.[3,4,10] In fact, several case reports document FES in women, children, infants and non-obese patients, although overweight men still represent the majority of patients with FES.[5]

Clinical Presentation

The symptoms include a nonspecific irritation, foreign body sensation, mucoid discharge, dryness, redness, photosensitivity and eyelid swelling.[7,8,11,12] Unfortunately, due to the similarity in symptoms, patients are often misdiagnosed with chronic infective conjunctivitis, blepharitis or dry eye syndrome, which may lead to delayed or missed treatment for months or even years.[11,12] The goal of the clinician is to recognize the specific traits of FES that differentiate it from inflammation of the eyelids and ocular surface conditions, as FES does not respond to standard anti-inflammatory therapy. Earlier detection will not only relieve the patient of discomfort but will also stop the progression toward more severe ocular sequelae if improperly managed.

Eyelid changes that may accompany FES include blepharoconjunctivitis, blepharoptosis, lower lid ectropion, hyperkeratotic skin lesion of the lids and face, keratinization of the conjunctival surface of the affected upper eyelid, upper eyelid ptosis, eyelash ptosis and loss of eyelash parallelism.[2,6,13]

Changes in tarsus integrity and redundancy of the eyelid can affect the stability of the tissue into which the cilia are anchored, causing downward displacement of the hairs, referred to as eyelash ptosis.[7,12,13] Abnormalities of the levator muscle are not indicated as a cause of symptoms. As previously mentioned, lash ptosis is a prominent clinical finding in FES where lashes are directed downward as a result of eyelid laxity.[7,13] In severe cases, trichiasis is seen with subsequent corneal abrasions.[13] All these factors have the potential to develop into corneal ulceration, scarring, vascularization with pannus and, rarely, corneal perforation.[1,3,13,14] Loss of elasticity, sagging and wrinkling of the facial skin on the same side as the FES are also often seen, giving the patient the appearance of premature aging or palsy on the contralateral side of the face.[7] In addition to laxity of the adnexal tissue and lids themselves, FES affects the eye in several other ways.

To see a video of floppy lid syndrome, visit www.reviewofoptometry.com, or scan the QR code.

Corneal complications in FES often stem from multifactorial processes.[3,8,12] Superficial punctate keratopathy is nearly always present in FES due to direct trauma to the corneal surface as a result of everted lids during sleep.[8,12] Involvement of the upper part of the cornea differentiates FES from nocturnal lagophthalmos, which tends to compromise the inferior third of the cornea. In addition, poor upper lid apposition to the globe can lead to poor ocular surface wetting and subsequent epitheliopathy. Furthermore, inflammation and abnormalities of the tarsal and accessory lacrimal gland, as a result of FES, can lead to decreased meibomian gland secretions. Dry eye syndrome and blepharitis are often a secondary manifestation of FES and may easily be assumed the primary cause of symptoms.[12] Though treatment of these two presentations may alleviate some of the patient's symptoms, it is not sufficient in addressing the primary cause. Several cases of ipsilateral keratoconus (KCN) have also been associated with FES.[3,12,13] One particular case noted that both conditions were isolated to the side on which the patient would sleep.[15]

Etiology

Unfortunately, the exact cause of FES is still unknown, but several theories exist. Several authors believe poor lid apposition between the lax eyelid and globe results in mechanical conjunctival irritation.[1,12,16,17] These studies show cases of patients sleeping on the affected side and suggest that chronic eyelid eversion may cause mechanical trauma to the tarsus.[1,9,12,15,16,18] The mechanical stress theory is further supported by the link between FES and KCN, as eye rubbing and presentation ipsilateral to the side on which the patient sleeps, are associated.[12,15,19,20] One study found a 7% prevalence of KCN in association with FES, which is significantly higher than in the general population (~0.004% to 0.6%).[20] To better understand the pathophysiology, researchers looked toward histopathology for answers.

Several histopathological studies demonstrate depleted levels of elastin within the tarsal plate in patients with FES relative to normal lids rather than changes in lid collagen between FES patients and controls.[14]

Researchers also noted reduced levels of elastin in the tarsal connective tissue, eyelid skin and ciliary roots.[18] The elastin fibers were structurally flawed and described as "moth-eaten." The same group's immunohistological findings revealed increased matrix metalloproteinase (MMP) enzymes, particularly MMP-7 and MMP-9,

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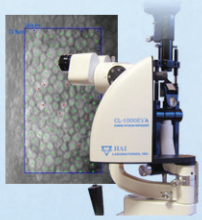

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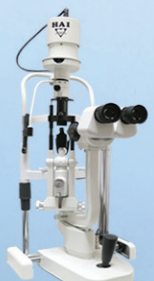

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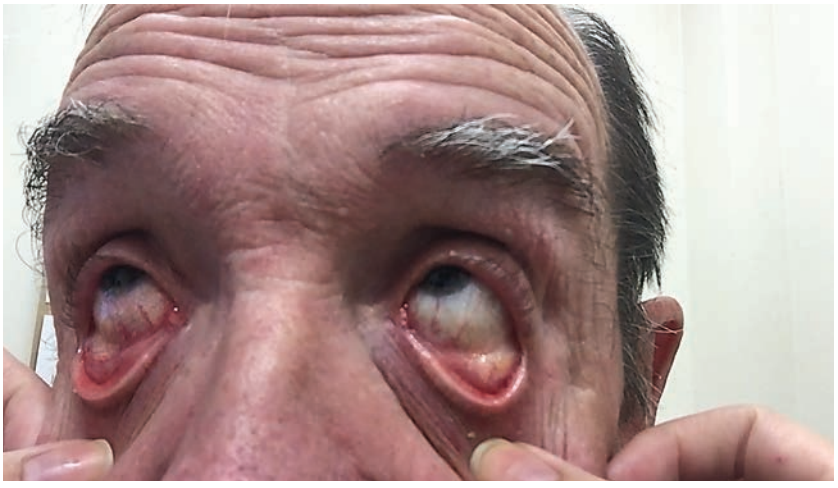
in areas without inflammatory involvement.[18] These findings were later corroborated by other groups as well.[14,18] MMP-7 and MMP-9 are specific enzymes that only control elastin degradation and have no effect on collagen fibers. A study found repeated mechanical trauma as the source of increased activity in these elastolytic enzymes.[16]

In studying the presence of the MMP-7 and MMP-9 elastolytic enzymes, another group found elevated leptin levels in proportion to body fat content.[21] Leptin is a hormone regulated by adipose cells shown to increase MMP expression and activity. Therefore, a cascade results in which an increase in leptin secretion causes increased activation of MMP-7 and MMP-9, which go on to damage elastin fibers of the tarsal plate in many of the obese patient population. Ischemia-reperfusion injury has also been proposed as a cause due to reports that it may also lead to upregulation of MMPs.[3] Interestingly, elevated MMP levels have also been noted in KCN cases, suggesting that FES and KCN have a partially shared underlying pathological mechanism that warrants further investigation.[12,15]

FES Comorbidities

Several systemic conditions are linked to FES findings, most commonly obesity (43%), hypertension (13%), diabetes mellitus (12%) and obstructive sleep apnea (85%).[1,3,5,7] The latter three conditions are common in patients who are obese and may not represent independent risk factors.[19] One of the most consistently reported associations is obstructive sleep apnea-hypopnea syndrome (OSAHS).[6-8,13,19,20,22] Investigators found the prevalence of OSAHS in patients with strictly defined FES higher than in the general population.[20] Although only 2% to 16% of OSAHS patients suffer from FES, the majority of patients with FES do suffer from symptoms of OSAHS.[5,7,9] FES is often seen as an indicator of severe OSAHS.[5,8] In addition to daytime somnolence, OSAHS carries significant risks of morbidity, with a high reported incidence of cardiovascular and cerebral sequelae.[8,23]



Among other issues, floppy eyelid syndrome is frequently associated with superficial punctate keratopathy due to direct trauma to the corneal surface from everted lids during sleep.

Preoperative FES

It may serve as an additional motivation for patients to get evaluated for OSAHS in a sleep study to tell them that early detection and management of FES will decrease contraindications and complications associated with anterior segment surgeries, such as cataract extraction and refractive correction. Blepharitis and keratoconjunctivitis, commonly associated findings in FES, left untreated preoperatively make patients more susceptible to postoperative endophthalmitis.[6,13,24] In addition, the patient will have to wear a shield during the entire healing process to prevent spontaneous lid eversion, which could lead to direct trauma to the incision wound and the already compromised corneal surface.

Blepharoptosis after cataract surgery has long been documented.[25] Trauma to the levator muscle from anesthetic injection, use of superior rectus bridle sutures, postoperative lid edema or prolonged patching may result in ptosis.[25-28] Many studies suggest the cause of blepharoptosis is multifactorial.[25,26,28] However, the use of only topical anesthetics in refractive surgery suggests that the rigid eye speculum may strongly contribute to developing postoperative lid laxity.

For refractive and cataract surgery to be performed safely and accurately, some form of lid retraction is necessary. In most cases, ptosis will improve with time, but in cases of an already compromised tarsal plate, healing may take much longer, or not occur at all. The most common form of postoperative ptosis is aponeurotic, meaning there is dehiscence or disinsertion of the levator aponeurosis from the tarsus.[25,27] Patients with FES have an already weakened tarsal plate, further increasing the risk of developing postoperative ptosis.

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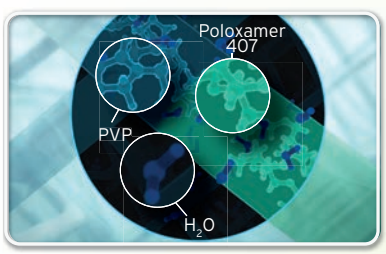

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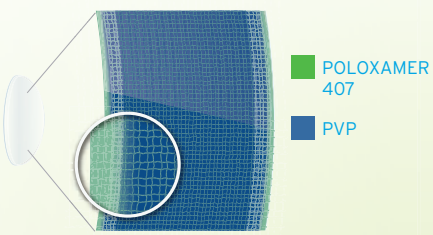

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REFERENCE: 1. Multiple-Packaged Lenses Comparison, Tyler's Quarterly – Professional Edition, September 2013 **2**. Twenty-two subjects participated in a randomized, double masked, contralateral eye study to evaluate water loss of Biotrue ONEday, 1-Day Acuvue Moist, 1-Day Acuvue TruEye contact lenses. After 4,8,12, and 16 hours of wear, lenses were removed and immediately weighed (wet weight). The lenses were then completely dried and reweighed (dry wet). The percent water loss was then calculated for each lens from the wet and dry weights.

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Complaints of dry eye will likely worsen after any anterior segment operation, and a poorly apposed lid will not allow complete and consistent lubrication across the ocular surface.

Surgical Treatment Options

Surgically, tightening the eyelids can reduce symptoms and signs of FES, as well as preserve ocular surface integrity.[29] Surgical intervention options include: full-thickness wedge excision, lateral tarsal strip, lateral canthal tendon plication and lateral tarsorrhaphy. The procedure can involve full-thickness, horizontal resection and shortening of the loose redundant tissue or tightening of the canthal tendons to improve lid apposition to the globe.[7,8] The goal is to prevent further spontaneous eversion of the lid during sleep. Studies show this surgery, in conjuction with management of OSAHS, may decrease instances of recurring FES.[7,8]

Patients with FES may benefit from sleep studies to determine if they have OSAHS. The eye care provider should notify the patient's primary physician about the findings and their associated systemic concerns. Then either the eye care provider or the primary care physician can refer the patient for a polysomnography, also known as a sleep study.[30] Patients should be prepared to stay overnight at these sleep centers where their brain activity, eye movement, heart rate, blood pressure, blood oxygen levels and volume of air inhaled and exhaled are recorded.[30] If the patient is indeed diagnosed with OSAHS, the primary care physician may schedule a continuous positive airway pressure (CPAP) titration where a technician will adjust the airflow through the CPAP machine to find the most optimal settings while the patient sleeps.[30] Since OSAHS involves shallow or pauses in breathing, the CPAP machine uses mild air pressure to maintain an open airway at night. Some patients may be required to return to study centers several times before the optimal settings for their CPAP machine is determined.[30]

Direct management of patients' systemic condition has proven to greatly reduce symptoms; in fact, CPAP treatment alone has shown to improve symptoms of both OSAHS and FES.[22] Patients with FES who were not treated for their OSAHS may actually develop recurrent signs of FES months or years after surgical tightening of their eyelids.[7] By screening for patients who are at potential risk for OSAHS, you can maximize efficacy of medical and surgical treatments. Close comanagement with the operating ophthalmologist is important to guarantee best possible outcomes for any surgeries in or around the globe.

Sleep History Questionnaire

One research team suggested offering a sleep history questionnaire for at-risk patients with the following questions:[11]

1. Do you have trouble sleeping at night? Why (heart failure, urination)?
2. Does someone sleep close enough to you to hear any nighttime noise, such as snoring?
3. Are you sleepy during the daytime or do you fall asleep at times when you should not?
4. Do you snore or have frequent awakenings? Why (urination, other)?
5. Do you have frequent headaches, especially after awakening?
6. Do you have a known sleep disorder or have you ever had a sleep study (polysomnography)?

If a patient were to answer yes to any of the above questions, it may suggest an issue with sleep-disturbed breathing. Careful evaluation of eyelids, eyelashes and adnexa of patients with persistent ocular surface complaints that are nonresponsive to topical lubrication is strongly recommended.

Non-Surgical Treatment Options

Conservative methods of treatment include shielding, patching or taping the eyes at night. These options do nothing to address the root cause of symptoms, but mechanically protecting the eyes at night often improves symptoms and reduces incidence of the suspected mechanical trauma associated with the papillary conjunctival change along with punctate keratopathy.

However, patients may report discomfort with these treatments, and compliance is subsequently affected. Tear substitutes, gels and bland ointments are only temporary treatments and only treat the dry eye component of the condition. The ester-based steroid loteprednol etabonate is also an alternative management of ocular surface and lid inflammation in FES but may be financially challenging for patients to stay on long-term.

In cases when ketone-based topical steroid drops are used to treat ocular surface inflammation, the increased risks of developing glaucoma, early cataracts and ocular infections are still a concern.

Close examination of the laxity, appearance and position of the tarsus should become a routine part of each dry eye and anterior segment evaluation prior to surgery.

Although floppy eyelid syndrome itself is not life threatening, it can lead to serious sight-threatening conditions and be an indicator of fatal systemic conditions. Poor response to usual dry eye treatments or a positive response to

sleep history questions or both, could indicate OSAHS, which is linked to morbid systemic conditions. FES is therefore predictive of OSAHS and should signify the primary eye care provider to consider a referral to evaluate the sleep habits of presenting patients. ■

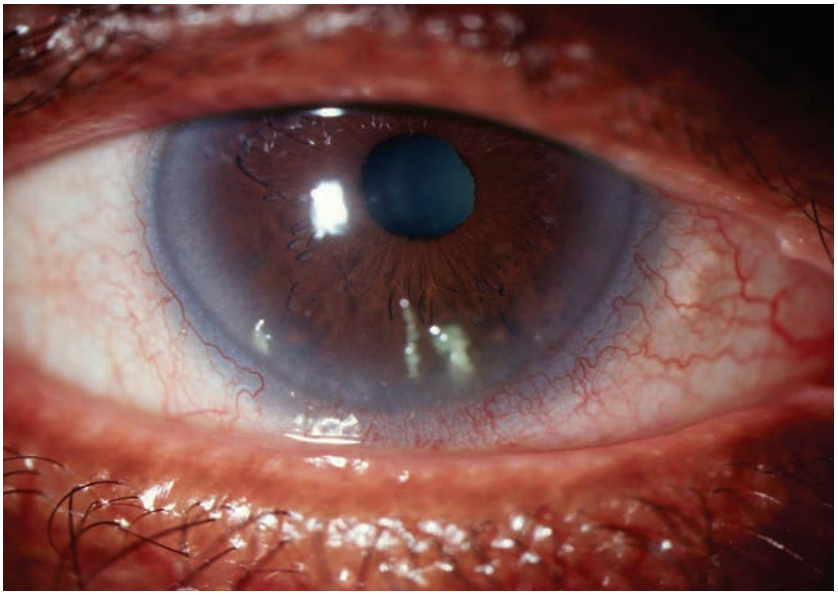
Dr. Roan graduated from the University of Missouri-St.Louis, College of Optometry and completed her residency at the Jonathan M. Wainwright VAMC.

1. Culbertson W, Ostler H. The floppy eyelid syndrome. Am J Ophthalmol. 1981;92:568-75.
2. Netland P, Sugrue S, Albert D, Shore J. Histopathologic features of the floppy eyelid syndrome. Ophthalmology. 1994 Jan;101(1):174-81.
3. Culbertson W, Tseng S. Corneal disorders in floppy eyelid syndrome. Cornea. 1994;13:33-42.
4. Paciuc M, Mier M. A woman with the floppy eyelid syndrome. Am J Ophthalmol. 1982;93:255-6.
5. Muniesa M, Huerva V, Sanchex-De-La-Torre M, et al. The relationship between floppy eyelid syndrome and obstructive sleep apnoea. Br J Ophthal. 2013 Nov;97(11):1387-90.
6. Woog J. Obstructive sleep apnea and floppy eyelid syndrome. Am J Ophthalmol. 1990:110: 223-225.
7. Mcnab A. The eye and sleep apnea. Sleep Med Rev. 2007 Aug;11(4):269-76.
8. Mcnab A. Floppy eyelid syndrome and obstructive sleep apnea. Ophthal Plast Reconstr Surg. 1997 Jun;13(2):98-114.
9. Karger R, White W, Park W, et al. Prevalence of floppy eyelid syndrome in obstructive sleep apnea-hypopnea syndrome. Ophthalmology. 2006;113:1669-74.
10. Rossiter J, Ellingham R, Hakin K, Twomey J. Corneal melt and perforation secondary to floppy eyelid syndrome in the presence of rheumatoid arthritis. Br J Ophthalmol. 2002;86:483.
11. Abdal H, Pizzimenti J, Purvis C. The eye in sleep apnea syndrome. Sleep Med. 2006;7(2):107–115.
12. Ezra D, Beaconsfield M, Collin R. Floppy eyelid syndrome: stretching the limits. Surv Ophthalmol. 2010 Jan-Feb;55(1):35-46.
13. Langford J, Linberg J. A new physical finding in floppy eyelid syndrome. Ophthalmology. 1998 Jan;105(1):165-9.
14. Netland P, Sugrue S, Albert D, Shore J. Histopathologic features of floppy eyelid syndrome. Involvement of tarsal elastin. Ophthalmology. 1994:101:174-81.
15. Donnenfeld E, Perry H, Gibralter R, et al. Keratoconus Associated with Floppy Eyelld Syndrome. Ophthalmology. 1991 Nov;98(11):1674-8.
16. Swartz M, Tschumperlin D, Kamm R, Drazen J. Mechanical stress is communicated between different cell types to elicit matrix remodeling. Proc Natl Acad Sci USA. 2001;98:6180-5.
17. Liu D, Di Pascuale M, Sawai J. Tear film dynamics in floppy eyelid syndrome. Invest Ophthalmol Vis Sci. 2005 Apr;46(4):1188-94.
18. Schöltzer-Schrehardt U, Stojkovic M, Hofmann-Rummelt C,et al. The pathogenesis of floppy eyelid syndrome. Involvement of matrix metallopoteinases in elastic fiber degradation. Ophthalmology. 2005;112:694-704.
19. Ezra D, Beaconsfield M, Sira M, et al. The associations of floppy eyelid syndrome: A case control study. Ophthalmology. 2010 Apr;117(4):831-8.
20. Fowler A, Dutton J. Floppy eyelid syndrome as a subset of lax eyelid conditions: relationships and clinical relevance (an ASOPRS thesis). Ophthal Plast Reconstr Surg. 2010 May-Jun;26(3):195-204.
21. Taban M, Perry J. Plasma Leptin levels in patients with floppy eyelid syndrome. Ophthal Plast Recontr Surg. 2006;22:375-7.
22. McNab A. Reversal of floppy eyelid syndrome with treatment of obstructive sleep apnoea. Clin Experiment Ophthalmol. 2000; 28:125-6.
23. Hashim S, Al Mansouri F, Farouk M, et al. Prevalence of glaucoma in patients with moderate to severe obstructive sleep apnea: ocular morbidity and outcomes in a 3 year follow-up study. Eye (Lond). 2014 Nov;28(11):1304-9.
24. Asadolah M, Djalilian A. Cataract surgery in the face of ocular surface disease. Current Opinion in Ophthalmology. 2012;23:68–72.
25. Paris G, Quickert M. Disinsertion of the aponeurosis of the levator palpebrae superioris muscle after cataract extraction. Am J Ophthalmol. 1976;81:337-40.
26. Kuo I. The effect of ptosis on cataract surgical planning. Case Rep Ophthalmol. 2015 Apr;6(1):132-8.
27. Deady J, Price N, Sutton G. Ptosis following cataract and trabeculectomy surgery. Br J Ophthal. 1989 Apr;73(4):283-5.
28. Singh S, Sekhar G, Gupta S. Etiology of ptosis after cataract surgery. J Cataract Refract Surg. 1997 Nov;23(9):1409-13.
29. Ezra D, Beaconsfield M, Sira M, et al. Long-term Outcomes of Surgical Approaches to the Treatment of Floppy Eyelid Syndrome. Ophthalmology. 2010 Apr;117(4):839-46.
30. Types of Sleep Studies. NHLBI, NIH. www.nhlbi.nih.gov/health/health-topics/topics/slpst/types. Accessed Sept. 10, 2015.

Customized Solutions for the DRY EYE PATIENT

When conventional therapies are insufficient, it may be time to turn to compounding pharmacies. **By Alan G. Kabat, OD**

Ask any primary eye care provider about dry eye disease and they'll likely tell you it's an issue they encounter daily. Familiar scenarios include patients who present with "the itchy-burnies," the "sore, chronically red eyes" or "intermittently blurred vision." Symptoms may be dramatically worse upon awakening for some, while for others the symptoms are more intense at the end of the day. Environmental considerations, such as ambient temperature, altitude, relative humidity, air conditioning, forced-air heating and airborne pollutants, may further confound matters. Sustained visual tasks, such as reading, driving or watching television, may constitute the focal point of the patient's complaints. Often too, the habitual or excessive use of electronic devices is consistent with more overtly symptomatic patients.



Filamentary keratitis is often seen as a complication of prolonged dry eye disease. N-acetylcysteine solution 5% to 10% used four times daily may help to alleviate this condition by providing both a mucolytic and anti-inflammatory effect.

Because our definition of dry eye disease is continuously evolving, determining its precise prevalence in the general population is difficult. One recent study demonstrates a self-reported prevalence of dry eye in 14.5% of subjects.[1] The

disease is more common in women (17.9%) than men (10.5%), according to the report.[1] Earlier studies show an estimated prevalence of 5% to 30% of the population aged 50 and older.[2,3] Of course, these values are dependent upon the inclusion criteria of individual studies, so wide variability is expected. But experts do tend to agree that the prevalence of dry eye disease is on the rise, presumably due to:

(1) Aging "baby boomers."

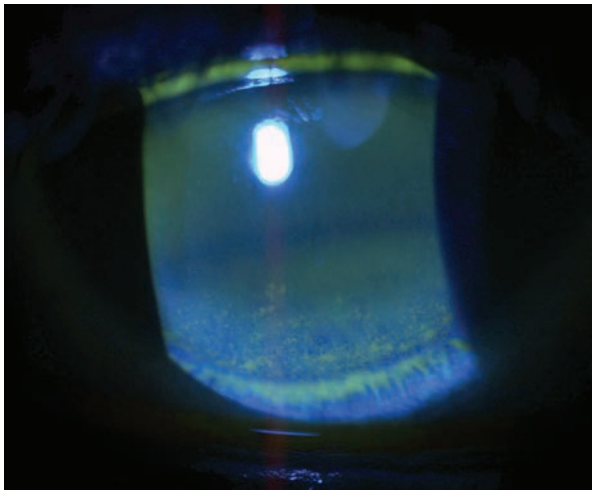
(2) Increased engagement with visual tasking.

(3) Worsening environmental conditions.

(4) Increased awareness of dry eye symptoms among patients.[4]

While the mainstay of treatment for most patients involves the use of ophthalmic lubricants—more commonly referred to as artificial tears—this therapy is often insufficient to thoroughly assuage the symptoms of moderate to severe dry eye disease. Additionally, the wide diversity of product attributes, which include such considerations as the formulation's active ingredients (e.g., carboxymethylcellulose, hydroxypropyl methylcellulose, polyethylene glycol, polyvinyl alcohol), inactive ingredients (e.g., hydroxypropyl guar, hyaluronic acid, castor oil, edetate disodium), viscosity, osmolarity and preservatives (or lack thereof) can make for a complex management decision. Unfortunately, artificial tear selection is often more a matter of what samples are most readily available, rather than what is actually most appropriate for the patient.

Another popular management option is Restasis (0.05% cyclosporine A ophthalmic emulsion, Allergan). At present, Restasis is the only prescription medication specifically approved by the Food and Drug Administration to treat dry eye disease. While several other drug candidates have been tested and submitted over the last 12 years, none have yet been able to demonstrate the required improvement in signs and symptoms mandated by the FDA. And while Restasis has been extremely successful in terms of prescriptions written and units sold, clinicians still encounter many patients who fail to achieve complete relief with this medication. Thus, practitioners who deal with dry eye disease on a regular basis are often forced to identify alternative therapies. Many turn to medications that are FDA-approved for other conditions, but are not specifically indicated for dry eye disease. These off-label therapies commonly include topical corticosteroids (e.g., loteprednol), oral antibiotics (e.g., doxycycline) and oral cholinergic agonists (e.g., pilocarpine).[5]



Advanced dry eye disease with ocular surface decompensation. For such presentations unresponsive to conventional therapy, options may include 0.2% cyclosporine ophthalmic ointment, 0.02% tacrolimus aqueous suspension or 1% medroxyprogesterone acetate solution.

Pharmaceutical compounding offers quite a different approach to the management of dry eye disease.

Compounding pharmacies provide much more than commercially available products. These specialized facilities actually formulate medications to the precise specifications of the physician. Individualized treatment of this type allows doctors and patients access to:[6]

(1) Alternative drug forms.

(2) Alternative drug concentrations.

(3) Alternative drug ingredients.

(4) Alternative drug formulations.

In other words, compounding pharmacists have the capability to produce far more diverse and patient-specific medications than the aforementioned treatment modalities for dry eye. Such pharmacists may be able to use drugs not currently available in topical form, but which are used systemically for similar pathologies. Likewise, they may formulate a higher concentration of a medication than is traditionally used for the average individual.

Compounding pharmacists can also change a formulation by adjusting the components responsible for intolerance or hypersensitivity, such as the preservative or the vehicle itself. Finally, they may be able to combine two or more active agents that are otherwise only available separately; this can enhance the efficacy of the final product while simultaneously simplifying the treatment regimen for the patient.

Let's look at some of the more commonly compounded products that are used to manage dry eye disease today.

Anti-inflammatory Agents

The notion that dry eye disease is inflammatory in nature has been widely accepted for some time. In 2007, the Report of the International Dry Eye WorkShop stated emphatically that dry eye disease "is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[7] Anti-inflammatory agents used in the treatment of dry eye disease include cyclosporine, tacrolimus and nonpreserved corticosteroids.

Cyclosporine. While Restasis has been commercially available in the United States since 2003, the use of topical ophthalmic cyclosporine A dates back over 25 years. Initially employed as a steroid-sparing agent to prevent corneal transplant rejection, physicians soon realized that this compound could also help to alleviate the discomfort associated with dry eye disease.[8] Researchers believe cyclosporine works by inhibiting T-cell activation and down-regulating inflammatory cytokines in the conjunctiva and lacrimal gland, thereby enhancing tear production.[9,10] Additionally, this medication can increase goblet cell density while decreasing epithelial cell apoptosis.[11] However, since cyclosporine is not water-soluble, early ophthalmic formulas had to be prepared using corn or peanut oil and at much higher concentrations.

Today, a number of options exist for patients who may benefit from topical cyclosporine but who have not had success with Restasis. Some of the more popular formulations include cyclosporine 0.05% in cyclodextran solution, cyclosporine 0.5% to 2% suspension in gum cellulose and cyclosporine 0.2% ophthalmic ointment. These alternative products are designed to improve tolerability through the use of different vehicles, and in some cases to enhance efficacy by using a higher concentration of the drug. Another option is cyclosporine 0.05% in combination with dexamethasone 0.01% (in cyclodextran solution). This formulation capitalizes on dexamethasone's much faster onset of action, helping to bypass the delayed effects of cyclosporine alone. These products may be used from two to four times daily, depending upon the severity of the condition. Cyclosporine ophthalmic ointment can be used in place of Restasis for those patients who prefer it, or as an adjunctive overnight therapy to Restasis.[6]

Tacrolimus. This drug has a mechanism of action that is similar to cyclosporine, but its potency in vitro has been shown to be significantly greater.[12,13] A calcineurin inhibitor, tacrolimus suppresses T- and B-lymphocyte activation, thereby preventing the release of inflammatory cytokines.[14] Numerous studies have demonstrated the efficacy of this agent in the treatment of dry eye disease.[15-17] While topical tacrolimus cream and ointment is commercially available for conditions like atopic dermatitis, the only way to obtain ophthalmic formulations of this drug is through a compounding pharmacy. Tacrolimus 0.02% eye drops represent a comparatively inexpensive option (costing roughly 40% less than 1% cyclosporine suspension) for patients who cannot tolerate or are inadequately controlled with cyclosporine. A 0.02% ointment is also available for alternative or complementary dry eye therapy. Typical dosing for ophthalmic tacrolimus is three to four times daily.

Corticosteroids. A seminal publication demonstrates that topical corticosteroid therapy was effective for patients with keratoconjunctivitis sicca whose symptoms persisted despite maximum tear replacement therapy.[18] Today, corticosteroids are frequently used in the treatment of dry eye disease. Unfortunately, all of the commercially-available steroid drops (e.g., Lotemax, Vexol, FML) in the United States contain preservatives, and this can be a deterrent for patients with hypersensitivity issues. Compounding pharmacies can prepare a variety of nonpreserved topical corticosteroid preparations. Some of the more popular options include dexamethasone sodium phosphate, loteprednol etabonate, methylprednisolone sodium succinate, prednisolone sodium phosphate and prednisolone acetate. These products can be compounded at various strengths and in numerous vehicles, depending upon the need. At lower concentrations, nonpreserved steroid drops may be used for longer durations of time with less concern for complications.

Hormonal compounds. There is clear and abundant evidence that androgens, estrogens and progestins play a role in autoregulation of the ocular surface and tear film.[19-27] These sex hormones are all produced to a greater or lesser degree by the testes, ovaries and adrenal glands, although androgens are traditionally thought of as "male" hormones while estrogens and progestins are regarded as "female" hormones. In general, androgens such as testosterone appear to have a protective, stabilizing effect on dry eye disease by promoting secretions from both the meibomian glands and lacrimal glands.[24,25] The role of estrogens and progestins is less clear, but these hormones are believed to be involved primarily in the regulation of secretory gland and ocular surface inflammation.[26,27]

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Prolonged use of ALREX® is associated with several warnings and precautions, including glaucoma with optic nerve damage, defects in visual acuity, cataract formation, secondary ocular infections, and exacerbation or prolongation of viral ocular infections (including herpes simplex).

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

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

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

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use Alrex® (loteprednol etabonate ophthalmic suspension 0.2%) safely and effectively. See full prescribing information for Alrex.

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ALREX Ophthalmic Suspension is indicated for the temporary relief of the signs and symptoms of seasonal allergic conjunctivitis.

CONTRAINDICATIONS
ALREX, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures. ALREX is also contraindicated in individuals with known or suspected hypersensitivity to any of the ingredients of this preparation and to other corticosteroids.

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

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

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

PRECAUTIONS
General: For ophthalmic use only. The initial prescription and renewal of the medication order beyond 14 days should be made by a physician only after examination of the patient with the aid of magnification, such as slit lamp biomicroscopy and, where appropriate, fluorescein staining.

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

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

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

Information for Patients: This product is sterile when packaged. Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the suspension. If redness or itching becomes aggravated, the patient should be advised to consult a physician.

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

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with up to 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (1500 and 750 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender.

Pregnancy: Teratogenic effects: Pregnancy Category C. Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (85 times the maximum daily clinical dose), a dose which caused no maternal toxicity. The no-observed-effect-level (NOEL) for these effects was 0.5 mg/kg/day (15 times the maximum daily clinical dose). Oral treatment of rats during organogenesis resulted in teratogenicity (absent innominate artery at ≥5 mg/kg/day doses, and cleft palate and umbilical hernia at ≥50 mg/kg/day) and embryotoxicity (increased postimplantation losses at 100 mg/kg/day and decreased fetal body weight and skeletal ossification with ≥50 mg/kg/day). Treatment of rats with 0.5 mg/kg/day (15 times the maximum clinical dose) during organogenesis did not result in any reproductive toxicity. Loteprednol etabonate was maternally toxic (significantly reduced body weight gain during treatment) when administered to pregnant rats during organogenesis at doses of ≥5 mg/kg/day.

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

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

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

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

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

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

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

In a summation of controlled, randomized studies of individuals treated for 28 days or longer with loteprednol etabonate, the incidence of significant elevation of intraocular pressure (≥10 mm Hg) was 2% (15/901) among patients receiving loteprednol etabonate, 7% (11/164) among patients receiving 1% prednisolone acetate and 0.5% (3/583) among patients receiving placebo. Among the smaller group of patients who were studied with ALREX, the incidence of clinically significant increases in IOP (≥10 mm Hg) was 1% (1/133) with ALREX and 1% (1/135) with placebo.

DOSAGE AND ADMINISTRATION
SHAKE VIGOROUSLY BEFORE USING.
One drop instilled into the affected eye(s) four times daily.

Revised: August 2013.

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Testosterone. Of the various hormonal compounds available for dry eye therapy, testosterone is likely the most effective and widely-publicized.

Charles Connor, PhD, OD—a professor at the Rosenberg School of Optometry, University of the Incarnate Word—developed one of the earliest formulations of topical testosterone for dry eye disease. A number of studies that use his proprietary, transdermal eyelid cream (3% to 5%) have been presented.[28-33] A topical 0.5% testosterone eye drop can also be obtained via most compounding pharmacies. Testosterone supplementation appears to have its greatest impact in post-menopausal women.[32,33] Research suggests that this treatment is equally suited to patients with aqueous-deficient dry eye disease and evaporative dry eye secondary to meibomian gland dysfunction.[20,34]

Estradiol. The most compelling argument for the role of topical estradiol in the treatment of dry eye disease comes from a randomized, controlled clinical trial published in 1998.[35] Eighty-four post-menopausal women on systemic hormone replacement therapy were treated with a 0.00025% suspension of 17ß-estradiol four times daily for four months. The results of this study show improved subjective and objective dry eye measures, both of which were statistically significant when compared to baseline, and to a control group using artificial tears.[35] A United States patent (also filed in 1998) suggested that solutions of 0.05% to 0.1% might be effective for treating keratoconjunctivitis sicca, even in the absence of concomitant oral estrogen therapy in post-menopausal women.[36] While no commercial formulations of estradiol have been approved by the FDA, some practitioners continue to use low-concentration estradiol drops (0.01% to 0.03%) for patients with dry eye disease.

Progesterone and medroxyprogesterone. The precise role of progestins in regulating ocular surface health is not fully understood, but they are believed to have an anti-inflammatory effect, down-regulating the expression of genes associated with immune processes.[37] In this capacity, they may work similarly to corticosteroids. Research with a 15% progesterone transdermal cream suggests this hormone could be useful in relieving dry eye symptoms and could potentially help a larger segment of the population than testosterone does.[38] A topical solution of 0.05% progesterone combined with 0.05% testosterone is available as a dry eye therapy from many compounding pharmacies, including Leiter's Compounding Pharmacy, one of the most well-known facilities of its type in the United States. Some pharmacies also carry 1% medroxyprogesterone acetate solution. This synthetic variant of progesterone has been advocated by some for dry eye, although it is most commonly employed as a collagenase inhibitor in conjunction with corneal surgery or trauma.[39,40]

Dehydroepiandrosterone. DHEA is a precursor molecule of both androgens and estrogens. It is produced by the adrenal glands and transformed in target tissues into various hormones, including testosterone and estradiol.[19,41] This compound becomes especially important later in one's life, as production of sex hormones by the testes and ovaries naturally declines.[41]

Tears vs. Serum—Tale of the Tape

The chemistry of blood plasma approximates that of tears to such a degree that serum, when properly diluted, provides an exceptional alternative to natural tears. The table below displays the similarities between these two vital solutions:[1,2]

	TEARS	SERUM
pH	7.4	7.4
Osmolality (mOsml/kg)	298-300	296
Albumin (g/100ml)	0.392	4.0 - 4.8
Epidermal Growth Factor (ng/ml)	0.2 - 3.0	0.5
Fibronectin (µg/ml)	21	205
Globulins (g/100ml)	0.2758	2.3
Lysozyme (mg/ml)	1.4	6
Surface Immunoglobulin A (µg/ml)	1190	2
Transforming Growth Factor Beta (ng/ml)	2 - 10	6 - 33
Calcium (mmol/L)	0.3 - 2.0	2.5
Potassium (mmol/L)	26 - 42	4.5
Sodium (mmol/L)	120 - 170	140
Water (%)	98.2	91
Vitamin A (mg/ml)	0.02	46
Vitamin C (mg/ml)	0.117	0.02

1. Geerling G, Maclennan S, Hartwig D. Autologous serum eye drops for ocular surface disorders. Br J Ophthalmol. 2004 Nov;88(11):1467-74.
2. Macsai M, Mojica G. Medical management of ocular surface disease. In: Holland EJ, Mannis MJ, Lee WB, eds. Ocular Surface Disease: Cornea, Conjunctiva and Tear Film. London: Elsevier, 2013. 271-81.

As with many compounded medications, there is a paucity of information in the peer-reviewed literature, but anecdotal reports suggest that 0.5% to 1% DHEA ophthalmic suspension may be of value for dry eye patients.[42,43] Presumably, its conversion into testosterone, estradiol or both at the ocular level imparts the same benefits outlined previously. Like testosterone, DHEA appears to be most effective in post-menopausal women.[44] Typical dosing is twice daily.[42]

Acetylcysteine. N-acetylcysteine (NAC) has been used medically for more than 50 years as a mucolytic agent, employed for a variety of respiratory conditions and illnesses (e.g., emphysema, cystic fibrosis).[43] In the presence of accumulated mucus, NAC serves to break the disulfide bridges of high-molecular-weight glycoproteins, resulting in reduced secretion viscosity. More recent research suggests that acetylcysteine may also play a role in alleviating oxidative stress, which, when unchecked, stimulates an inflammatory response at the cellular level.[45]

Most eye care practitioners are familiar with topical NAC as a treatment option for filamentary keratitis, a condition which is often associated with severe dry eye disease.[46,47] But this compound may do more than simply diminish mucus plaques; studies demonstrate a capacity for NAC to inhibit matrix metalloproteinase secretion and, at lower concentrations, facilitate wound healing in damaged corneal epithelial cells.[48,49] Two recent clinical trials even showed that topical acetylcysteine may be effective for patients with meibomian gland dysfunction.[50,51] Most practitioners employing NAC for ocular surface disease use solutions of 5% or 10%, four times daily. Patients should be aware that the compound may sting upon instillation, so refrigeration is recommended. The solution is also notorious for having a sulfurous or "rotten-egg" odor. Because topical NAC is prepared without a preservative, it is recommended that any unused portion be discarded after 30 days.

Autologous Serum

As we know, the initial management strategy for most forms of dry eye disease involves tear replacement therapy. Unfortunately, while artificial tears provide lubricating elements to the ocular surface, they have little inherent capacity to downregulate inflammation or facilitate healing of damaged tissues.

Natural tears contain enzymes, growth factors, proteins and vitamins that are crucial to metabolism, and many of these elements are altered or depleted in the dry eye state. Interestingly, blood plasma contains many of the same vital elements as tears, often in similar or higher concentrations.[52-55] Since the 1970s, physicians have recognized the value of blood plasma (or serum) for managing ocular surface disorders, but widespread clinical use was uncommon until about 15 years ago.[55]

Because this therapy is derived from the patient's own blood and not donor tissue, the term "autologous" serum eye drops (ASED) is commonly used. ASED are patient-specific, hypoallergenic and non-preserved, and are ideally suited for individuals with hypersensitivity issues. Blood is obtained through venipuncture and separated under sterile conditions to remove the blood elements, leaving only the serum. This serum is then diluted using preservative-free demulcent solutions, usually to a 20% concentration, and bottled in small (1mL or 3mL) eye drop containers. Because ASED drops are non-preserved, the bottles must be kept frozen until the patient is ready to use them; once thawed, they may be used for up to a week, but must be kept refrigerated between doses.[53] Frequency of dosing may vary, but typically the initial schedule is one drop every two hours while awake.

Although ASED provides an excellent option for patients with dry eye disease, it requires an elaborate process. Not all compounding pharmacies are equipped to formulate ASED; in fact, most require that the blood draw be performed elsewhere, such as a laboratory facility or university hospital. For this reason, many practitioners prefer to prescribe topical albumin.

Albumin. Albumin is one of the most abundant proteins found in blood plasma.[56-59] Human albumin can be extracted from donated blood, purified and used for a variety of medical conditions. Typically, it is administered by infusion for the treatment of hypovolemia, hypoalbuminemia, cirrhosis, ovarian hyperstimulation syndrome and hemolytic disease of the newborn.[60] In the body, albumin serves to regulate fluid distribution, bind and transport elements both crucial and harmful to metabolism (e.g., cholesterol, metal ions, fatty acids, toxins), scavenge free radicals and maintain normal capillary permeability.[61]

Like NAC, prepared 5% albumin solution can be formulated into eye drops with minimal effort using aseptic techniques. Several prospective trials using both animal and human models have demonstrated beneficial effects of albumin drops when used to treat severe dry eye and persistent corneal epithelial defects.[56-59] This 5% solution can be

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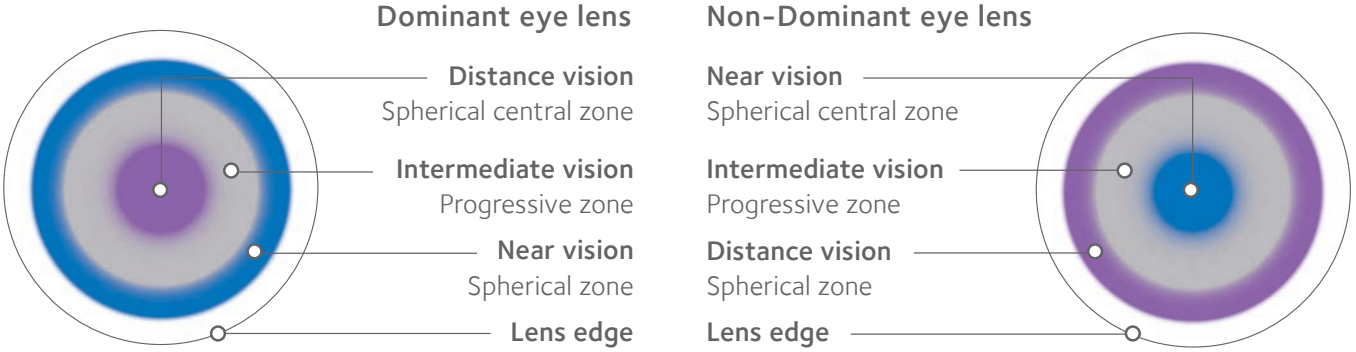

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stored in the refrigerator (at ~44° F) for up to four weeks, but any unused portion should be discarded after that.

Despite the frequency with which we encounter dry eye disease and the seemingly vast array of products designed to manage it, patients with advanced presentations can quickly exhaust these options. For such individuals, "outside-the-box" thinking is often required. Pharmaceutical compounding offers an abundance of medical therapies that may alleviate discomfort and promote ocular surface healing when conventional methods fail to deliver. ■

Disclosure: Dr. Kabat is a consultant for Bio-Tissue and serves in an advisory capacity for BlephEx, Ocusoft and TearScience. Additionally, he is a speaker for Shire and a clinical investigator for Thermi. Dr. Kabat has no financial interest in any of the products mentioned in this article.

1. Paulsen AJ, Cruickshanks KJ, Fischer ME, et al. Dry eye in the beaver dam offspring study: prevalence, risk factors, and health-related quality of life. Am J Ophthalmol. 2014 Apr;157(4):799-806.
2. The epidemiology of dry eye disease: report of the Epidemiology Subcommittee of the International Dry Eye WorkShop (2007). Ocul Surf. 2007 Apr;5(2):93-107.
3. Moss SE, Klein R, Klein BE. Prevalence of and risk factors for dry eye syndrome. Arch Ophthalmol. 2000 Sep;118(9):1264-8.
4. Lin MC, Polse KA. Improving care for patients with dry eye symptoms: See What the Experts Say. Optom Vis Sci. 2015 Jul 13. [Epub ahead of print].
5. Management and therapy of dry eye disease: report of the Management and Therapy Subcommittee of the International Dry Eye WorkShop (2007). Ocul Surf. 2007 Apr;5(2):163-78.
6. Autry JC. Mix it up: When to call a compounding pharmacist. Review of Optometry. 2012 Jul; 149(7):30-7.
7. The definition and classification of dry eye disease: report of the Definition and Classification Subcommittee of the International Dry Eye WorkShop (2007). Ocul Surf. 2007 Apr;5(2):75-92.
8.Donnenfeld ED, Perry HD. Experience expands the reach of restasis. Review of Ophthalmology. 2003 Aug;10(8):54-6.
9. Laibovitz RA, Solch S, Andriano K, et al. Pilot trial of cyclosporine 1% ophthalmic ointment in the treatment of keratoconjunctivitis sicca. Cornea. 1993 Jul;12(4):315-23.
10. Stern ME, Gao J, Siemasko KF, at al. The role of the lacrimal gland functional unit in the pathophysiology of dry eye. Exp Eye Res. 2004 Mar;78(3):409-16.
11. Sall K, Stevenson OD, Mundorf T, Reis B. Two multicenter, randomized studies of the efficacy and safety of cyclosporine ophthalmic emulsion in moderate to severe dry eye disease. CsA Phase 3 Study Group. Ophthalmology. 2000 Apr;107(4):631-9.
12. Hessen M, Akpek E. Dry eye: an inflammatory ocular disease. J Ophthal Vis Res. 2014 Apr;9(2):240-50.
13. Kino T, Hatanaka H, Hashimoto M, et al. FK-506, a novel immunosuppressant isolated from Streptomyces. I. Fermentation isolation, and physio-chemical and biological characteristics. J Antibiot (Tokyo). 1987 Sep;40(9):1249-55.
14. Fujita E, Teramura Y, Shiraga T, et al. Pharmacokinetics and tissue distribution of tacrolimus (FK506) after a single or repeated ocular instillation in rabbits. J Ocul Pharmacol Ther. 2008 Jun;24(3):309-19.
15. Sanz-Marco E, Udaondo P, Garcia-Delpech S, et al. Treatment of refractory dry eye associated with graft versus host disease with 0.03% tacrolimus eyedrops. J Ocul Pharm Ther. 2013 Oct;29(8):776-83.
16. Moscovici BK, Holzchuh R, Chiacchio BB, et al. Clinical treatment of dry eye using 0.03% tacrolimus eye drops. Cornea. 2012 Aug;31(8):945-9.
17. Moscovici BK, Holzchuh R, Sakassegawa-Naves FE, et al. Treatment of Sjogren's syndrome dry eye using 0.03% tacrolimus eye drop: Prospective double-blind randomized study. Cont Lens Anterior Eye. 2015 May 5. [Epub ahead of print].
18. Marsh P, Pflugfelder SC. Topical nonpreserved methylprednisolone therapy for keratoconjunctivitis sicca in Sjögren syndrome. Ophthalmology. 1999 Apr;106(4):811-6.
19. Truong S, Cole N, Stapleton F, Golebiowski B. Sex hormones and the dry eye. Clin Exp Optom. 2014 Jul;97(7):324-36.
20. Sullivan DA, Yamagami H, Liu M, et al. Sex steroids, the meibomian gland and evaporative dry eye. Adv Exp Med Biol. 2002;506(Pt A):389-99.
21. Suzuki T, Schaumberg DA, Sullivan BD, et al. Do estrogen and progesterone play a role in the dry eye of Sjögren's syndrome? Ann N Y Acad Sci 2002 Jun;966:223-5.
22. Pflugfelder SC. Hormonal deficiencies and dry eye. Arch Ophthalmol. 2004 Feb;122(2):273-4.
23. Wickham LA, Rocha EM, Gao J, et al. Identification and hormonal control of sex steroid receptors in the eye. Adv Exp Med Biol 1998;438:95-100.
24. Yamagami H, Schirra F, Liu M, et al. Androgen influence on gene expression in the meibomian gland. Adv Exp Med Biol. 2002;506(Pt A):477-81.
25. Sullivan D, Block L, Pena J. Influence of androgens and pituitary hormones on the structural profile and secretory activity of the lacrimal gland. Acta Ophthalmol Scand. 1996 Oct;74(5):421-35.
26. Mostafa S, Seamon V, Azzarolo AM. Influence of sex hormones and genetic predisposition in Sjögren's syndrome: A new clue to the immunopathogenesis of dry eye disease. Exp Eye Res. 2012 Mar;96(1):88-97.
27. Suzuki T, Sullivan DA. Estrogen stimulation of proinflammatory cytokine and matrix metalloproteinase gene expression in human corneal epithelial cells. Cornea. 2005 Nov;24(8):1004-9.
28. Connor CG. Transdermal testosterone delivery for the treatment of dry eye. Poster presented at the American Academy of Optometry meeting, Orlando, FL. December 12-15, 2002.
29. Connor CG. Symptomatic relief of dry eye assessed with the OSDI in patients using 5% testosterone cream. Invest Ophthalmol Vis Sci. 2005; 46(13):2032.
30. Connor CG, Jones M. Long term observation of patients using transdermal testosterone cream for the treatment of dry eye. Poster presented at the American Academy of Optometry meeting, San Diego, CA. December 8-11, 2005.
31. Connor CG. Evaporative dry eye treated with transdermal testosterone. Invest Ophthalmol Vis Sci. 2008; 49(13):120.
32. Connor CG. Androgenic cream treatment most efficacious for women 40 to 60 with dry eye. Invest Ophthalmol Vis Sci. 2009;50(13):46-56.
33. Connor CG. Testosterone cream increases contact lens wear time in menopausal dry eye patients. Invest Ophthalmol Vis Sci. 2010;51(13):62-71.
34. Schiffman RM, Bradford R, Bunnell B, et al. A multi-center, double-masked, randomized, vehicle-controlled, parallel group study to evaluate the safety and efficacy of testosterone ophthalmic solution in patients with meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2006;47(13):5608.
35. Sator MO, Joura EA, Golaszewski T, et al. Treatment of menopausal keratoconjunctivitis sicca with topical oestradiol. Br J Obstet Gynaecol. 1998 Jan;105(1):100-2.
36. Lubkin, V. Drugs for topical application of sex steroids in the treatment of dry eye syndrome, and methods of preparation and application. Virginia Lubkin, assignee. Patent US6096733 A. 10 Dec. 1998. Print.
37. Suzuki T, Schirra F, Richards SM, et al. Estrogen and progesterone control of gene expression in the mouse meibomian gland. Invest Ophthalmol Vis Sci. 2008 May;49(5):1797-808. 33.
38. Connor CG. The use of progesterone cream to treat dry eye. Invest Ophthalmol Vis Sci. 2007;48(13):378.
39. Mazzotta C, Balestrazzi A, Tarantello A, et al. The effects of medroxyprogesterone in corneal melting after Boston type I keratoprosthesis implantation. Minerva Oftalmologica. 2012 Dec;54(4):159-63.
40. Foster CS. Connective Tissue / Collagen Vascular Diseases. In: Smolin G, Foster CS, Azar DT, Dohlman CH, eds. Smolin and Thoft's The Cornea: Scientific Foundations and Clinical Practice, 4th Edition. Philadelphia: Lippincott Williams & Wilkins, 2005. 515-50.
41. Samaras N, Papadopoulou MA, Samaras D, Ongaro F. Off-label use of hormones as an antiaging strategy: a review. Clin Interv Aging. 2014 Jul 23;9:1175-86.
42. Giannoni AG. Bring Tears to Their Eyes. Optometric Management. 2012 Nov;47(11):24-31.
43. Reed K. Dry Eye Treatment: The Unusual Suspects. Review of Cornea & Contact Lenses. 2013 Mar;150(2):24-6.
44. Connor CG, Fender J. Comparison of androgenic supplemented artificial tears. Invest Ophthalmol Vis Sci. 2002;43(13):66.
45. Aitio ML. N-acetylcysteine -- passe-partout or much ado about nothing? Br J Clin Pharmacol. 2006 Jan;61(1):5-15.
46. Absolon MJ, Brown CA. Acetylcysteine in kerato-conjunctivitis sicca. Br J Ophthalmol. 1968 Apr;52(4):310-6.
47. Albietz J, Sanfilippo P, Troutbeck R, Lenton LM. Management of filamentary keratitis associated with aqueous-deficient dry eye. Optom Vis Sci. 2003 Jun;80(6):420-30.
48. Ramaesh T, Ramaesh K, Riley SC, et al. Effects of N-acetylcysteine on matrix metalloproteinase-9 secretion and cell migration of human corneal epithelial cells. Eye (Lond). 2012 Aug;26(8):1138-44.
49. Aldavood SJ, Behyar R, Sarchahi AA, et al. Effect of acetylcysteine on experimental corneal wounds in dogs. Ophthalmic Res. 2003 Nov-Dec;35(6):319-23.
50. Akyol-Salman I, Azizi S, Mumcu U, Baykal O. Efficacy of topical N-acetylcysteine in the treatment of meibomian gland dysfunction. J Ocul Pharmacol Ther. 2010 Aug;26(4):329-33.
51. Akyol-Salman I, Azizi S, Mumcu UY, Ate O, Baykal O. Comparison of the efficacy of topical N-acetyl-cysteine and a topical steroid-antibiotic combination therapy in the treatment of meibomian gland dysfunction. J Ocul Pharmacol Ther. 2012 Feb;28(1):49-52.
52. Quinto GG, Campos M, Behrens A. Autologous serum for ocular surface diseases. Arq Bras Oftalmol. 2008 Nov-Dec;71(6 Suppl):47-54.
53. Geerling G, Maclennan S, Hartwig D. Autologous serum eye drops for ocular surface disorders. Br J Ophthalmol. 2004 Nov;88(11):1467-74.
54. Pan Q, Angelina A, Zambrano A, et al. Autologous serum eye drops for dry eye. Cochrane Database Syst Rev. 2013 Aug 27;8:CD009327.
55. Azari AA, Rapuano CJ. Autologous serum eye drops for the treatment of ocular surface disease. Eye Contact Lens. 2015 May;41(3):133-40.
56. Schargus M, Kohlhaas M, Unterlauft JD. Treatment of severe ocular surface disorders with albumin eye drops. J Ocul Pharmacol Ther. 2015 Jun;31(5):291-5.
57. Higuchi A, Ueno R, Shimmura S, et al. Albumin rescues ocular epithelial cells from cell death in dry eye. Curr Eye Res. 2007 Feb;32(2):83-8.
58. Unterlauft JD, Kohlhaas M, Hofbauer I, et al. Albumin eye drops for treatment of ocular surface diseases. Ophthalmologe. 2009 Oct;106(10):932-7.
59. Shimmura S, Ueno R, Matsumoto Y, et al. Albumin as a tear supplement in the treatment of severe dry eye. Br J Ophthalmol. 2003 Oct;87(10):1279-83.
60. Boldt J. Use of albumin: an update. Br J Anaesth. 2010 Mar;104(3):276-84.
61. Caraceni P, Tufoni M, Bonavita ME. Clinical use of albumin. Blood Transfus. 2013 Sep;11 Suppl 4:s18-25.

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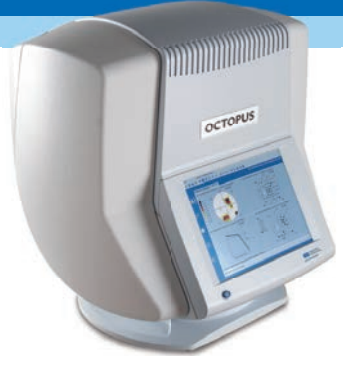

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NEUROIMAGING 101
For the Optometrist

The inside scoop on looking inside the skull.

By Christopher L. Suhr, OD, and Michael DelGiodice, OD

Based on physical signs and symptoms, optometrists can often make diagnoses in the primary optometric office. However, identifying the cause of a patient's complaint won't always be so easy. In some situations, imaging technology can help pinpoint a diagnosis and ultimately help the patient receive the best care.

The most common clinical indications for neuroimaging are: ptosis, proptosis, diplopia, ophthalmoplegia, nystagmus and optic disc abnormalities. Visual loss, visual field defects, anisocoria and other pupillary defects may also be an indication for neuroimaging, but only under particular circumstances.[1]

This article reviews various common imaging techniques and provides an overview of common neuro-ophthalmic disorders that may require neuroimaging.

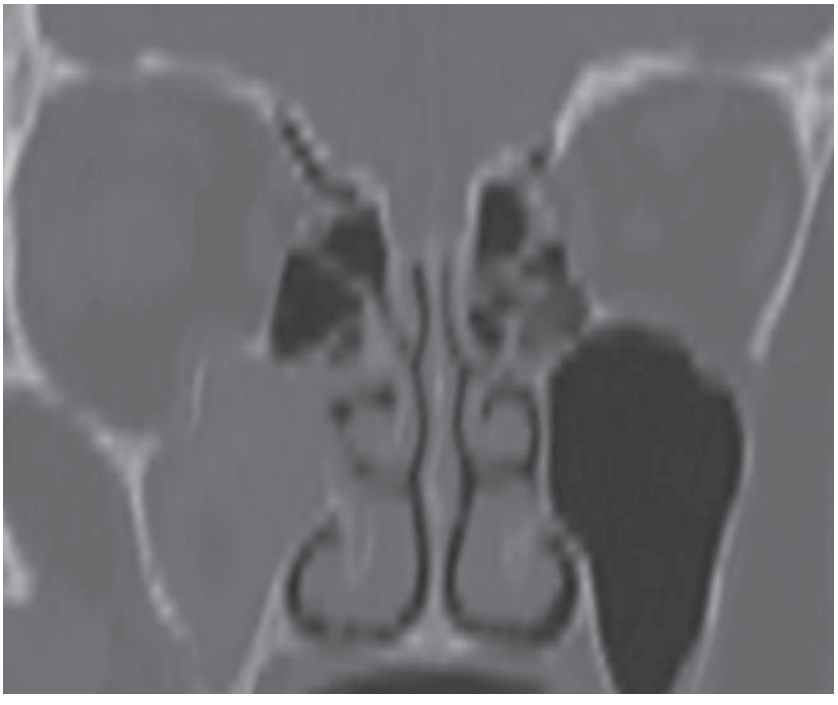
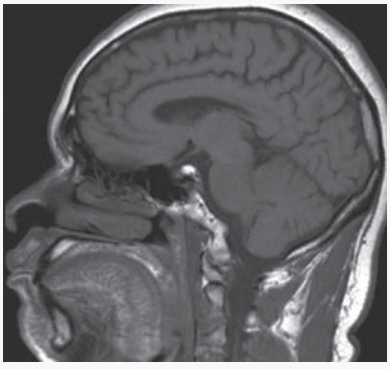
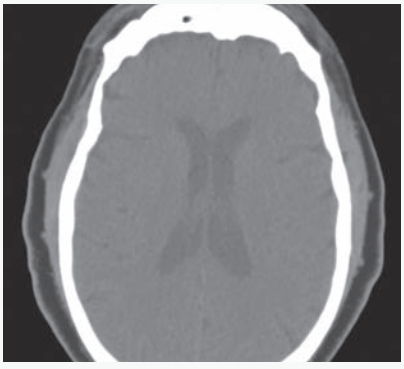
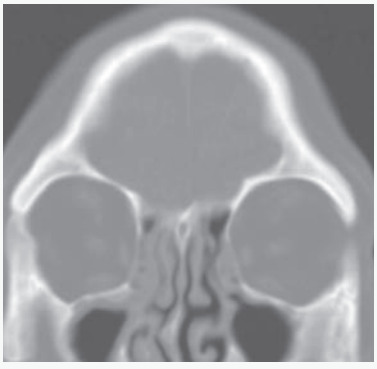


Fig. 1. The inferior aspect of the right orbit has been fractured and the maxillary sinus is opacified in this computed tomography scan, which uses both x-rays and sensors to gather data and make soft tissue visible. In these scans, air-filled pockets are black.

Viewing Angles

To understand the various types of neuroimaging, consider the image angles, or planes. At left, a sagittal section occurs when a slice is taken from the top of the body to the bottom (the Y-Z plane), and the image is viewed from the side. In the center, an axial, or transverse, section slices the body from front to back (the X-Z plane) and looks at the image from below. At right, a coronal section is obtained when a slice is taken from top to bottom (Y-X plane) and viewed from the front of the image.

Techniques

Compared with modern options, such as computed tomography (CT) and magnetic resonance imaging (MRI), x-ray is somewhat antiquated. However, it still can be beneficial to eye care professionals in instances when you want a patient to get an MRI but they have a history of metal in the eye; a plain film x-ray can detect any remaining metallic foreign body in the orbit, which would be a contraindication to MRI testing. An x-ray may also be used following facial trauma to evaluate the orbit for a fracture.

Computed Tomography

A CT scan uses both x-rays and sensors. A computer processes the data gathered by the sensors to make organs and other soft tissues viewable, which provides for greater analysis of the head and neck region. When viewing a CT scan with or without contrast, the bones are visible as white and the air-filled spaces, similar to an x-ray, appear black. Soft tissue appears in gray tones. The differences in the gray tones are a result of the soft tissue's various densities. As an example, the extraocular muscles are a lighter gray than the brain.

CT is both cost- and time-efficient in providing vital information with respect to the brain, orbit and bone. It is the imaging modality of choice for evaluating acute hemorrhage, calcification and bony abnormalities. CT scanning is painless, noninvasive, accurate and less sensitive to patient movement than MRI. Unlike MRI, CT can also be performed on patients with implanted medical devices of any kind. Disadvantages of CT include ionizing radiation, beam-hardening artifacts and iodinated contrast-induced allergy. Because of the ionizing radiation, CT imaging is not indicated in children and pregnant women. CT has a weight limit of approximately 450lbs. Although CT depicts soft tissue to some degree, an MRI would be the better study to evaluate the brain and orbital soft tissues.

You can order a CT scan with and without iodine-based contrast. CT contrast agents are used to highlight specific areas within organs, blood vessels and tissues. Following iodine injection into the bloodstream, the CT's x-ray beam generally weakens as it passes through blood vessels. Organs that have taken up the contrast are bright on the CT images. When the test is finished, the kidneys and liver eliminate the contrast from the body. Because of this, use care with patients who have kidney dysfunction, as well as individuals who may have iodine sensitivities.

In eye care, a CT scan is often used to image the bony orbit and view the anatomic position of the extraocular muscles (EOM). In instances when a patient presents with orbital trauma, a non-contrast CT of the orbit can be advantageous. Clinical signs that help to identify patients who require imaging include: resistance to forced duction, diplopia, afferent pupillary defect, bony displacement of the orbital globe, orbital crepitus, enophthalmos and subconjunctival hemorrhage, which can indicate a retrobulbar hemorrhage. One clinical caveat is when a patient suffers orbital trauma and presents with significant ecchymosis but with full range of motion and no evidence of globe displacement. In these cases,

it is helpful to determine the status of the maxillary division (V2) of the 5th cranial nerve by gently touching the fibrous end of a cotton tip applicator along the V2 distribution. Denervation within V2 will confirm the need for emergent noncontrast orbital CT to evaluate for a break in the orbital floor and to discount a hemorrhage within the sinus cavities. In patients who present with ophthalmoplegia, specifically an inability to look upward, it is prudent to suspect that there may be inferior rectus entrapment from an inferior orbital floor fracture *(Figure 1)*.

CT in Practice

The following clinical cases will highlight the importance of CT imaging:

Scenario One. A 75-year-old male presents with a chief complaint of acute peripheral vision loss. The patient recently underwent a comprehensive eye exam, and the findings at that time were unremarkable. During the current exam, pupillary responses are sluggish, and confrontation fields reveal a right homonymous defect.

You ask the patient how long he has noticed the visual field defect and whether it started acutely and note that the patient appears confused and not overly attentive. In addition, he has a mild facial paralysis. You now suspect the patient may have had a stroke, and at the completion of the exam the patient indicates an excruciating headache. With the suspicion of a cerebrovascular accident (CVA) and the new headache symptom, a STAT referral to the emergency room is warranted. At the emergency room, a CT scan will likely be done to rule out a hemorrhagic stroke due to the neurologic changes and the acute headache.[2,3]

Photo: Denise Goodwin, OD.

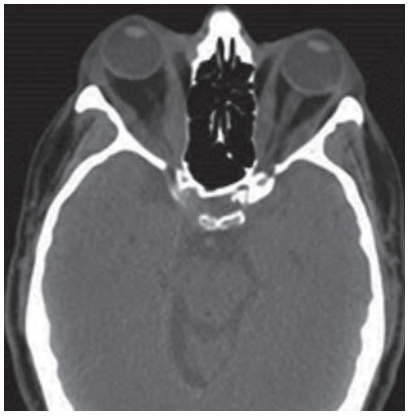


Fig. 2. The thickening of the extraocular muscle bellies ultimately causes the eyes to become proptotic.

Scenario Two. A 57-year-old female presents with a chief complaint that her eyes are "bulging out." Her systemic history is notable for hypertension and, she says, she was told she has "thyroid problems." During the clinical examination, you grossly observe bilateral exophthalmos and restricted extraocular motilities. The patient also reports concomitant diplopia. Though the patient is thin, you notice that her anterior neck appears enlarged and suspicious for goiter. Best-corrected acuities are 20/20 in both eyes. Along with the exophthalmos, there is a moderate amount of punctate keratitis. Dilated fundus evaluation is unremarkable. With these findings you suspect an orbital process most likely related to Graves' disease and thyroid eye disease. You refer the patient to an endocrinologist for further evaluation. Laboratory testing indicates a low thyroid stimulating hormone (TSH) and an elevated free thyroxine (T4) level. In this instance, a CT will allow for localization of the small bones of the orbit and can help the clinician assess the orbit and its structures for possible fracture or hemorrhage.[4,5] In this case, CT will also help the clinician evaluate for enlarged extraocular muscle bellies (*Figure 2*). The orbital coronal CT image will be helpful in evaluating the proximity of the extraocular muscles to the optic nerve at the orbital apex; compressive optic neuropathy needs to be considered in these patients.

Magnetic Resonance Imaging

MRI also provides valuable imaging of the orbital soft tissues and the brain.[6,7] Unlike a traditional x-ray or CT, MRI analyzes biological tissues using magnetic fields and radio waves, providing a detailed image of the soft tissue. Note: MRI is contraindicated in patients with potentially loose magnetic metallic items such as cochlear implants, aneurysm clips, pacemakers, defibrillators, spinal stimulators and other electronic medical devices. Some implantable devices may be MRI conditional, meaning that an MRI may be allowed under certain conditions, such as specific magnet strengths. You can contact the manufacturer of the device to get the specific conditions under which an MRI can be safely obtained and share this information with the radiology facility. When in doubt, consult with the radiologist and other health professionals involved.

The advantage of MRI is enhanced definition between different soft tissues, making it the procedure of choice for imaging the soft tissue of the brain and orbit. Unlike CT, MRI is not contraindicated in pregnant patients or children. MRI is advantageous in diagnosing early-onset cerebrovascular accident, multiple sclerosis (MS), tumors (e.g., meningioma), inflammation and infection.

As many patients feel claustrophobic in a traditional MRI, modern technology now allows for

ADD SIMBRINZA® Suspension to a PGA for Even Lower IOP[1]*

INDICATIONS AND USAGE

SIMBRINZA® (brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2% is a fixed combination indicated in the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration

The recommended dose is one drop of SIMBRINZA® Suspension in the affected eye(s) three times daily. Shake well before use. SIMBRINZA® Suspension may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

IMPORTANT SAFETY INFORMATION

Contraindications

SIMBRINZA® Suspension is contraindicated in patients who are hypersensitive to any component of this product and neonates and infants under the age of 2 years.

Warnings and Precautions

Sulfonamide Hypersensitivity Reactions—Brinzolamide is a sulfonamide, and although administered topically, is absorbed systemically. Sulfonamide attributable adverse reactions may occur. Fatalities have occurred due to severe reactions to sulfonamides. Sensitization may recur when a sulfonamide is readministered irrespective of the route of administration. If signs of serious reactions or hypersensitivity occur, discontinue the use of this preparation.

Corneal Endothelium—There is an increased potential for developing corneal edema in patients with low endothelial cell counts.

Severe Hepatic or Renal Impairment (CrCl <30 mL/min)—SIMBRINZA® Suspension has not been specifically studied in these patients and is not recommended.

Contact Lens Wear—The preservative in SIMBRINZA® Suspension, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of SIMBRINZA® Suspension but may be reinserted 15 minutes after instillation.

Severe Cardiovascular Disease—Brimonidine tartrate, a component of SIMBRINZA® Suspension, had a less than 5% mean decrease in blood pressure 2 hours after dosing in clinical studies; caution should be exercised in treating patients with severe cardiovascular disease.

Adverse Reactions

SIMBRINZA® Suspension

In two clinical trials of 3 months' duration with SIMBRINZA® Suspension, the most frequent reactions associated with its use occurring in approximately 3-5% of patients in descending order of incidence included: blurred vision, eye irritation, dysgeusia (bad taste), dry mouth, and eye allergy. Adverse reaction rates with SIMBRINZA® Suspension were comparable to those of the individual components. Treatment discontinuation, mainly due to adverse reactions, was reported in 11% of SIMBRINZA® Suspension patients.

Prescribe SIMBRINZA® Suspension as adjunctive therapy to a PGA for appropriate patients

SIMBRINZA® Suspension should be taken at least five (5) minutes apart from other topical ophthalmic drugs

Learn more at myalcon.com/simbrinza

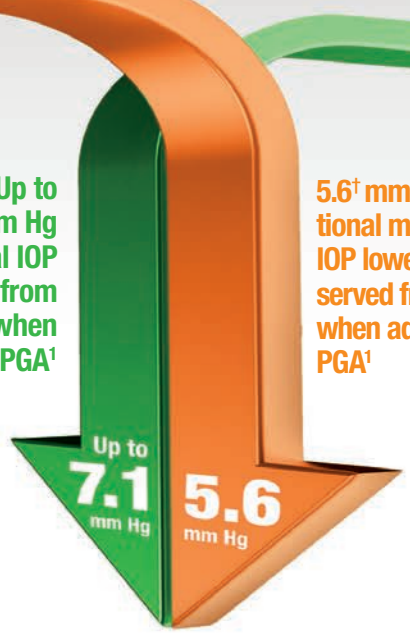
For additional information about SIMBRINZA® Suspension, please see Brief Summary of full Prescribing Information on adjacent page.

Reference: 1. Data on file, 2014.

Alcon
a Novartis company

Up to 7.1 mm Hg additional IOP reduction from baseline when added to a PGA[1]

5.6† mm Hg additional mean diurnal IOP lowering observed from baseline when added to a PGA[1]



		IOP Time Points (mm Hg)[1‡]			
Treatment Arm		8 AM	10 AM	3 PM	5 PM
PGA + SIMBRINZA® Suspension (N=83)	Baseline[§]	24.5	22.9	21.7	21.6
	Week 6	19.4	15.8	17.2	15.6
PGA + Vehicle (N=92)	Baseline[§]	24.3	22.6	21.3	21.2
	Week 6	21.5	20.3	20.0	20.1

‡Least squares means at each Week 6 time point. Treatment differences (mm Hg) and *P*-values at Week 6 time points between treatment groups were: -2.14, *P*=0.0002; -4.56, *P*<0.0001; -2.84, *P*<0.0001; -4.42, *P*<0.0001.
§Baseline (PGA Monotherapy).

		Mean Diurnal IOP (mm Hg)[1‖]
Treatment Arm		
PGA + SIMBRINZA® Suspension (N=83)	Baseline[¶]	22.7
	Week 6	17.1
PGA + Vehicle (N=92)	Baseline[¶]	22.4
	Week 6	20.5

‖Treatment difference (mm Hg) and *P*-value at Week 6 was -3.4, *P*<0.0001.
¶Baseline (PGA Monotherapy).

Study Design: A prospective, randomized, multicenter, double-blind, parallel-group study of 189 patients with open-angle glaucoma and/or ocular hypertension receiving treatment with a PGA. PGA treatment consisted of either travoprost, latanoprost, or bimatoprost. Patients in the study were randomized to adjunctive treatment with SIMBRINZA® Suspension (N=88) or vehicle (N=94). The primary efficacy endpoint was mean diurnal IOP (IOP averaged over all daily time points) at Week 6 between treatment groups. Key secondary endpoints included IOP at Week 6 for each daily time point (8 AM, 10 AM, 3 PM, and 5 PM) and mean diurnal IOP change from baseline to Week 6 between treatment groups.[1]

*PGA study-group treatment consisted of either travoprost, latanoprost, or bimatoprost.
†Treatment difference (mm Hg) and *P*-value at Week 6 was -3.7, *P*<0.0001.

SIMBRINZA®
(brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2%

BRIEF SUMMARY OF PRESCRIBING INFORMATION
INDICATIONS AND USAGE

SIMBRINZA® (brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2% is a fixed combination of a carbonic anhydrase inhibitor and an alpha 2 adrenergic receptor agonist indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION

The recommended dose is one drop of SIMBRINZA® Suspension in the affected eye(s) three times daily. Shake well before use. SIMBRINZA® Suspension may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure.
If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

DOSAGE FORMS AND STRENGTHS

Suspension containing 10 mg/mL brinzolamide and 2 mg/mL brimonidine tartrate.

CONTRAINDICATIONS

Hypersensitivity - SIMBRINZA® Suspension is contraindicated in patients who are hypersensitive to any component of this product.

Neonates and Infants (under the age of 2 years) - SIMBRINZA® Suspension is contraindicated in neonates and infants (under the age of 2 years*) see Use in Specific Populations*

WARNINGS AND PRECAUTIONS

Sulfonamide Hypersensitivity Reactions - SIMBRINZA® Suspension contains brinzolamide, a sulfonamide, and although administered topically is absorbed systemically. Therefore, the same types of adverse reactions that are attributable to sulfonamides may occur with topical administration of SIMBRINZA® Suspension. Fatalities have occurred due to severe reactions to sulfonamides including Stevens-Johnson syndrome, toxic epidermal necrolysis, fulminant hepatic necrosis, agranulocytosis, aplastic anemia, and other blood dyscrasias. Sensitization may recur when a sulfonamide is re-administered irrespective of the route of administration. If signs of serious reactions or hypersensitivity occur, discontinue the use of this preparation *[see Patient Counseling Information]*

Corneal Endothelium - Carbonic anhydrase activity has been observed in both the cytoplasm and around the plasma membranes of the corneal endothelium. There is an increased potential for developing corneal edema in patients with low endothelial cell counts. Caution should be used when prescribing SIMBRINZA® Suspension to this group of patients.

Severe Renal Impairment - SIMBRINZA® Suspension has not been specifically studied in patients with severe renal impairment (CrCl < 30 mL/min). Since brinzolamide and its metabolite are excreted predominantly by the kidney, SIMBRINZA® Suspension is not recommended in such patients.

Acute Angle-Closure Glaucoma - The management of patients with acute angle-closure glaucoma requires therapeutic interventions in addition to ocular hypotensive agents. SIMBRINZA® Suspension has not been studied in patients with acute angle-closure glaucoma.

Contact Lens Wear - The preservative in SIMBRINZA® Suspension, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of SIMBRINZA® Suspension but may be reinserted 15 minutes after instillation *[see Patient Counseling Information]*.

Severe Cardiovascular Disease - Brimonidine tartrate, a component of SIMBRINZA® Suspension, has a less than 5% mean decrease in blood pressure 2 hours after dosing in clinical studies; caution should be exercised in treating patients with severe cardiovascular disease.

Severe Hepatic Impairment - Because brimonidine tartrate, a component of SIMBRINZA® Suspension, has not been studied in patients with hepatic impairment, caution should be exercised in such patients.

Potentiation of Vascular Insufficiency - Brimonidine tartrate, a component of SIMBRINZA® Suspension, may potentiate syndromes associated with vascular insufficiency. SIMBRINZA® Suspension should be used with caution in patients with depression, cerebral or coronary insufficiency, Raynaud's phenomenon, orthostatic hypotension, or thromboangiitis obliterans.

Contamination of Topical Ophthalmic Products After Use - There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers have been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface *[see Patient Counseling Information]*.

ADVERSE REACTIONS

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

SIMBRINZA® Suspension - In two clinical trials of 3 months duration 435 patients were treated with SIMBRINZA® Suspension, and 915 were treated with the two individual components. The most frequently reported adverse reactions in patients treated with SIMBRINZA® Suspension occurring in approximately 3 to 5% of patients in descending order of incidence were blurred vision, eye irritation, dysgeusia (bad taste), dry mouth, and eye allergy. Rates of adverse reactions reported with the individual components were comparable. Treatment discontinuation, mainly due to adverse reactions, was reported in 11% of SIMBRINZA® Suspension patients.

Other adverse reactions that have been reported with the individual components during clinical trials are listed below.

Brinzolamide 1% - In clinical studies of brinzolamide ophthalmic suspension 1%, the most frequently reported adverse reactions reported in 5 to 10% of patients were blurred vision and bitter, sour or unusual taste. Adverse reactions occurring in 1 to 5% of patients were blepharitis, dermatitis, dry eye, foreign body sensation, headache, hyperemia, ocular discharge, ocular discomfort, ocular keratitis, ocular pain, ocular pruritus and rhinitis.

The following adverse reactions were reported at an incidence below 1%: allergic reactions, alopecia, chest pain, conjunctivitis, diarrhea, diplopia, dizziness, dry mouth, dyspnea, dyspepsia, eye fatigue, hypertonia, keratoconjunctivitis, keratopathy, kidney pain, lid margin crusting or sticky sensation, nausea, pharyngitis, tearing and urticaria.

Brimonidine Tartrate 0.2% - In clinical studies of brimonidine tartrate 0.2%, adverse reactions occurring in approximately 10 to 30% of the subjects, in descending order of incidence, included oral dryness, ocular hyperemia, burning and stinging, headache, blurring, foreign body sensation, fatigue/drowsiness, conjunctival follicles, ocular allergic reactions, and ocular pruritus.

Reactions occurring in approximately 3 to 9% of the subjects, in descending order included corneal staining/erosion, photophobia, eyelid erythema, ocular ache/pain, ocular dryness, tearing, upper respiratory symptoms, eyelid edema, conjunctival edema, dizziness, blepharitis, ocular irritation, gastrointestinal symptoms, asthenia, conjunctival blanching, abnormal vision and muscular pain.

The following adverse reactions were reported in less than 3% of the patients: lid crusting, conjunctival hemorrhage, abnormal taste, insomnia, conjunctival discharge, depression, hypertension, anxiety, palpitations/arrhythmias, nasal dryness and syncope.

Postmarketing Experience - The following reactions have been identified during postmarketing use of brimonidine tartrate ophthalmic solutions in clinical practice. Because they are reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reactions, which have been chosen for inclusion due to either their seriousness, frequency of reporting, possible causal connection to brimonidine tartrate ophthalmic solutions, or a combination of these factors, include: bradycardia, hypersensitivity, iritis, keratoconjunctivitis sicca, miosis, nausea, skin reactions (including erythema, eyelid pruritus, rash, and vasodilation), and tachycardia.

Apnea, bradycardia, coma, hypotension, hypothermia, hypotonia, lethargy, pallor, respiratory depression, and somnolence have been reported in infants receiving brimonidine tartrate ophthalmic solutions *[see Contraindications]*.

DRUG INTERACTIONS

Oral Carbonic Anhydrase Inhibitors - There is a potential for an additive effect on the known systemic effects of carbonic anhydrase inhibition in patients receiving an oral carbonic anhydrase inhibitor and brinzolamide ophthalmic suspension 1%, a component of SIMBRINZA® Suspension. The concomitant administration of SIMBRINZA® Suspension and oral carbonic anhydrase inhibitors is not recommended.

High-Dose Salicylate Therapy - Carbonic anhydrase inhibitors may produce acid-base and electrolyte alterations. These alterations were not reported in the clinical trials with brinzolamide ophthalmic suspension 1%. However, in patients treated with oral carbonic anhydrase inhibitors, rare instances of acid-base alterations have occurred with high-dose salicylate therapy. Therefore, the potential for such drug interactions should be considered in patients receiving SIMBRINZA® Suspension.

CNS Depressants - Although specific drug interaction studies have not been conducted with SIMBRINZA® Suspension, the possibility of an additive or potentiating effect with CNS depressants (alcohol, opiates, barbiturates, sedatives, or anesthetics) should be considered.

Antihypertensives/Cardiac Glycosides - Because brimonidine tartrate, a component of SIMBRINZA® Suspension, may reduce blood pressure, caution in using drugs such as antihypertensives and/or cardiac glycosides with SIMBRINZA® Suspension is advised.

Tricyclic Antidepressants - Tricyclic antidepressants have been reported to blunt the hypotensive effect of systemic clonidine. It is not known whether the concurrent use of these agents with SIMBRINZA® Suspension in humans can lead to resulting interference with the IOP lowering effect. Caution is advised in patients taking tricyclic antidepressants which can affect the metabolism and uptake of circulating amines.

Monoamine Oxidase Inhibitors - Monoamine oxidase (MAO) inhibitors may theoretically interfere with the metabolism of brimonidine tartrate and potentially result in an increased systemic side-effect such as hypotension. Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

USE IN SPECIFIC POPULATIONS

Pregnancy - *Pregnancy Category C:* Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1, 3, and 6 mg/kg/day (20, 60, and 120 times the recommended human ophthalmic dose) produced maternal toxicity at 6 mg/kg/day and a significant increase in the number of fetal variations, such as accessory skull bones, which was only slightly higher than the historic value at 1 and 6 mg/kg. In rats, statistically decreased body weights of fetuses from dams receiving oral doses of 18 mg/kg/day (180 times the recommended human ophthalmic dose) during gestation were proportional to the reduced maternal weight gain, with no statistically significant effects on organ or tissue development. Increases in unossified sternebrae, reduced ossification of the skull, and unossified hyoid that occurred at 6 and 18 mg/kg were not statistically significant. No treatment-related malformations were seen. Following oral administration of ^{14}C-brinzolamide to pregnant rats, radioactivity was found to cross the placenta and was present in the fetal tissues and blood.

Developmental toxicity studies performed in rats with oral doses of 0.66 mg brimonidine base/kg revealed no evidence of harm to the fetus. Dosing at this level resulted in a plasma drug concentration approximately 100 times higher than that seen in humans at the recommended human ophthalmic dose. In animal studies, brimonidine crossed the placenta and entered into the fetal circulation to a limited extent.

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

Nursing Mothers - In a study of brinzolamide in lactating rats, decreases in body weight gain in offspring at an oral dose of 15 mg/kg/day (150 times the recommended human ophthalmic dose) were observed during lactation. No other effects were observed. However, following oral administration of ^{14}C-brinzolamide to lactating rats, radioactivity was found in milk at concentrations below those in the blood and plasma. In animal studies, brimonidine was excreted in breast milk.

It is not known whether brinzolamide and brimonidine tartrate are excreted in human milk following topical ocular administration. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from SIMBRINZA® (brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2%, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use - The individual component, brinzolamide, has been studied in pediatric glaucoma patients 4 weeks to 5 years of age. The individual component, brimonidine tartrate, has been studied in pediatric patients 2 to 7 years old. Somnolence (50-83%) and decreased alertness was seen in patients 2 to 6 years old. SIMBRINZA® Suspension is contraindicated in children under the age of 2 years *[see Contraindications]*.

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

OVERDOSAGE

Although no human data are available, electrolyte imbalance, development of an acidotic state, and possible nervous system effects may occur following an oral overdose of brinzolamide. Serum electrolyte levels (particularly potassium) and blood pH levels should be monitored.

Very limited information exists on accidental ingestion of brimonidine in adults; the only adverse event reported to date has been hypotension. Symptoms of brimonidine overdose have been reported in neonates, infants, and children receiving brimonidine as part of medical treatment of congenital glaucoma or by accidental oral ingestion. Treatment of an oral overdose includes supportive and symptomatic therapy; a patent airway should be maintained.

PATIENT COUNSELING INFORMATION

Sulfonamide Reactions - Advise patients that if serious or unusual ocular or systemic reactions or signs of hypersensitivity occur, they should discontinue the use of the product and consult their physician.

Temporary Blurred Vision - Vision may be temporarily blurred following dosing with SIMBRINZA® Suspension. Care should be exercised in operating machinery or driving a motor vehicle.

Effect on Ability to Drive and Use Machinery - As with other drugs in this class, SIMBRINZA® Suspension may cause fatigue and/or drowsiness in some patients. Caution patients who engage in hazardous activities of the potential for a decrease in mental alertness.

Avoiding Contamination of the Product - Instruct patients that ocular solutions, if handled improperly or if the tip of the dispensing container contacts the eye or surrounding structures, can become contaminated by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions *[see Warnings and Precautions]*. Always replace the cap after using. If solution changes color or becomes cloudy, do not use. Do not use the product after the expiration date marked on the bottle.

Intercurrent Ocular Conditions - Advise patients that if they have ocular surgery or develop an intercurrent ocular condition (e.g., trauma or infection), they should immediately seek their physician's advice concerning the continued use of the present multidose container.

Concomitant Topical Ocular Therapy - If more than one topical ophthalmic drug is being used, the drugs should be administered at least five minutes apart.

Contact Lens Wear - The preservative in SIMBRINZA® Suspension, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of SIMBRINZA® Suspension, but may be reinserted 15 minutes after instillation.

U.S. Patent No:
6,316,441

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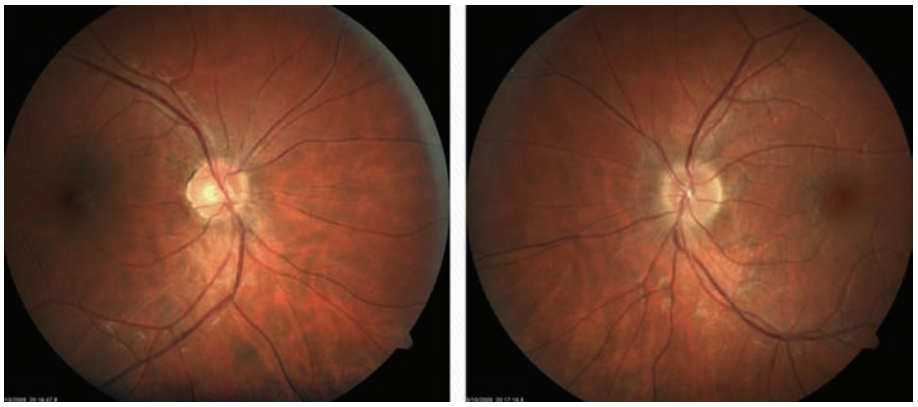

Fig. 3. A patient's fundus images showed no abnormalities of the right eye. However, in the left eye the image shows a crowded nerve with blurred margins.

an open MRI, and even a variant that allows patients to be seated. Though these options are available, there is widespread understanding that the tubular version provides better images.[8] Of course, if the patient is unable to lie flat, or cannot undergo traditional MRI testing, these options are better than not being able to get any images.[8]

MRI uses three primary techniques: proton density, T1- and T2-weighted sequences.

Proton density scans provide an image depicting the density of protons in tissue. A T1-weighted scan represents the time it takes tissue to recover from a radiofrequency pulse. With T1-weighted images, fluid is extremely dark, water-based tissue is gray and lipid-based tissues are bright. A T2-weighted scan represents the time the signal lasts after giving a radiofrequency pulse. Clinically, it results in water-based tissues being bright and lipid-based tissues appearing darker. A T2-weighted scan uses a gradient echo (GRE) sequence, which is most often used in suspected cases of intracranial microhemorrhage. These images provide good contrast between gray and white matter in the brain, iron-laden tissues and venous vessels.

Inversion recovery, fluid attenuated inversion recovery (FLAIR) and standard short T1-inversion recovery (STIR) are techniques used to suppress certain anatomic tissue. FLAIR sequences produce heavily T2-weighted images with suppression of cerebrospinal fluid, allowing for more sensitive resolution in suspected cases of demyelination or lesions anatomically close to the ventricles. Clinically speaking, this is the imaging sequence of choice when ordering an MRI for patients with optic neuritis. STIR sequence can be used with T1- or T2-weighted images to obtain fat signal suppression. Other pulse sequences of MRI include magnetization transfer contrast, diffusion-weighted, echo planar, perfusion and functional.[9]

Gadolinium contrast is often administered intravenously prior to an MRI to increase the visibility of intracranial infection, inflammation, early ischemia and meningeal lesions. It also helps to distinguish active demyelinating plaques from quiescent ones. Prior to ordering an MRI with contrast, a patient should have labs performed to check for adequate kidney function, including serum creatinine and blood urea nitrogen (BUN).[10] Individuals with a history of gadolinium allergies, those who have kidney impairment and those who take medications excreted through the kidneys are at risk when using contrast. Usually the kidney functions need to be collected within four to six weeks of the use of contrast; the exact time limitations are facility dependent. In some instances, diabetic patients who take metformin may be advised to forego taking their medications for two days prior to an MRI with contrast.[10] For orbital studies, MRI with and without contrast should be considered to rule out mass lesions or other potential findings.

MRI in Practice

An MRI may be beneficial for eye care practitioners in these scenarios:

Scenario One. A 28-year-old female presents to the office with vague visual symptoms. She indicates that she has blurry vision only intermittently. The periods of poor vision have occurred several times throughout the last five years, but since vision returns to normal after a couple weeks, she never felt the need for an eye exam. Since her vision has been reduced for the past two weeks without improvement, she felt it was necessary to have an examination. Preliminary testing revealed normal extraocular motilities and confrontation visual fields. Pupil evaluation elicited 1+ RAPD in the left eye. Red cap testing revealed desaturation of the left eye.

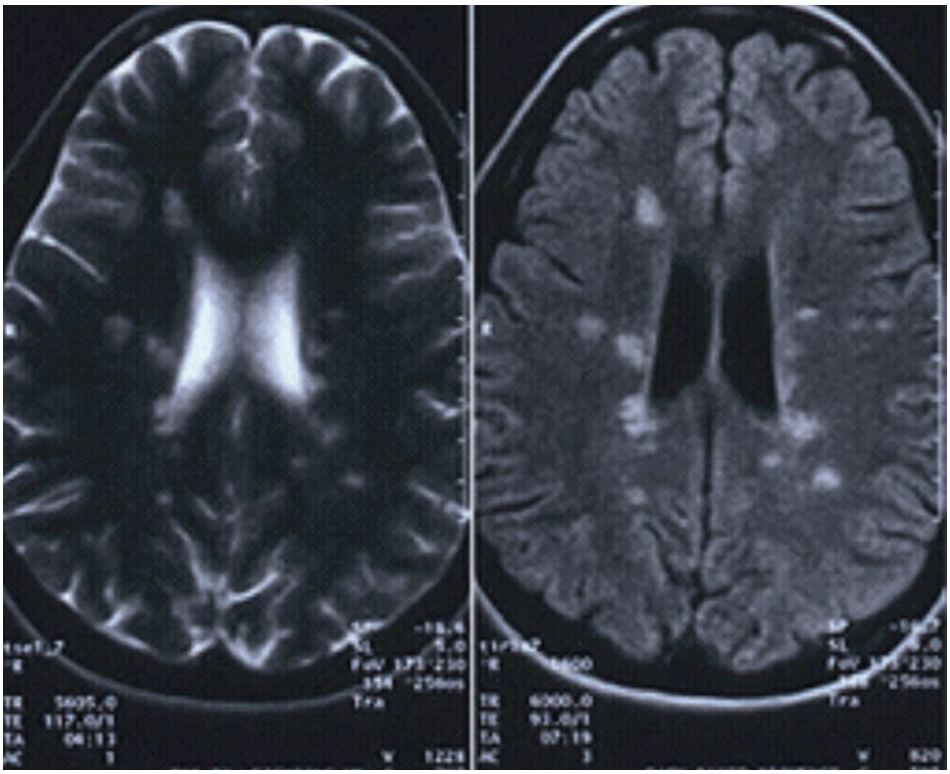

Fig. 4. The patient's MRI reveals classic white matter lesions perpendicular to the lateral ventricles, consistent with MS. These lesions are most easily seen using th FLAIR MRI sequence. T1-weighted imaging may also help identify any active lesions.

With a minimal refractive error, best-corrected visual acuities were 20/20 OD and 20/25- OS. Dilated fundus examination revealed no abnormalities of the right eye. The left eye had a crowded nerve with blurred margins (*Figure 3*). Because the macular region was unremarkable, a diagnosis of optic neuritis was suspected and a referral to a neurologist, or neuro-ophthalmologist, is necessary since optic neuritis can be associated with demyelination and MS.[11] If findings are atypical for a diagnosis of demyelinating optic neuritis, laboratory testing can rule out an infectious etiology such as Lyme disease, syphilis or tuberculosis.[12] An MRI is warranted in cases of optic neuritis to confirm the optic nerve inflammation and to look for the classic white matter lesions perpendicular to the lateral ventricles, consistent with MS (*Figure 4*).[13] The lesions associated with MS are most easily seen with the FLAIR MRI sequence. With T1-weighted imaging, active lesions may also enhance with contrast.[14] It should be noted that the patient may be relatively asymptomatic and still have lesions; MRI findings may not make the diagnosis of MS, particularly if patients are asymptomatic.[15] The imaging and proper diagnosis leads to a prompt treatment, which can speed visual recovery and maintain quality of life as best as possible, since it decreases as the disease progresses.[16]

Scenario Two. A 55-year-old male patient presents with complaints of increasing difficulty reading the newspaper, headaches and decreased vision in the right eye. Systemic health is unremarkable. Pupillary testing reveals a 1+ RAPD of the right eye and color vision deficiency in the right eye. Confrontation visual fields reveal an inferior temporal defect in the right eye. The visual field is full for the left eye. Best-corrected acuities are 20/40 OD and 20/20 OS. Anterior segment examination is unremarkable. Dilated fundus evaluation is unremarkable for the left eye, but the right optic nerve has blurred disc margins.

A clinician must consider a neurologic cause to these signs and symptoms, and neuroimaging is warranted. In this case, an MRI of the brain and orbits with and without contrast reveals an optic nerve glioma.[17]

Scenario Three. A 54-year-old male presents indicating that his progressive bifocals are inadequate for his near tasks. Preliminary testing revealed normal pupillary responses and extraocular motilities, but there was a restricted temporal visual field in each eye. Best-corrected vision was 20/25 in each eye at distance and near. Dilated fundus examination was

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 ACU-10314367-S September 2015

unremarkable for all structures except the optic discs. The optic discs showed temporal pallor bilaterally. A Humphrey visual field 24-2 was performed, revealing a superior temporal defect in each eye. Based on the clinical findings, the optometrist would suspect a lesion in the suprasellar cistern, most commonly a pituitary adenoma, and would refer for the appropriate imaging to confirm the diagnosis. Prompt confirmation of the pituitary adenoma can streamline the process to neuro-surgical intervention, which is important because continued compression of the chiasm may lead to irreversible optic nerve damage. Patients with severe headache and sudden bitemporal vision loss need to be imaged immediately to rule out pituitary apoplexy. Pituitary apoplexy and adenoma can both cause hormonal changes, as the pituitary gland is responsible for many endocrine functions.[18] Because of the risk for adrenal failure and death from untreated pituitary apoplexy, patients suspected of having this need to be sent to the ER rather than having outpatient imaging.[19]

Photo: Heather Shelsta, MD.

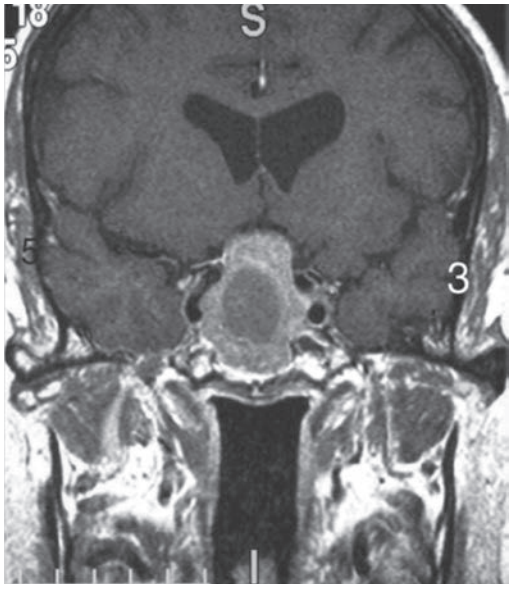

Fig. 5. T1-weighted MRI with gadolinium can help confirm a diagnosis of a pituitary adenoma.

Neurovascular Imaging

The ability to visualize the neurovascular anatomy involves expressing the blood vessels and suppressing the surrounding structures. In digital subtraction angiography (DSA) and computerized tomography angiography (CTA), iodinated intravascular contrast is injected to create a detailed analysis of the vascular system. For patients with iodine sensitivity or kidney dysfunction, magnetic resonance angiography (MRA) is a safe alternative.

CTA. Compared with DSA, both CTA and MRA are relatively noninvasive procedures for studying the vascular system. The advantages of CTA over MRA include: cost, accessibility, less time consuming, applicable for patients with implantable devices and more conducive to those with claustrophobia. Clinically, CTA can detect aneurysms as small as 1.7mm and is superior for imaging aneurysms of the head and neck, characterizing thrombi, and detecting vasospasm, arterial stenosis and carotid-cavernous fistulas. The drawbacks of CT angiography include difficult detection and delineation of cavernous sinus and posterior inferior cerebellar artery aneurysms, feeding vessels for dural carotid cavernous fistulas and risks involving radiation exposure and contrast agents.[20,21]

MRA. With MRA, a low dose of gadolinium contrast is injected into a vein, and images are acquired during the first pass of the agent through the arteries. MRA can help clinicians evaluate the extracranial circulation (i.e., carotid artery stenosis, plaques and dissections in the evaluation of transient visual loss) and intracranial circulation (i.e., aneurysms, arteriovenous malformations, occlusive disease and carotid artery fistulas). The limitations of MRA include the possibility of false-positive results in tightly wound vessel loops, as well as a tendency to exaggerate vessel stenosis.[22]

DSA. This provides images of blood vessels by inserting a tube into a large artery and threading it through the circulatory system, and then injecting the tube with contrast dye. A series of radiographs is taken as the contrast spreads throughout the arterial system. A second series of radiographs is taken as the dye exits via the venous system. DSA allows clinicians to perform endovascular treatment immediately based on the findings. However, major complications in DSA do exist, including: CVA, allergic reaction to the anesthetic or contrast medium, damage to one of the access veins or thrombosis and embolism formation. Minor complications include bleeding or bruising at the site of the dye injection.

The most common uses of DSA in neuro-ophthalmic disorders is to detect and localize small intracranial aneurysms in the presence or absence of sub-arachnoid blood.

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Neuroimaging

It is widely used in interventional neuroradiology, as it allows for detailed anatomy of the vasculature in order to introduce flexible microcatheters, balloons, coils, chemical agents and other devices.[23] Common indications are for evaluating and treating arteriovenous malformations and carotid cavernous and dural fistulas with intracranial coils.[23]

In general, MRI is the imaging modality of choice for most neuro-ophthalmological disorders, while CT is advantageous for viewing the skull, bony orbit, EOM entrapment and blood following head and facial trauma. Because MRI has many different imaging sequences, it allows for detailed assessment of soft tissue. When ordering a neuroimaging study, with the exception of T1- and T2-weighted images, it is important to provide the radiologist with specific sequences and state what you are looking for to tailor the study to the patient's needs. In addition, the nature of the suspected pathology is also important in deciding the protocol and if contrast is required. If there is any doubt as to which imaging study is recommended, consult with the neuroradiologist. Direct communication with the interpreting physician will decrease the chance of omitting important clinical information and will allow them to focus their attention on the area(s) of concern. ■

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1. Johnson M, Policeni B, Lee A, Smoker W. Neuroimaging in Ophthalmology. New York, NY: Oxford University Press; 2011.
2. Walecki J, Mruk B, Nawrocka-Laskus E, et al. Neuroimaging of cerebral venous thrombosis (CVT)-old dilemma and the new diagnostic methods. Pol J Radio. 2015 July;80:368-73.
3. Brouwers H, Battey T, Musial H, et al. Rate of contrast extravasation on computed tomographic angiography predicts hematoma expansion and mortality in primary intracerebral hemorrhage. Stroke. 2015 Sep;46(9):2498-503.
4. Rabinowitz M, Carrasco J. Update on advanced imaging options for thyroid-associated orbitopathy. Saudi J Ophthalmol. 2012 Oct;26(4):385-92.
5. Kirsch E, von Arx G, Hammer B. Imaging in Graves' orbitopathy. Orbit. 2009;28(4):219-25.
6. Verma R, Gupta M, Mehta V. Thyroid associated orbitopathy. BMJ Case Rep. 2013 June. [Epub].
7. Daubner D, Spieth S, Engellandt K, von Kummer R. Diagnosis and differential diagnosis of Graves' orbitopathy in MRI. Radiologe. 2012;52(6):550-9.
8. Hailey D. Open magnetic resonance imaging (MRI) scanners. Issues Emerg Health Technol. 2006 Nov;(92):1-4.
9. van Everdingen KJ. Diffusion-weighted magnetic resonance imaging in acute stroke. Stroke. 1998 Sept;29(9):1783-90.
10. American College of Radiology (ACR) Committee on Drugs and Contrast Media. ACR Manual on contrast Media. 2015;10. www.acr.org/~/media/37D84428BF1D4E1B9A3A2918DA9E27A3.pdf.
11. van der Walt A, Kolbe S, Mitchell P. Parallel changes in structural and functional measures of optic nerve myelination after optic neuritis. PLoS One. 2015 May;10(5):1-11.
12. Kemp PS, Winges, KM, Wall M. Optic neuritis. EyeRounds.org. Posted Sept. 30, 2012. www.EyeRounds.org/cases/159-optic-neuritis.htm.

13. Sasaki T, Nakayama T, Kitamura M, et al. Case of 55-year-old female with primary central nervous system lymphoma, presenting with brain and eye lesions and long-term relapsing/remitting course. Rinsho Shinkeigaku. 2015 Aug 21;55(8):567-72.
14. Patrella JR, Grossman RI, McGowan JC, et al. Multiple sclerosis lesions: relationship between MR enhancement pattern and magnetization transfer effect. AJNR AM J Neuroradiol. 1996 June;17(6):1041-9.
15. Barkhof F, Simon JH, Fazekas F, et al. MRI monitoring of immunomodulation in relapse-onset multiple sclerosis trials. Nat Rev Neurol. 2011;8(1):13-21.
16. Tauhid S, Chu R, Sasane R, et al. Brain MRI lesions and atrophy are associated with employment status in patients with multiple sclerosis. J Neurol. 2015 Jul 24. [Epub ahead of print].
17. Matoob S, Fan JC, Danesh-Meyer HV. Multifocal malignant optic glioma of adulthood presenting as acute anterior optic neuropathy. J Clin Neurosci. 2011 Jul;18(7):974-7.
18. Weed MC, Longmuir RA, Thurtell MJ. Pituitary adenoma causing compression of the optic chiasm: 49-year-old white female with painless progressive vision loss. www.EyeRounds.org. September 9, 2013.
19. Xu K, Yuan Y, Zhou J, Yu J. Pituitary adenoma apoplexy caused by rupture of an anterior communicating artery aneurysm: Case report and literature review. World J Surg Oncol. 2015 Jul 30;13(1):228.
20. Chow LC, Rubin GD. CT angiography of the arterial system. Radiol Clin North Am. 2002 Jul;40(4):729-49.
21. Goddard AJ, Tan G, Becker J. Computed tomography angiography for the detection and characterization of intra-cranial aneurysms: current status. Clin Radiol. 2005 Dec;60(12):1221-36.
22. Sands MJ, Levitin A. Basics of magnetic resonance imaging. Semin Vasc Surg. 2004 Jun;17(2):66-82.

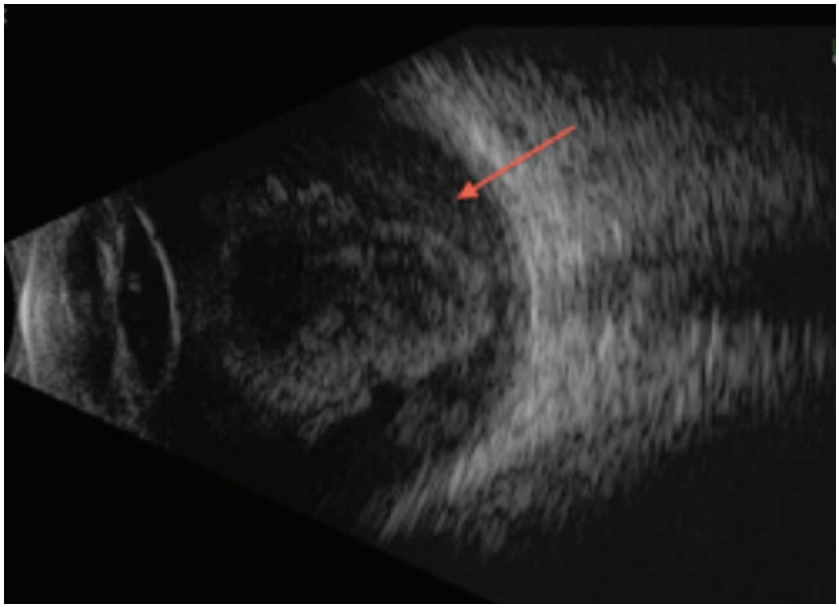

Fig. 6. The retina, which is derived from the neuroectoderm, can be imaged using ultrasound, such as this B-scan of a retinal detachment.

23. Wintermark M, Meuli R, Browaeys P, et al. Comparison of CT perfusion and angiography and MRI in selecting stroke patients for acute treatment. Neurology. 2007 Feb 27;68(9):694-7.

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Ocular Health: A Matter of the Heart

Vascular dysregulation can have a huge impact on the eyes. Here is what you need to know. By Candice Tolud, OD, and Joy Harewood, OD

It has been said that the eye is the window to the soul. Maybe so, but it's the state of the *heart* that's often revealed in the presence of ocular disease. New insights into the pathogenesis of optic neuropathies and retinal vascular disease highlight the importance of having a multidisciplinary approach to comprehensive eye care.

Of particular interest is vascular dysregulation syndrome. Many studies over the years have described a potential link between vascular dysregulation and the eye.[1-5] As our understanding of its ocular effects increases, the treatment options for various conditions are expanding.

This article will review vascular dysregulation, its ocular manifestations and its relationship to common systemic diseases. Finally, it will discuss the use of medications that traditionally support cardiovascular health to better treat ocular disease.

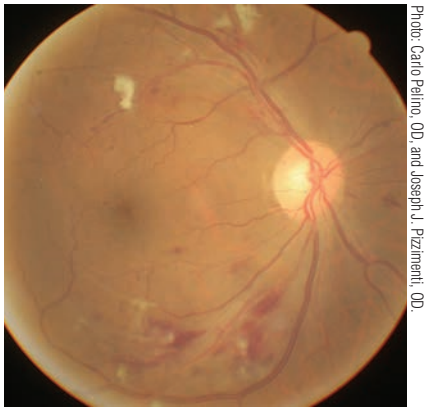

Photo: Carlo Pelino, OD, and Joseph J. Pizzimenti, OD

Significant hypotensive retinopathy shows cotton-wool spots, striated retinal hemorrhages and narrowed retinal arterioles.

Vascular Dysregulation

Inappropriate constriction or dilation of arteries, veins or capillaries in a particular tissue in the body is classified as either primary vascular dysregulation syndrome (PVD) or secondary vascular dysregulation syndrome (SVD).[1] PVD derives from a process endogenous to the vasculature while SVD describes dysregulation due to an underlying non-vascular systemic disease.

Normal fluctuations in blood flow occur with activities of daily living. Blood flow varies due to changes in our natural circadian rhythm, alterations in our mental and physical activity and even simple changes in body position.

Patients with PVD respond abnormally to regular stressors like positional changes, coldness and other stimuli. Risk factors for PVD include female sex, slim build and Asian origin.[6] The pathogenesis of PVD has yet to be determined. Affected patients are usually asymptomatic until they are exposed to one of the aforementioned stressors.

Classic symptoms of PVD include acute sensitivity to cold, reduced feeling of thirst, falling asleep, tinnitus,

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Goal Statement: Clinicians should always look at the eye as an extension of the cardiovascular system. Learning more about how vascular dysregulation affects the eye will alter our treatment approach to various ocular diseases. This article reviews vascular dysregulation, its ocular manifestations, its relationship to common systemic diseases, and the use of cardiovascular medications to better treat ocular disease.

Faculty/Editorial Board: Candice Tolud, OD, and Joy Harewood, OD
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Disclosure Statement: Drs. Tolud and Harewood have no financial relationships to disclose.

sudden hearing loss and a predisposition to migraines. The main signs include arterial hypotension, cold extremities and an increased sensitivity to cold. Other signs include low blood pressure (BP) and silent myocardial infarction. Upregulation of factor endothelin (ET-1) is also found and has specific effects on the eye (*see "Secondary Vascular Dysregulation," pg. 127*).

In a healthy individual, ocular blood flow is autoregulated by the local vasculature. Autoregulation is the eye's intrinsic ability to maintain blood flow despite changes in perfusion pressure. The vascular endothelium, neural and glial cells are responsible for much of this regulation.[2] The vessels in the eye constrict and dilate to maintain a constant flow of blood and nutrients, thus maintaining ocular perfusion pressure (OPP) within a certain range.

Perfusion pressure in the body is the difference between arterial and venous pressures. OPP is calculated as the difference between retinal arterial pressure and intraocular pressure (IOP).[7] Retinal arterial pressure can be measured using ophthalmodynamometry, during which pressure is applied to the side of the globe with a ophthalmodynamometer while observing the central retinal artery with an ophthalmoscope.[8] The point at which pulsations are eliminated is the retinal arterial pressure. This measurement can also be approximated using the following formula: mean retinal arterial pressure = diastolic BP + 1/3(systolic BP – diastolic BP).[9] Retinal venous pressure is thought to be well approximated by the IOP. This approximation is not always accurate, but widely accepted in the field.

PVD affects the eye by reducing ocular blood flow, which causes stiff retinal blood vessels, thus reducing the integrity of the blood/brain barrier and creating dysfunctional ocular autoregulation. These factors exacerbate pre-existing systemic conditions and their effect on the eye, as well as increase the risk of developing numerous primary ocular diseases. PVD is an independent risk factor for glaucoma, and those affected are more predisposed to retinal artery and vein occlusions and central serious chorioretinopathy.[1,4-6,10-13] Patients with PVD and glaucoma may have a tendency to progress despite having normal IOP.[11]

Review of Ocular Vasculature

The eye is exquisitely sensitive to changes in blood flow, in part because the retina has the highest oxygen consumption per volume in the body. It takes a complex system with built-in redundancy to ensure the eye functions normally. The four-part circulation of the eye consists of:

1. Anterior segment circulation, mainly provided by the ciliary body that manufactures aqueous humor.
2. Retinal circulation.
3. Choroidal vasculature.
4. Circulation to the optic nerve head.

Due to the high oxygen demand of the retina, the choroid has the highest blood flow per volume in the body.

Hypotension

Hypotension, defined as blood pressure below 90/60mm Hg, can result from low cardiac output, excessive blood pressure-lowering medications, postoperative complications, poor body positioning, or conditions such as anemia or congestive heart failure. Nocturnal hypotension is of particular interest to eye care providers because of its effect on the posterior segment. It can reduce perfusion to the eye, resulting in changes to both the optic nerve and the retinal vasculature.

Treatment for hypotension is aimed at the cause of low pressure. If it is a result of a surgical procedure, supplemental oxygen is typically given and the infusion rate is increased to combat hypovolemia, or low blood volume. Trendelenburg positioning, where the feet are elevated above the head, can help.[14]

• ***Hypotensive effects on optic nerve head perfusion.*** Ischemic disorders of the optic nerve head (ONH) are multifactorial in nature. Certain risk factors act in combination, with some simply predisposing the optic nerve to ischemia, while others are directly responsible for the final insult that produces ONH ischemia.[4]

While untreated hypertension poses it own risks (*see "Hypertension," pg. 125*), over-treatment of systemic hypertension can be deleterious as well. Although not unique to BP treatment, the *J-curve phenomenon* is seen in aggressive treatment of hypertensive patients when blood pressure is lowered so much that the beneficial effects of therapy are lost and the incidence of adverse events increases.[4,15]

Researchers speculate that the J-curve phenomenon is an important factor in ONH and ocular ischemic disorders.[4,11] When systemic BP falls below the threshold necessary for the eye to maintain autoregulation, OPP drops, resulting in death of retinal ganglion cells and vision loss.[4,11,15] Optic nerves experiencing the J-curve

Fig. 1. Mechanism of ONH Ischemia Due to Nocturnal Hypotension in Predisposed Eyes:[4]

Decrease in BP during the night
↓
Fall of perfusion pressure below the critical autoregulatory level in the ONH vascular bed
(J-curve phenomenon)
↓
Hypoperfusion/nonperfusion of the ONH
↓
ONH ischemia

phenomenon are particularly susceptible during sleep hours when BP is at its lowest.[4,12]

Investigators have hypothesized that nocturnal hypoperfusion of the optic nerve is the precipitating factor in ONH ischemia in susceptible patients.[4,11] As such, there is a significant association seen between progressive visual field loss and nocturnal hypotension in patients on oral hypotensive therapy (*Figure 1*).[4]

• ***Hypotensive effects on glaucoma.*** The pathogenesis of glaucomatous optic neuropathy is classified into the mechanical theory and the vascular theory. The former is well-documented: Increased IOP over time causes interruption of axoplasmic flow and subsequent death of optic nerve fibers.[3,12] The vascular theory focuses on the potential development of intraneural ischemia resulting from decreased optic nerve perfusion.[4]

Much evidence suggests that vascular pathologies play an important role in the etiology and progression of both open angle glaucoma (OAG) and normal tension glaucoma (NTG).[12] Low systemic BP favors the occurrence of visual field defects or development of greater visual field loss at any intraocular pressure, even in non-glaucoma subjects.[4,16] Additionally, vascular insufficiency is considered even more severe in patients with NTG.[16]

NTG is a multifactorial optic neuropathy, and insufficient vascular autoregulation is considered to be present in at least some patients.[16,17] Low BP, excessive BP dips and low ocular perfusion pressure are linked to NTG pathology and disease progression.[11] Population-based epidemiological studies have shown that low diastolic BP and low diastolic OPP are major risk factors for NTG. Additionally, increased variability of mean arterial pressure over 24 hours is a risk factor for NTG development and progression.[16]

Lowering IOP in NTG patients is beneficial in preventing progression, as found in both the Collaborative Normal-Tension Glaucoma (CNTG) Study and the Early Manifest Glaucoma Trial.[18,19] Prostaglandins are typically used as first-line therapy in NTG.[18] The CNTG's recommendation for treatment of NTG was a 30% reduction in IOP.[18,19] Interestingly, a more recent study found that brimonidine 0.2% BID, compared with timolol 0.5% BID, had less progression of visual field loss.[15] Additionally, laser trabeculoplasty has also been shown to be effective in the treatment of NTG.[20]

• ***Non-IOP medical interventions.*** Although IOP plays a major role in both OAG and NTG, IOP-independent factors—such as oxidative stress, glutamate toxicity and vascular factors—still play important roles in the development of primary open-angle glaucoma (POAG) and NTG.[12] As such, studies have explored treatments other than topical ocular hypotensives for their effectiveness in treating glaucomatous optic neuropathy.[11,21] This is of particular interest for NTG patients who show disease progression in spite of normal IOP, decreased IOP or both.

Some research suggests that therapeutic increases in blood pressure in NTG patients may help increase ocular perfusion pressure.[22] Investigators have studied several categories of drugs, including nitric oxide synthase inhibitors, antioxidants, excitotoxins, calcium channel blockers and nerve growth factors (*Table 1*). Research has shown that calcium channel blockers have favorable and significant effects on visual field and optic nerve fiber progression in

Table 1. Topical IOP-mediated and Oral Non-IOP Drugs: Effects on Ocular Perfusion[1,2]

Potential Medications for Increased Blood Perfusion	Proposed Mechanism of Action
Topical Medications	
Calcium channel blockers	Less progression of visual field
Betaxolol	Improved choroidal flow, better visual field preservation
Dorzolamide	Increased retinal blood flow velocity in humans
Brimonidine	Increased retinal ganglion cell survival in rat optic nerve crush injury
Non-IOP Mediated Oral Medications	
N-methyl-D-aspartate (NMDA) receptor antagonist (Memantine)	Prevents binding of glutamate and resultant calcium influx; blocks rat ganglion cells from glutamate toxicity and blocks toxic level of glutamate in vitreous
Serotonin S2 receptor antagonist (Naftidrofuryl)	Arteriolar vasodilation, improved blood flow in Raynaud syndrome
Free radical scavengers - Ginkgo biloba	Scavenges free radicals and nitric oxide, improves blood flow

1. Netland PA, Chaturvedi N, Dreyer EB. Calcium channel blockers in the management of low-tension and open-angle glaucoma. Am J Ophthalmol. 1993 May;115(5):608-13.
2. Freudenthal J et al. Low-tension glaucoma medications. Medscape. 2014 Oct. www.emedicine.medscape.com/article/1205508-medication.

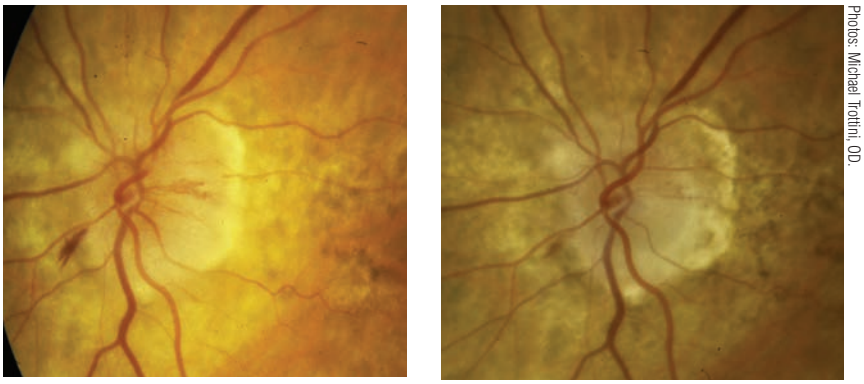
NTG patients in a manner that was not equally seen in patients with POAG.[11]

Recent research has also shown that free radical scavengers such as ginko biloba extract (GBE) and anthocyanins might be effective in NTG.[23] A small sample study showed that GBE increased the end-diastolic velocity in the ophthalmic artery without changes in arterial blood pressure, heart rate or IOP.[24]

• ***NAIONs.*** Non-arteritic ischemic optic neuropathy (NAION) results from ischemia of the retrolaminar part of the optic nerve head.[25] Classically, it causes painless loss of vision or visual field, with more than 75% of patients noticing vision loss upon waking.[21] NAION is the most common acute optic neuropathy in people over 50.[26]

Acutely, optic disc swelling and hemorrhages with pallor of the disc develop over time.[4,21] Research has also noted that some discs have certain anatomic features that seem to predispose them to NAION, referred to as "discs at risk."[4,21,27,28] These features include a small nerve head with a small or absent physiologic cup, abnormal branching of the central vessels and full nerve fiber bundles that obscure the disc margin.[11,21,28] Systemic risk factors of NAION include arterial hypertension and hypotension, atherosclerosis, sleep apnea, migraine and arteriosclerosis.[4,11,28,29]

The exact pathophysiology of NAION is not fully known, although it is generally accepted that the delayed optic disc filling via branches of the short posterior ciliary arteries in the retolaminar region of the optic nerve ultimately causes nerve cell death. In terms of systemic risk factors, investigators believe nocturnal hypotension to be the final insult for the development of non-arteritic ischemic optic neuropathy in patients with a disc at risk.[21]



Photos: Michael Trottini, OD.

NAION in an 80-year-old white female. At left, the optic nerve on presentation. Note disc edema and hemorrhaging. At right, one month later. Note improved edema and some early inferotemporal disc pallor.

There is currently no consistently effective therapy for NAION, and observation is typically recommended with testing, including: complete ocular examination, visual field, blood work such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) if GCA is suspected and referral to internal medicine for the management of any underlying vascular issues.[28]

The most common treatment for NAION is oral steroids.[28] Researchers found that systemic corticosteroids were effective in improving visual function compared with the natural history; however, the untreated group had more vascular risk factors than the treated group.[28] They treated patients with 80mg/day of prednisone with a gradual taper over several weeks, although it is not considered standard of care.[28] Intravitreal Avastin (Genentech) was studied as a proposed method of increasing perfusion to the ONH, but investigators found it had no beneficial effect. Other treatments studied include diphenylhydantoin, erythropoietin and hyperbaric oxygen, although none showed a consistent benefit.[28]

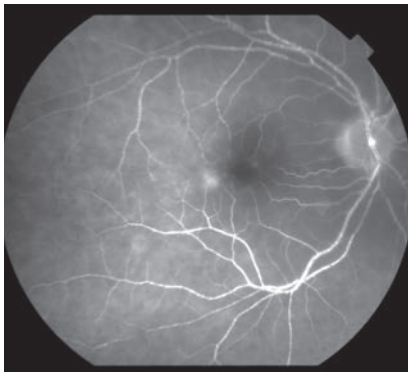
• ***NAIONs and PDE5 inhibitors.*** Phosphodiesterase-5 (PDE5) inhibitors are often used in the treatment of erectile dysfunction (ED). PDE5 is a naturally occurring enzyme that works to inhibit smooth muscle relaxation. PDE5 inhibitors promote smooth muscle relaxation, causing vasodilation and facilitating the erectile process.[21]

Many anecdotal case reports have described NAION in patients who take erectile dysfunction medications, especially PDE5 inhibitors. Researchers hypothesize that these medications have a mild hypotensive effect, which increases physiological nocturnal hypotension and thus results in ONH ischemia in susceptible patients.[29] However, a large pharmaco-epidemiological nested case-control study of 1,109 cases of NAION recently found no association between PDE5 inhibitor medication use and a diagnosis of NAION.[30]

PDE6, which is found in the retina and is responsible for retina phototransduction, can be inadvertently inhibited by ED medications. This, in turn, can lead to cyanopsia—a bluing of vision—which occurs shortly after taking the medication. The effect of this is transient and is not considered harmful.[31]

Hypertension

High blood pressure is a well-known cause of adverse ocular sequelae.[32-34] Generally, a patient is considered hypertensive when their pressure is



Fluorescein angiogram of a CRAO in a 78-year-old Caucasian woman with a history of bacterial endocarditis.

140/90 or higher.[35] Hypertension is generally an asymptomatic condition except in cases of malignant hypertension (BP greater than 180/120). Symptoms of malignant hypertension can include headache, nausea, vomiting, nosebleeds and changes in vision.[35] Optometrists can identify ocular signs of hypertension and can communicate these findings with the patient's internist or cardiologist for treatment modification.

The recommended first-line treatments of hypertension are: a thiazide-type diuretic (i.e., hydrochlorothiazide), a calcium channel blocker (i.e., amlodipine), angiotensin converting enzyme inhibiors (ACEIs) such as lisinopril or an angiotensin receptor blocker such as Cozaar (losartan, Merck).[35] Beta blockers have fallen out of favor as first-line therapy because of the side effects' impact on cardiovascular health, though they remain widely used in patients with heart failure.[35-37]

• ***Hypertensive effects on retinal vasculature.*** When blood pressure is chronically or acutely elevated, the retinal vessels show characteristic changes. Initial response to elevated blood pressure is vasospasm and increase in vasomotor tone, which manifests into retinal-arteriolar narrowing.[38] Chronic changes cause focal areas of narrowing (silver wiring) and compression of retinal veins by arteries (arteriovenous nicking). When elevated blood pressure is severe, there can be blood and lipid leakage, as well as focal areas of ischemia (cotton-wool spots) and papilledema. These signs can be predictive of cardiovascular events such as stroke, congestive heart failure and coronary artery disease.[34,39-41]

When these retinal signs are clinically apparent, it is important to target the microvasculature to reduce risk of morbidity and mortality. This includes tight control of blood sugar in patients with Type 2 diabetes.[42] Some evidence suggests that ACEIs and other antihypertensive medications could directly benefit microvasculature in addition to lowering blood pressure.[43,44] If retinal emboli are present, anti-coagulation therapy, such as aspirin or Coumadin (warfarin, Bristol-Myers-Squibb) should be a consideration. This should be a coordinated effort between the eye care professional and the patient's cardiologist or primary care doctor.

• ***Ocular perfusion pressure and hypertension.*** OPP is very sensitive to changes in blood pressure, especially when ocular autoregulation is impaired.[6,45] Changes in the perfusion pressure to the optic nerve in particular have severe consequences for optic nerve health.[6,45]

Given the formula used to determine ocular perfusion pressure (OPP = arterial BP - IOP), elevated arterial blood pressure should increase ocular perfusion pressure. Although it seems hypertension could have a positive effect on the perfusion of the optic nerve, it is not that simple. Elevated BP leads to short-term constriction of blood vessel walls as the body attempts to control the flow. Long-term, it leads to arteriosclerosis, which will reduce ocular blood flow. Chronic hypertension is also a disease where vascular autoregulation is impaired.[6,45] Hypertension has a negative effect on ocular perfusion, contributing to potential glaucomatous damage.[6,45]

• ***Retinal vein occlusions and PVD.*** One of the most common retinal vascular diseases, retinal vein occlusion (RVO) can cause vision loss, dilated tortuous retinal veins, retinal hemorrhages, cotton-wool spots and macular edema.[46] Reduced ocular blood flow, glaucoma and PVD all increase the risk of RVO. Atherosclerosis and hypertension are the main risk factors for a vein occlusion; however, hypercoagulopathies and vasculitis are also associated with retinal vein occlusion, particularly in younger patients.[32]

Retinal artery occlusion is less common. It is divided into central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO). Symptoms are sudden, painless, unilateral vision loss.[47-49]

A BRAO may cause a visual field defect with minimal effect on vision, while a CRAO causes catastrophic vision loss.[50] Risk factors for branch retinal artery occlusion include: aortic and mitral valve disease, acute myocardial infarction, subacute bacterial endocarditis and prosthetic valves.[48,49] PVD is also a risk factor for the development of artery occlusion and is often the etiology in young, healthy patients.

It should be noted retinal artery occlusions, specifically CRAO, are true ocular emergencies. Treatment options for retinal artery occlusions are limited in their efficacy. Restoring ocular circulation using digital massage of the eyeball, anterior chamber paracentesis to rapidly lower intraocular pressure and breathing into a paper bag to induce carbon-dioxide-related vasodilation are potential therapies.[51,52] Often it is more important to identify the etiology to prevent further complications.

Patients with artery occlusions should undergo echocardiogram and

carotid Doppler testing to try and identify the source of the emboli.[53] Giant cell arteritis and other vasculitic disorders should also be considered as underlying etiologies of artery occlusions, especially if a retinal embolus is not identified.[48,49] The close correlation between PVD and retinal vascular disease and optic neuropathies means that some medications used to promote cardiovascular health may help.[33] Lifestyle changes for patients with PVD include not keeping BMI too low, eating a healthy diet of antioxidant-rich fruits and vegetables and avoiding stressful situations.

Secondary Vascular Dysregulation

SVD occurs as a consequence of other diseases, such as multiple sclerosis, retrobulbar neuritis, rheumatoid arthritis, fibromyalgia and giant cell arteritis.[5,33] In inflammatory disease processes, molecules release into the corresponding tissue and the blood stream, changing the molecular concentration in the circulating blood and subsequently affecting remote organs.[5,33] Of particular ocular interest is the molecule factor endothelin-1 (ET-1).[5,13,33,34]

• ***Endothelin and its ocular effects.*** Endothelin is a potent vasoconstrictor expressed as three isoforms, ET-1, -2 and -3. ET-1 was found to have a strong vasoconstrictive effect on human ophthalmic circulation.[13] This effect is exacerbated in diabetic and hypertensive patients where a dysfunction of these endothelial mechanisms in the peripheral arteries already exists.[13] As such, ET-1 is believed to play an important role in the pathophysiology of ophthalmic complications by weakening the blood/retinal barrier, leading to retinal edema, hemorrhages and exudate.[5,13] Interestingly, patients with retinal vein occlusion have increased plasma levels of ET-1 during the acute phase. Weeks or months later, the concentration decreases, but rarely normalizes completely.[5,54]

In patients with glaucoma, ET-1 is thought to contribute to endothelial cell dysfunction and may represent the underlying cause of, or at least contribute to, alterations in ophthalmic blood flow.[13] ET-1 levels were found to be higher in those with progressive ocular nerve head damage than in those in which the damage had stabilized.[13] Increased plasma ET-1 levels have been described in normal tension glaucoma patients, although this finding was not confirmed in multiple studies.[13] Using animal models, researchers found chronic administration of ET-1 can produce an optic neuropathy similar to glaucoma.[13]

ET-1 is a potential target for future pharmacological intervention that may provide treatment for ocular diseases for which no effective drug therapy is currently available.[13] ET blockers have been proposed and preliminarily studied to reduce vasospasm; however, routine use is not yet recommended due to potential side effects.[5]

Our understanding of ocular pathology continues to grow, and eye care providers should always look at the eye as an extension of the cardiovascular system. Learning more about how vascular dysregulation affects the eye will alter our treatment approach to various ocular diseases. Incorporating a multidisciplinary approach will allow for better treatment of not only the eye, but for the body as whole. ■

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1. Flammer J, Haefliger IO, Orgul S, Resink T. Vascular dysregulation: a principle risk factor for glaucomatous damage? J Glaucoma. 1999;8:2121-19.
2. Kur J, Newman EA, Chan-Ling T. Cellular and physiological mechanisms under-lying blood flow and regulation in the retina and choroid in health and disease. Progress Retin Eye Res. 2012;31:377-406.
3. Flammer J, Orgül S, Costa VP, et al. The impact of ocular blood flow in glaucoma. Prog Retin Eye Res. 2002 Jul;21(4):359-93.
4. Hayreh SS, Podhajsky P, Zimmerman MB. Role of nocturnal arterial hypotension in optic nerve head ischemic disorders. Ophthalmologica. 1999;213:76-96.
5. Flammer J, Konieczka K, Flammer AJ. The primary vascular dysregulation syndrome: implications for eye diseases. EPMA J. 2013; 4(1):14.
6. Flammer J, Konieczka K, Flammer AJ. The primary vascular dysregulation syndrome: implications for eye diseases. EPMA Journal. 2013;4:14.
7. Leske MC. Ocular perfusion pressure and glaucoma: clinical trial and epidemiologic findings. Curr Opin Ophthalmol. 2009 Mar;20(2):73-8.
8. Perry RB, Rose JC. The clinical measurement of retinal arterial pressure. Circulation. 1953;18:864-70.
9. Costa VP, Harris A, Anderson D, et al. Ocular perfusion pressure in glaucoma. Acta Ophthalmologica. 2014:92e252-e255.
10. Kaiser JH, Flammer J, Messerli J. Vasospasm – a risk factor for nonarteritic anterior ischemic optic neuropathy? Neuro-ophthalmology. 1996;16(1):5-10.
11. Hayreh SS. The Role of age and cardiovascular disease in glaucomatous optic neuropathy. Survey of Ophthalmology. 1999;43(1):27-42.
12. Yamamoto T, Kitazawa Y. Vascular pathogenesis of normal-tension glaucoma: possible pathogenic factor, other than intraocular pressure, of glaucomatous optic neuropathy. Progress in Retinal and Eye Research. 1998;17(1);127-43.
13. Salvatore S, Vingolo EN. Endothelin-1 role in human eye: a review. J Ophthalmol. 2010;2010:354645
14. Ostrow CL. Use of the Trendelenurg position by critical care nurses: Trendelenburg survey. Am J Crit Care. 1997 May;6(3):172-6.
15. Krupin T, Liebmann JM, Greenfield DS, 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 Apr;151(4):671-81.
16. Mi XS, Yuan TF, So KF. The current research status of normal tension glaucoma. Clin Interv Aging. 2014 Sept;16;9:1563-71.
17. Cioffi, G. Three common assumptions about ocular blood flow in glaucoma. Surv of Ophthalmology. 2001;45(3)S325-S331.
18. Anderson D. Normal-tension glaucoma (Low-tension glaucoma). Indian J Ophthalmol. 2011 Jan; 59(Suppl1):S97-S101.
19. Collaborative Normal-Tension Glaucoma Study Group: The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. Am J Ophthalmol. 1998;126:495-505.
20. Tanna A. Managing NTG: Strategies for success. Review of Ophthal. 2009 Feb;16(2):70.
21. Schumer RA, Podos SM. The nerve of glaucoma! Arch. Ophthalmol. 1994;112:37-44.
22. Pechere-Bertschi A, Sunaric-Megevand G, Haefliger I, et al. Renal sodium handling in patients with normal pressure glaucoma. Clin Sci (Lond). 2007 Jun;112(6):337-44.
23. Shim SH, Kim JM, Choi CY, et al. Ginkgo biloba extract and bilberry anthocyanins improve visual function in patients with normal tension glaucoma. J Med Food. 2012 Sep;15(9):818-23.
24. Chung HS, Harris A, Kristinsson JK, et al. Ginkgo biloba extract increases ocular blood flow velocity. J Ocul Pharmacol Ther. 1999;15:233–40.
25. James CB, Smith SE. The effect of posture on the intraocular pressure and pulsatile ocular blood flow in patients with non-arteritic anterior ischaemic optic neuropathy. Eye. 1991;5:309-14.
26. Kim IG, Kim DY. Anterior ischemic optic neuropathy associated with udenafil. Korean J Ophthalmol. 2012;26(3):235-38.
27. Burde RM. Optic disk risk factors for non-arteritic anterior ischemic optic neuropathy. Am J Ophthalmol. 1993;116:759-64.
28. Miller NR, Arnold AC. Current concepts in the diagnosis, pathogenesis and management of nonarteritic anterior ischaemic optic

neuropathy. Eye. 2015;29:65-79.
29. Hayreh SS. Erectile dysfunction drugs and non-arteritic anterior ischemic optic neuropathy: Is there a cause and effect relationship? Journal of Neuro-Ophthalmology. 2005 Dec;25(4):295-98.
30. Nathoo NA, Etminan M, Mikelberg FS. Association between phosphodiesterase-5 inhibitors and nonarteritic anterior ischemic optic neuropathy. J Neuroophthalmol. 2015 Mar;35(1):12-5.
31. Smith WB. PDE5 inhibitors: considerations for preference and long-term adherence. Int J of Clin Pract. 2013;67(8):768-80.
32. Wong T, Mitchell P. The eye in hypertension. Lancet. 2007;369:425-35.
33. Flammer J, Konieczka K, Bruno RM, et al. The eye and the heart. European Heart Journal. 2013;34:1270-8.
34. Duncan BB, Wong TY, Tyroler HA, et al. Hypertensive retinopahy and incident coronary heart disease in high risk men. BR J Ophthalmol. 2002;86:1002-6.
35. James PA, Oparil S, Carter B, et al. 2014 evidence-based guideline for the management of high blood pressure in adults. Report from the panel members appointed to the eighth joint national committee. JAMA. 2014;311(5):507-20.
36. Dahlof B, Devereux RB, Kjeldsen SE, et al. LIFE Study Group. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint Reduction in Hypertension Study (LIFE); a randomized trial against atenolol. Lancet. 2002;359(9311):995-1003.
37. Antihypertensive and Lipid-lowering Treatment to Present Heart Attack Trial Collaborative Research Group. Diuretic versus alpha-blocker as a first-step antihypertensive therapy: final results from the Antihypertensive and Lipid-lowering Treatment to Prevent Heart Attack Trial (ALLHAT). Hypertension. 2003;42(3):239-46.
38. Gugleta K, Orgül S, Stümpfig D, et al. Fludrocortisone in the treatment of systemic hypotension in primary open-angle glaucoma patients. Int Ophthalmol. 1999;23(1):25-30.
39. Hankey GJ, Slattery JM, Warlow CP. Prognosis and prognostic factors of retinal infarction: a prospective cohort study. BMJ. 1991;302:499-504.
40. Michelson EL, Morganroth J, Nichols CW, MacVaugh H 3rd. Retinal arteriolar changes as an indicator of coronary artery disease. Arch Intern Med. 1979;(139):1139-41.
41. Wong TY, Rosamond W, Chang PP, et al. Retinopathy and risk of congestive heart failure. JAMA. 2005;293:63-9.
42. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. BMJ. 1998;317:703-13.
43. Levey BI, Ambrosio G, Pries AR, Struijker-Boudier HA. Microcirculation in hypertension: a new target for treatment? Circulation. 2001;104:735-40.
44. Arnold JM, Yusuf S, Young, et al. HOPE Investigators. Prevention of heart failure in patients in the Heart Outcomes Prevention Evaluation (HOPE) Study. Circulation. 2003;107:1284-90.
45. Lee J, Choi J, Jeong D, et al. Relationship between daytime variation in blood pressure or ocular perfusion pressure in glaucomatous visual field progression. Am J Ophthalmol. 2015:1-15.
46. Wong TY, Larsen EK, Klein R, et al. Cardiovascular risk factors for retinal vein occlusion and arteriolar emboli. Ophthalmol. 2005;112:540-7.
47. Hayreh SS. Prevalent misconceptions about acute retinal vascular occlusive disorders. Prog Retin Eye Res. 2005;24:493-519.
48. Recchia FM, Brown GC. Systemic disorders association with retinal vascular occlusion. Curr Opin Ophthalmol. 2000;11:462-7.
49. Wilson LA, Warlow CP, Russel RW. Cardiovascular disease in patients with retinal arterial occlusion. Lancet. 1979;1:292-4.
50. Recchia FM, Brown GC. Systemic disorders associated with retinal vascular occlusion. Current Opinion in Ophthalmology: 2000 Dec;11(6):462-7.
51. Atebara NH, Brown GC, Cater J. Efficacy of anterior chamber paracentesis and Carbogen in treating acute nonarteritic central retinal artery occlusion. Ophthalmol. 1995;102:2029-34.
52. Rumelt S, Dorenboim Y, Rehany U. Aggressive systemic treatment for central retinal artery occlusion. Am J Ophthalmol. 1999;128:733-8.
53. Wong TY, Klein R. Retinal arteriolar amboli: epidemiology and risk of stroke. Curr Opin Ophthalmol. 2002;13:142-6.
54. Haufschild T, Prünte C, Messerli J, Flammer J. Increased endothelin-1 plasma level in young adults with retinal vascular occlusive diseases. Klin Monbl Augenheilkd. 2004 May;221(5):357-9.

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1. Blood flow varies due to:
a. Changes in our natural circadian rhythm.
b. Alterations in mental and physical activity.
c. Changes in body position.
d. All of the above.

2. Which of these is not a PVD risk factor?
a. Female gender. b. Slim build.
c. High stress. d. African American race.

3. All of these are causes of SVD, except:
a. Giant cell arteritis. b. Multiple sclerosis.
c. Hypotension. d. Rheumatoid arthritis.

4. Which is not true regarding hypotension?
a. Blood pressure typically falls at night.
b. Drug induced nocturnal hypotension is associated with decreased glaucomatous optic nerve damage.
c. The J-curve phenomenon states that over-treatment of systemic hypertension can lead to morbidities.
d. Nocturnal hypotension is a risk factor for NAION.

5. Which of the following has shown favorable effects on visual field and optic nerve fiber progression in NTG patients?
a. Calcium channel blockers.
b. Beta blockers.
c. Ace inhibitors.
d. Thiazide diuretics.

6. Which is not true of NAION?
a. Typically occurs in individuals >50.
b. A pre-disposing risk factor is a small optic nerve cup.
c. Clinical findings include disc edema and retinal hemorrhages.
d. It will present as a painful loss of vision.

7. All of the following are risk factors for a vein occlusion, except:
a. Vacuities.
b. Hypertension.
c. PDE-5 inhibitors.
d. Hypercoagulopathies.

8. PDE-5 inhibitors can cause:
a. Cyanopsia.
b. Retinal artery occlusion.
c. Retinal vein occlusion.
d. Ocular migraines.

9. All of the following are characteristic retinal findings associated with hypertension, except:
a. A/V nicking.
b. Macular edema.
c. Cotton wool spots.
d. Retinal hemorrhages.

10. Which of the following is not true about artery occlusions?
a. Can be caused by giant cell arteritis.
b. Is an ocular emergency.
c. Patients should undergo echocardiogram and carotid doppler testing.
d. Typically causes bilateral vision loss.

11. All of the following are typically included in the work up of patients with NAION, except:
a. Visual field testing.
b. VEP testing.
c. ESR and CRP.
d. Assessment of vascular disorders.

12. Endothelin:
a. Causes vasodilation.
b. Causes vasoconstriction.
c. Has four isoforms.
d. Decreases blood pressure.

OSC QUIZ

13. According to research, patients with a retinal vein occlusion have:
a. Elevated plasma levels of ET-1 acutely, which rarely normalize.
b. Elevated plasma levels of ET-1 acutely, which typically normalize within a few weeks to months.
c. Decreased plasma levels of ET-1 acutely, which typically normalize within a few weeks to months.
d. Decreased plasma levels of ET-1 both acutely and weeks to months later.

14. In animal models, chronic administration of ET-1 has been shown to:
a. Induce macular edema.
b. Induce retinal detachment.
c. Induce optic neuropathy.
d. Induce retinal neovascularization.

15. Which has not been attempted as a possible treatment for NAION?
a. Hyperbaric oxygen therapy.
b. Intravitreal bevacizumab.
c. Erythropoietin.
d. Anecortave acetate.

16. The CNTG study recommends a _____ reduction in IOP for treatment of NTG.
a. 10%. b. 20%.
c. 30%. d. 40%.

17. Hypotension is defined as having blood pressure of less than:
a. 70/50mm Hg. b. 80/50mm Hg.
c. 90/50mm Hg. d. 90/60mm Hg.

18. OPP is calculated as:
a. Venous BP – IOP. b. Arterial BP + IOP.
c. Venous BP + IOP. d. Arterial BP – IOP.

19. Which is not a symptom of PVD?
a. Sudden hearing loss.
b. Vertigo.
c. Tinnitus.
d. Inability to fall asleep.

20. What is not typically used as first-line therapy in non-diabetic patients in the treatment of hypertension?
a. Beta-blockers.
b. Thaizide-type diuretics.
c. ACE inhibitors.
d. All of the above.

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16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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Out-of-Character Ocular Disease

A patient with ocular hypertension presents unusual findings on OCT.

By James L. Fanelli, OD

A 49-year-old white male presented to the office as a new patient to establish care after moving to the area. He presented with essentially no complaints visually, though he did need an updated pair of glasses.

His medical history was significant for an apparent episode of questionable optic neuritis in the left eye approximately 10 years earlier. At that time, he had complaints of acute visual disturbances in the left eye and, reportedly, several specialists had suspected a differential diagnosis of optic neuritis. He also mentioned that the earlier providers were concerned about the possibility of multiple sclerosis (MS). However, subsequent MR scanning and neurology consult did not firmly establish a diagnosis of MS, and the patient had no other associated symptoms. His vision returned to normal after a few months, and he has since had no visual disturbances. He had maintained good liaison with his eye doctors and neurologists for the subsequent five years, after which time he was seen on a PRN basis.

Diagnostic Data

On initial presentation, the only medication he was taking was naprosyn OTC on a PRN basis, and he reported no allergies to medications. Entering visual acuities through myopic astigmatic correction were 20/30+ OD and 20/25-2 OS. Pupils were ERRLA with no APD. Close attention was paid to the pupillary responses given his history, and no APD was elicited. Best corrected visual acuity was 20/20 OU through increased myopic and astigmatic correction, along with a low add for near reading comfort.

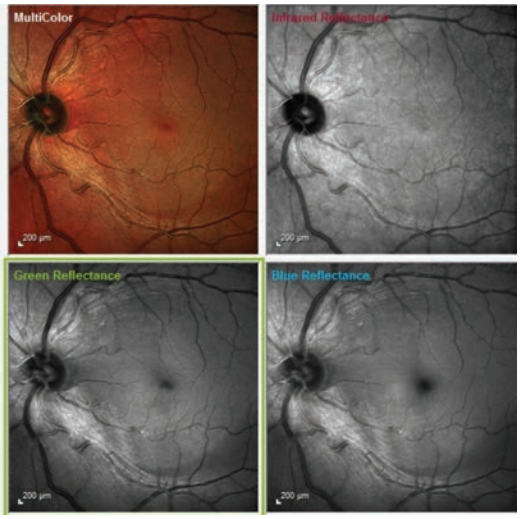

Fig. 1 These images demonstrate a notable wedge defect along the inferotemporal arcuate fibers, well away from the optic nerve. Also note the loss of nerve fiber robustness in the papillomacular bundle area.

A slit lamp examination of his anterior segments was essentially unremarkable. Angles were open in both eyes. Applanation tensions were 25mm Hg OD and 26mm Hg OS. Pachymetry readings were 533µm OD and 545µm OS. The patient was dilated in the usual fashion. His crystalline lenses were clear in both eyes. There were some anterior vitreous floaters in each eye and no evidence of PVD in either eye.

His cup-to-disc ratio was 0.5 x 0.55 in both eyes. The sizes of the optic nerves were normal. The temporal rim in the left eye was slightly pale in comparison to the remaining ipsilateral neuroretinal rim, which was plush and well perfused, as was the neuroretinal rim of the right. The pallor was subtle and not distinct enough to label it sectoral atrophy, but considering his history, the clinical findings appeared to match some previous event affecting the left eye. The retinal vascular, macular and peripheral retinal evaluations were entirely normal.

Further Testing

Given the ocular hypertension, we ordered HRT3 (Heidelberg) retinal tomograph imaging and OCT imaging, as well as stereo photography. The HRT3 scan was consistent with the clinical findings on fundoscopy regarding the cup-to-disc ratio and optic disc size. The RNFL circle scan on OCT demonstrated an area of slight thinning inferotemporally in the left eye.

We scheduled the patient for threshold fields and reassessment of IOP after three months, along

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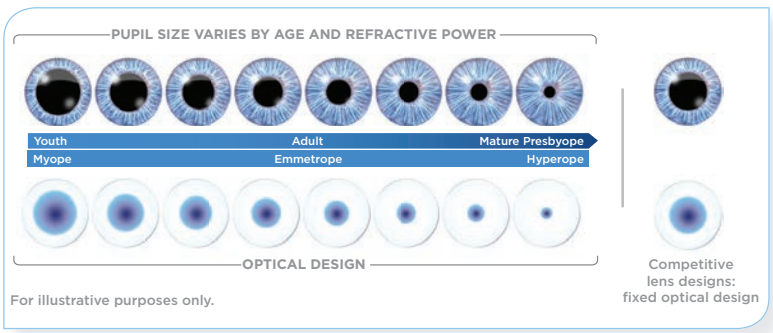

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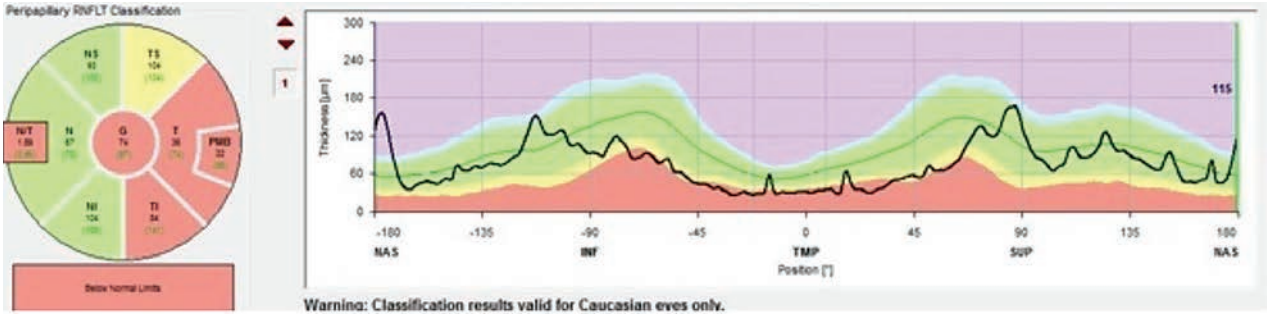

Fig. 2. Note this is not the traditional TSNIT layout for RNFL scans. Rather, this is an RNFL circle scan beginning and ending nasally, leaving a continuous scan through the papillomacular bundle region. The entire temporal RNFL scan is depressed, consistent with the clinical picture.

with gonioscopy. When the patient presented for this visit, one month ago, his IOP was 26mm Hg OU at 11:50am. Threshold visual fields demonstrated paracentral aberrations in both eyes, though the reliability indices were low. Testing showed no obvious evidence of early glaucomatous field defects, nor evidence of central field loss associated with the subtle pallor noted in the left eye. Gonioscopy demonstrated 360 open angles to the ciliary body, with normal trabecular pigmentation and a flat iris approach to the angle.

At this visit I took the opportunity to obtain multi-modal posterior segment imaging using the Spectralis multi-color imaging technology (Heidelberg), as well as run the neurological OCT protocol available on the same technology platform (*Figures 1 and 2*).

Discussion

This case is representative of clinical situations when the results of our evaluation and testing are not cut and dry. While the patient does indeed have a vague history of some type of visual disturbance in his left eye many years earlier, no definitive diagnosis was made. I am in the process of obtaining old records to see if they can shed light on what happened in the past, but the reality is that we must deal with the situation at this time.

What exactly is that situation? What's known is that we have a case of ocular hypertension with no definitive optic nerve defects, other than an RNFL scan that shows inferotemporal thinning on a statistical basis. We must be careful in interpreting diagnostic scans, as we tend to heavily rely on normative databases to make diagnoses. We need to remember that normative databases are simply a representation of the statistical findings of the population studied and that the database essentially gives us the typical 'bell curve' of data. While the majority of patients will fall into the center of the bell curve, outliers, like this patient, may or may not be normal; the databases simply help identify outliers, not necessarily disease.

This case still raises several questions: What happened to the left eye several years ago? Is the papillomacular bundle thinning visible on multicolor imaging and neuro RNFL scans related to that unknown incident? Is the inferotemporal RNFL thinning related to broad papillomacular bundle defects extending somewhat inferiorly, or is it related to early glaucomatous damage, due to the ocular hypertension? Lastly, is the wedge defect in the inferior arcuate fibers related to glaucoma, neuronal deterioration from another cause, or normal for this particular patient?

Obviously we have more questions than answers. When faced with conflicting clinical data, sometimes the most prudent course of action is to simply monitor the situation and see what changes over time. Certainly, there will be cases and situations when there is immediate intervention is necessary, but in cases such as this, when no acute risk of visual compromise, the best plan of action is to wait and see.

Today's technology is outstanding, but it still requires a clinician's interpretation of the information it provides. Using it, we're able to tailor management plans for particular patients, rather than make global management decisions.

In moving forward with this patient, I chose simply to monitor the patient on a four to six month basis with specific emphasis looking for change in the neuroretinal rim, the RNFL scans and the wedge defect, all from a structural perspective, and changes in the visual fields from a functional perspective.

Slow and steady is the course needed here. ■

20TH ANNUAL OPHTHALMIC *Product Guide*

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REVIEW® OF OPTOMETRY

A Wrinkle, In Time

This patient's blurred vision and history of leaking fluid may reveal his diagnosis.

By Mark T. Dunbar, OD

A 55-year old Hispanic male presented for an evaluation of blurred vision in his right eye. He explained that approximately one year ago, he noted distortion in the right eye and color vision changes. An ophthalmologist noted fluid leaking in the back of his eye. The distortion resolved and his vision improved in a few months.

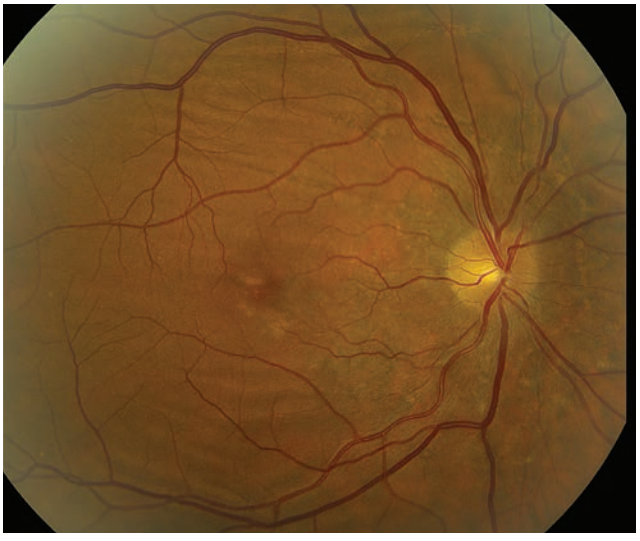
Then, three months ago he reported another episode of blurred vision and distortion, mostly while driving. He felt the vision had improved, but it still was not "right." He has never had any eye problems and his medical history was unremarkable.

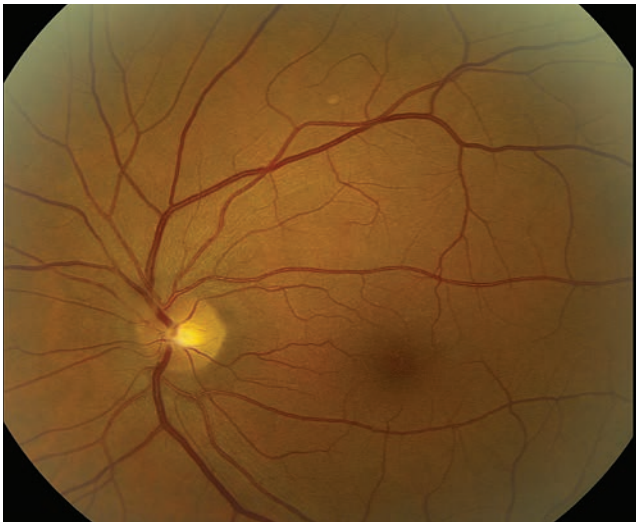
Upon examination, his entering visual acuity measured 20/30 OD and 20/20 OS. With a manifest refraction of +2.50D he was 20/20 OU. The left eye had a minimal hyperopic correction. Confrontation fields were full to careful finger counting in both eyes. Extraocular motility testing was unremarkable. His pupils were equally round and reactive; there was no afferent pupillary defect.

Anterior segment examination was unremarkable. Dilated fundus examination showed small cups with good rim coloration and perfusion in both of his eyes.

The posterior pole of the right eye showed changes (*Figure 1*). Fundus images of the left eye are also available (*Figure 2*).

An SD-OCT is also available for review (*Figures 3a and 3b*).





Figs. 1 & 2. Fundus images of the right (above) and left (below) eyes of our patient. Note the unusual changes in the right eye.

Take the Quiz

1. What are the changes seen in the posterior pole of the right eye?
a. Subretinal fluid.
b. Choroidal folds.
c. Retinal stria.
d. SubRPE tracking from a parasite.

2. What is the etiology?
a. Inflammation.
b. Hypotony.
c. Orbital tumor.
d. Idiopathic.

3. What is the likely explanation for

the fluid in the back of his eye?
a. Impossible to tell.
b. Cystoid macular edema.
c. Choroidal neovascular membrane.
d. Idiopathic central serous chorioretinopathy (ICSC).

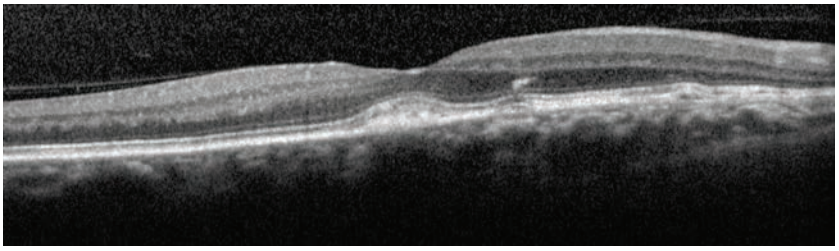
4. What additional test would be helpful to confirm the diagnosis?
a. Fluorescein angiography.
b. MRI.
c. Orbital ultrasound.
d. Visual field.

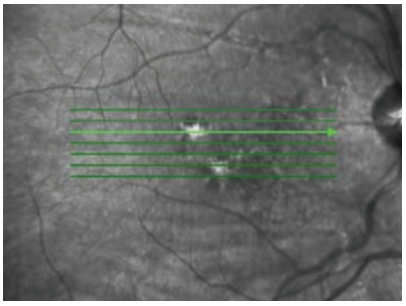
5. How should this patient be managed?
a. Observation.
b. Intravitreal anti-VEGF medication.
c. Refer to neuro-ophthalmologist.
d. Start anti-helminthic medications.

For answers, see page 154.

Discussion

Our patient has choroidal folds in the right eye. On clinical exam, we can see, throughout the entirety of the posterior pole, horizontal linear lines that alternate in color between light and dark. These are folds, or wrinkles, in the inner portion of the choroid, retinal pigment epithelium (RPE) and neurosensory retina. They develop as a result of shrinkage of the sclera or from scleral thickening.[1] This may occur in a number of conditions including orbital inflammatory disease (posterior scleritis or inflammatory pseudotumor), hypotony following intraocular surgery, choroidal neovascularization and orbital tumors, among others. Any of these conditions can result in shrinkage of the sclera which, in turn, causes a reduction in the area of the inner surface of the sclera.[1] When this occurs, the choroid and RPE become redundant relative to the area of the sclera and choroidal folds develop.





Figs. 3a and 3b. Do these SD-OCT images point to the patient's pathology?

In the majority of cases, choroidal folds will result from idiopathic causes. They are usually an incidental finding in patients during a routine eye examination.[1] They may involve most of the posterior pole of the eye, or can be confined to an area either above or below the macula and optic disc. Their distribution in both eyes is usually symmetric. In most instances visual acuity is unaffected.[1]

Choroidal folds can also be seen in moderate or high hyperopia due to these patients' shorter axial length and thick scleras.[1] This can occur at any age, but is most commonly seen in middle-aged adults. They can be unilateral, but are more often bilateral.[1]

It has been ingrained in most clinicians that unilateral choroidal folds may be the result of an orbital tumor pressing on the back of the globe. However, it is important to note that orbital tumors will not cause choroidal folds, unless scleral thickening or shrinkage occurs.[1]

The diagnosis of choroidal folds is generally based on the clinical appearance; however, fluorescein angiography may highlight the folds better than what is seen on clinical exam. In our patient, the folds were quite obvious, so we did not feel we needed a fluorescein angiography to confirm the diagnosis. If there was concern of an orbital mass or inflammatory process, an orbital ultrasound would have been helpful. We did not feel there was any resistance to retropulsion so we did not perform an ultrasound. We did perform an OCT, as there was RPE modeling in the macula. The OCT show some irregularity at the level of the RPE but there was no CNV (*Figures 3a and 3b*).

So why does our patient have choroidal folds? Based on the history our patient provided, we thought he might have had an episode of central serous chorioretinopathy.

The previous doctor told the patient there was fluid in the back of his eye. This may have been a neurosensory detachment. As most cases of ICSC will resolve on its own without treatment, that may very well be what happened.[1] The RPE mottling in the macula is consistent with resolved ICSC, and old ICSC has been known to result in choroidal folds.[1]

We discussed these findings with our patient and explained to him that, should he notice any other changes in his vision, he should return to the office immediately for further evaluation. ■

1. Gass JD. Choioretinal folds. In: Gass JD, editor. Stereoscopic atlas of macular disease: Diagnosis and treatment. 4th ed. St. Louis: Mosby; 1997. pp. 288–301.


Mighty Mite is on the Way

In the *Demodex* age, don't overlook this other creepy crawler.

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

A 60-year-old man presented complaining of a bilateral, irritated, itchy eyelid and lash area for approximately two months. He had used hot compresses and scrubbed his eyes with baby shampoo, to no avail. He noticed that it began immediately after returning from a trip to Las Vegas and he was wondering if he caught something on the plane.

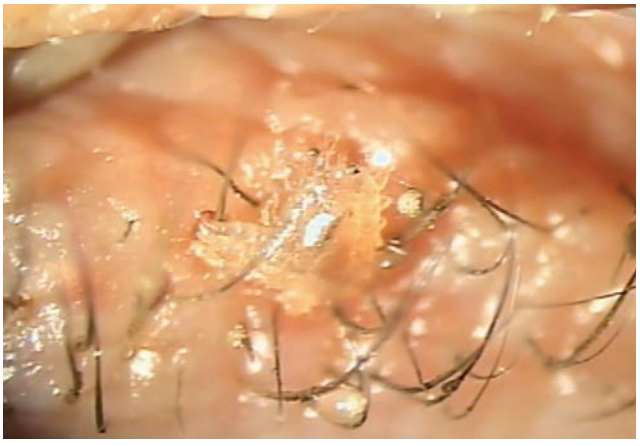
Examination

Gross inspection revealed pronounced blepharoconjunctivitis and pruritic lid margin. However, upon closer biomicroscopic examination, what was thought to be lash scaling was actually ruptured and unruptured egg sacks.

Additionally, in the lower lash region, we observed several oval, crab-like organisms clutching onto the cilia with claws. Upon rolling and partially everting the upper eyelids, more live organisms were found. The diagnosis was quite clear: the patient had a crab louse infection.

Lice Infestations

The three major types of lice that infest humans are *Pediculus humanus capitis* (head louse), *Phthirus pubis* (crab louse) and *Pediculus humanus* (body louse).[1] *Phthirus pubis* are the most common mite in eyelid and eyelash infestation. When this occurs, it is termed *Phthiriasis palpebrarum*. Thus, the more commonly used term, *pediculosis*, is actually incorrect because this represents infestation with another organism.[2-5] *Pediculus* mites that typically infest the head hair of the patient measure 2mm to 4mm long.



The louse's broad, oval, crab-like body features "claws" that firmly grip the patient's eyelashes.

Eyelash Infestation

Infestation of the eyelashes is rare and only occurs in the most severe cases. A *Phthirus* is 2mm long with a broad-shaped, crab-like body. Its thick, clawed legs make it less mobile than the *Pediculus* species and lend it to infesting areas where the adjacent hairs are within its grasp (eyelashes, beard, chest, axillary region, pubic region). Also, this morphology makes the organism readily identifiable.

Pediculus and *Phthirus* mites lay eggs on the hair shafts, remaining firmly adherent, resisting both superficial mechanical and chemical removal. Hence, hot compresses and lid scrubs are usually ineffective. *Pediculus* mites possess good mobility and can pass from person to person either by close contact with an infested individual or by contact with contaminated bedding. Conversely, *Phthirus* mites are slow-moving organisms that cannot typically pass unless cilia are brought into close proximity with infested cilia, typically through sexual contact. Both species are associated with crowding or poor personal hygiene.[1]

Ocular signs and symptoms include visible organisms within the eyelashes, visible blue skin lesions (louse bites), reddish brown deposits (louse feces), secondary blepharitis with preauricular adenopathy, follicular conjunctivitis and, in severe cases, marginal keratitis. The patient often presents with ocular itching and irritation. Superinfection of bites can lead to preauricular gland swelling.[3-5] The condition may be unilateral or bilateral.

Treatment

Doctors can treat infestations

chemically or mechanically. Topical ophthalmic ointments, such as an antibiotic twice daily for two weeks, can smother the organisms and protect against infection from bites. A steroid-antibiotic ointment can smother the organisms while also providing symptomatic relief, though the extremely minor risk of intraocular pressure elevation may not justify the steroid. Local application of a pediculocide such as yellow mercuric oxide 1% ophthalmic ointment or 0.25% physostigmine (eserine) ointment applied twice daily for a minimum of two weeks will also work.[5] Eserine will poison the respiratory system of the organism. Another method for treating *Phthiriasis palpebrarum* involves a single application of 20% fluorescein. It is nontoxic and non irritating.[6] Ongoing treatment is required because the organisms often survive a single application and reinfestation will occur when eggs hatch.

Many advocate mechanical removal of the organisms and eggs.[2-8] Doctors can perform this at the biomicroscope with jeweler's forceps. Be aware that the organism will hold on tenaciously and many lashes may be inadvertently epilated during the procedure. This can be quite uncomfortable for the patient. Application of an ophthalmic ointment can make removal easier in that the organism cannot grasp as tightly to the lashes; however, the ointment may make grasping the organisms themselves more difficult. Once removed, dipping the lice in alcohol will kill them.

Our preference involves mechanically removing all organisms and as many of the eggs as possible, followed by topical antibiotic ointment treatment BID for two weeks.

Beyond the Eyes

When doctors diagnose *Phthiriasis palpebrarum*, they must also suspect genital involvement. Instruct patients to obtain and use a medicated pediculocidal shampoo. These include, but are not limited to:

- Kwell (lindane 1%, Aspen Pharma)
- Permethrin 1% (Nix cream rinse, Warner-Lambert; Elimite cream, Allergan; Acticin cream, Bertek)
- Pyrethrins/piperonyl butoxide (A-200 Pyrinate, Hogil; Rid, Bayer)

These are safe, effective, nonprescription pediculocides. Due to toxicity, these agents cannot be used on the eyelids. There has been increasing resistance to these pediculocides. An alternate therapy is the oral use of Stromectol (ivermectin 250mg/kg, Merck) for two doses given a week apart to kill the lice and subsequent hatchlings.[9,10]

Family members, sexual contacts and close companions should be examined and treated appropriately; clothing, linen and personal items should be disinfected with heat of 50 degrees C for 30 minutes.[7]

Also consider concurrent infection and other sexually transmitted diseases such as HIV, syphilis and chlamydia. Pubic lice infestation is predictive of a concurrent chlamydia infection in adolescents. Adolescents infested with pubic lice should be screened for other STDs, including chlamydia and gonorrhea.[11]

In children, consider the possibility of sexual abuse and proceed with reporting according to your state law.[12] Alternately, contact the child's pediatrician to share your concern.

For this patient, all of the organisms and egg sacs were painstakingly removed with a jeweler's forceps and smothered in alcohol. The patient was also prescribed bacitracin ointment to smear onto both eyelids for 10 days.

Clinicians often refer to the unruptured egg sacs as "nits," while general population uses the term to connote any lice infestation. Physical removal of crab louse and egg sacs (typically with a biomicroscope and forceps) is a tedious and labor-intensive procedure that requires concentration and an exacting eye for detail. Interestingly, this is where the terms "nit-picky" and "nit-picking" come from: to describe a person with an exacting degree of detail and concentration.

In this case, since the patient's symptoms occurred in conjunction with his trip to Las Vegas, we attempted to elicit a more detailed social history; however, the patient was elusive with his response.

We discussed the likely sexual transmission of this organism and the patient left the exam with the necessary information to make informed social decisions. He never returned for a follow-up. ■

Drs. Kabat and Sowka have no financial interest in any products mentioned in this article.

1. Ko CJ, Elston DM. Pediculosis. J Am Acad Dermatol. 2004;50(1):1-12.
2. Yoon KC, Park HY, Seo MS, et al. Mechanical treatment of Phthiriasis palpebrarum. Korean J Ophthalmol. 2003;17(1):71-3.
3. Ikeda N, Nomoto H, Hayasaka S, et al. Phthirus pubis infestation of the eyelashes and scalp hairs in a girl. Pediatr Dermatol. 2003;20(4):356-7.
4. Lin YC, Kao SC, Kau HC, et al. Phthiriasis palpebrarum: an unusual blepharoconjunctivitis. Zhonghua Yi Xue Za Zhi (Taipei). 2002;65(10):498-500.
5. Yi JW, Li L, Luo da W. Phthiriasis palpebrarum misdiagnosed as allergic blepharoconjunctivitis in a 6-year-old girl. Niger J Clin Pract. 2014;17(4):537-9.
6. Mathew M, D'Souza P, Mehta DK. A new treatment of pthiriasis palpebrarum. Ann Ophthalmol. 1982;14(5):439-41.
7. Keklikci U, Cakmak A, Akpolat N, et al. Phthiriasis palpebrarum in an infant. J Pediatr Ophthalmol Strabismus. 2009;46(3):173-4.
8. Couch JM, Green WR, Hirst LW, et al. Diagnosing and treating Phthirus pubis palpebrarum. Surv Ophthalmol. 1982;26(4):219-25.
9. Do-Pham G, Monsel G, Chosidow O. Lice. Semin Cutan Med Surg. 2014;33(3):116-8.
10. Burkhart CN, Burkhart CG. Oral ivermectin therapy for phthiriasis palpebrum. Arch Ophthalmol. 2000;118(1):134-5.
11. Pierzchalski JL, Bretl DA, Matson SC. Phthirus pubis as a predictor for chlamydia infections in adolescents. Sex Transm Dis. 2002; 29(6):331-4.
12. Ryan MF. Phthiriasis palpebrarum infection: a concern for child abuse. J Emerg Med. 2014;46(6):e159-62.


Cataracts + Glaucoma = MIGS

Fewer complications and improved IOP control make this a viable option for many.

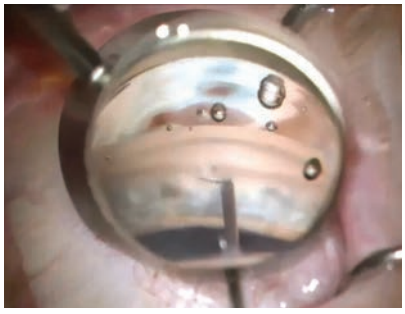
By Walter O. Whitley, OD, MBA, and Derek N. Cunningham, OD

When it comes to the treatment and management of glaucoma, one of today's hottest topics is minimally invasive glaucoma surgeries, or MIGS. MIGS procedures have been gaining momentum and are being adopted by surgeons in the United States and worldwide due to their efficacy and safety profile. Traditional glaucoma filtration surgeries have been limited, consisting of either a trabeculectomy or drainage implant. Although these traditional procedures can lower pressures to the single digits, they come with complications, including scar formation, bleb leaks, hypotony, choroidal hemorrhage, infection and cataract formation.

With the introduction of MIGS, eye care providers can offer patients an alternative glaucoma procedure to control and treat their glaucoma with less risk of complications. These procedures are indicated for mild to moderate open-angle glaucoma and are used in conjunction with cataract surgery, unlike canaloplasty and the Trabectome, which are performed as a single surgery.

MIGS

In previous columns, we introduced several early MIGS procedures. The first player in the market was the canaloplasty (Ellex) procedure (www.reviewofoptometry.com/content/c/33594/), followed by the Trabectome (Neomedix) (www.reviewofoptometry.com/content/c/35213/) and later the iStent (Glaukos) (www.reviewofoptometry.com/content/c/45133/). These procedures have been adopted by both glaucoma specialists and cataract surgeons alike due to their numerous benefits. Nonetheless, MIGS do have some disadvantages (*see "MIGS Pros and Cons."*)



Using a gonioprism, the iStent is secured into Schlemm's canal.

Comanagement

As comanaging optometrists, it is important to have a strong understanding of these procedures and the benefits they have over traditional glaucoma treatments and cataract surgery alone. About 19% of patients undergoing cataract surgery have a concurrent diagnosis of glaucoma.[1] Although patients may seem to be adequately treated with glaucoma medications, patient compliance and IOP control are not always guaranteed. Researchers have found that compliance increases with decreased dosage regimen and complexity.[2] Patients who are prescribed a once-daily medication show 79% compliance vs. 51% for QID regimens.[2] MIGS help address these concerns, in addition to improving vision and quality of life for patients with cataracts and glaucoma.

Over the next few columns, we will address several newer MIGS procedures currently available or under clinical investigation, including: the Hydrus Microstent (Ivantis), CyPass Micro-Stent (Transcend Medical), Xen Gel Stent (AqueSys), Solx Gold Shunt (Solx) and the ICE procedure (iStent + cataract surgery + endoscopic cyclophotocoagulation). Stay tuned to learn more about these exciting MIGS procedures. ■

1. Tseng VL, Yu F, Lum F, et al. Risk of fractures following cataract surgery in Medicare beneficiaries. JAMA. 2012;308(5):493-501.
2. Claxton AJ, Cramer J, Pierce C. A systematic review of the Associations between dose regimens and medication compliance. Clinical Therapeutics. 2001;23(8).

MIGS Pros and Cons[1,2]

Benefits:

- Combined with cataract surgery
- No bleb
- Spares the conjunctiva
- Reduces IOP
- Less glaucoma medications
- Decreased IOP fluctuations
- Less operating time
- Fewer serious complications
- Faster recovery
- Fewer follow-up appointments

Drawbacks:

- Hyphema
- IOP spikes
- Stent obstruction
- Less IOP lowering compared with traditional glaucoma surgery
- Inflammation

1. iStent. Connecting patients to the promise of micro invasive glaucoma surgery. www.glaukos.com/istent/.
2. Neomedix. Trabectome benefits. www.trabectome.com/Patients/TrabectomeBenefits/.

Down, Boy.

Help Tame Postoperative Ocular Inflammation and Pain With LOTEMAX® GEL

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.

LOTEMAX® GEL
loteprednol etabonate
ophthalmic gel 0.5%

BAUSCH + LOMB

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: Pregnancy Category C.

Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (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 Incorporated
Tampa, Florida 33637 USA

US Patent No. 5,800,807

US/LGX/15/0042
Based on 9269100-9269200 Revised: 9/2012

Product **Review**

Artificial Tears

Program Supplies First Responders With Eye Drops

Because first responders often work in conditions that can cause dry eye symptoms, Allergan is announcing the Refresh America campaign to help first responders alleviate their discomfort from dry eye. Every purchase of specially-marked packages of Refresh Optive between August 1, 2015 and July 31, 2016 will lead to donations of Refresh eye drops to certain first responder groups nationwide, the company says.

Allergan's partnership with the U.S. First Responders Association will help designate where donations will do the most good for our nation's best, the company says.

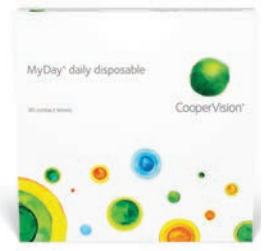
Visit www.helpRefreshAmerica.com.

Contact Lenses

New Daily Disposables

Contact lens wearers can now consider CooperVision's MyDay silicone hydrogel daily disposable contact lenses.

The lenses are designed to improve oxygen permeability with only 4.4% silicon, according to CooperVision. The lenses feature sphere powers from +6.00D to -10.00D; a base curve of 8.4mm; a diameter of 14.2mm; a Dk/t of 100 (at -3.00D); and a modulus of 0.4 MPa.



CooperVision has begun limited distribution and expects to begin shipping nationwide by fall 2015.

Visit www.coopervision.com.

Ophthalmic Lenses

New AR Coatings

Eye care professionals have three new anti-reflective lens coatings from Zeiss to offer spectacle wearers. The company's DuraVision line includes platinum, blueprotect and silver options. The platinum coating has a low residual reflectance (0.8%), better durability and an oil- and water-repelling topcoat and an anti-static layer, Zeiss says. Blueprotect also aims to reduce high-energy blue light often associated with digital device use. The silver is an affordable option for patients interested in anti-reflective coating.

Visit www.zeiss.com/vision.

Progressive Lenses

The new Varilux progressive lens designs are now available on managed vision care plans offered by VSP. The two new lenses are designed with technology that identifies and reduces aberrations, according to Essilor.

The Comfort W2+ lenses offer sharp vision even in low light, and the Physio W3+ lenses provide sharp vision and smooth transitions from distance to near fields of vision even in low light, Essilor says.

The company will provide ECPs with comprehensive training that includes a new ABO course, complementary dispenser training through ECP University and in-office sales training.

Visit www.varilux.com.

Lens Series Renamed

Kodak-branded lenses and services are now reorganized under the new Kodak Lens Professional Series name, according to Signet Armorlite.

The Kodak Lens Professional Series includes Kodak progressive lenses, digital single vision lenses, softwear lenses and anti-reflective lens coatings.

All Kodak lenses and lens coatings in the professional series will be available exclusively through independent eye care practices, Signet Armorlite says. This includes a new dual-side progressive lens design scheduled for release in August 2015, according to the company.

Visit www.kodaklens.com.

Patternless Edger

The latest finishing product from Coburn Technologies, the HPE-810 patternless edger, is now available. It provides users an accurate look with detailed images of bevel/groove positions, and faster processing allows users to modify the next job while a current job is under way, the company says.

It features customizable bevel heights for frames with short groove depth, concave shape processing and a clip editing function that allows far and near sight glasses, and even sunglasses, to be used on a single frame, according to Coburn.

Visit www.coburntechnologies.com. ■

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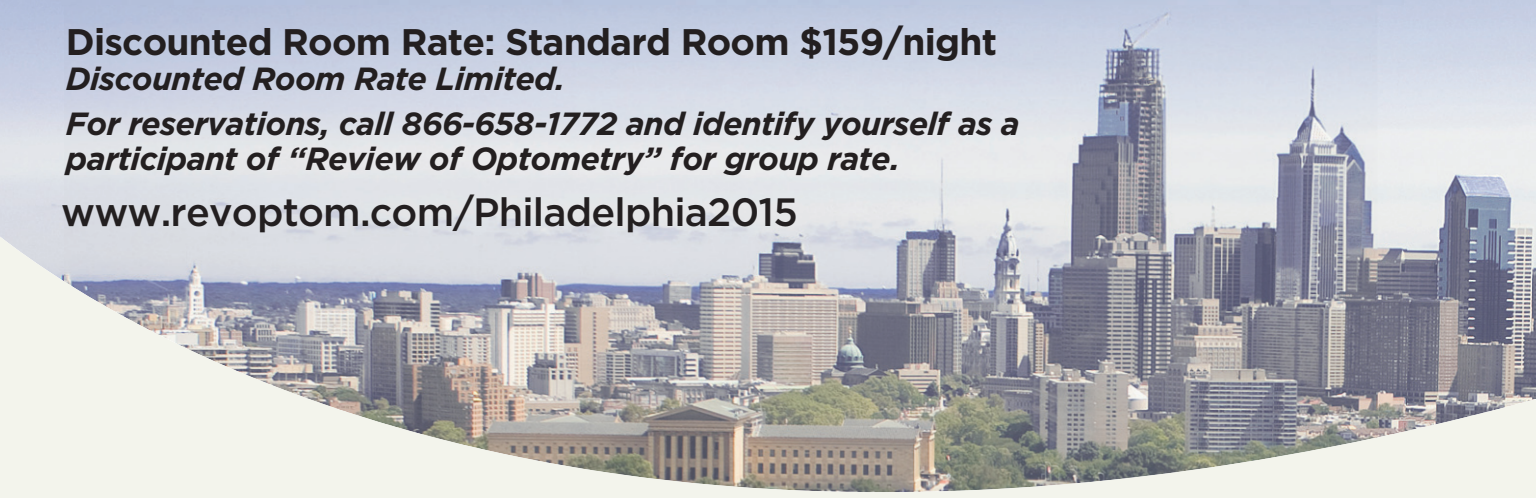

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■ **10-11.** *Forum on Ocular Disease*. Swan and Dolphin Hotel, Orlando, FL. Hosted by: PSS EyeCare. Key faculty: Ron Melton, Randall Thomas, Deepak Gupta, Jerome Sherman. CE hours: 18. To register, email education@psseyecare.com or go to www.psseyecare.com.

■ **11.** *Pediatrics & Low Vision Course*. Marshall B. Ketchum University, Fullerton, CA. Hosted by: SCCO at Marshall B. Ketchum University. Key faculty: Carmen Barnhardt, Sue Cotter, Lynn Lowell, John Tassinari. CE hours: 8. To register, email Antoinette Smith and Bonnie Dellatorre at ce@ketchum.edu, call (714) 449-7495 or go to www.ketchum.edu/ce.

■ **13-14.** *Michigan Optometric Association 47th Annual Fall Seminar*. Lansing Center, Lansing, MI. Hosted by: Michigan Optometric Association. CE hours: 12 to 14. To register, email Amy Root at amy@themoa.org or go to www.themoa.org.

■ **15-18.** *MOA Annual Conference, Trade Show & Golf Tournament*. Downtown Marriott, Kansas City, MO. Hosted by: Missouri Optometric Association. CE hours: 16. To register, email Sue Brown at sue@moeyecare.org, call (573) 635-6151 or go to www.moeyecareconference.org.

■ **16-25.** *Classic China 2015*. Beijing, Xi'an, Shanghai, China. Hosted by: iTravel CE. Key faculty: John McGreal. CE hours: 16. To register, email info@iTravelCE.com or go to www.iTravelCE.com.

■ **17.** *San Francisco Optometric Glaucoma Symposium*. Marriott Union Square, San Francisco, CA. Hosted by: Review of Optometry. Key faculty: John Flanagan, Andrew Iwach. CE hours: 6. To register, go to www.reviewofoptometry.com/conferences.

■ **23-25.** *GOA Fall Education Conference*. UGA Hotel and Conference Center, Athens, GA. Hosted by: Georgia Optometric Association. CE hours: 18. To register, email Vanessa Grosso at VanessaGOA@aol.com, call (770) 961-9866 ext. 1 or go to www.GOAeyes.com.

■ **24-25.** *VOA 2015 Fall Conference*. Kingsmill Resort, Williamsburg, VA. Hosted by: Virginia Optometric Association. CE hours: 8. To register, email office@thevoa.org, call (804) 643-0309 or go to www.thevoa.org.

■ **24-25.** *CE in Fort Worth*. Marriott Dallas/Fort Worth Hotel & Golf Club at Champions Circle, Fort Worth, TX. Hosted by: University of Houston College of Optometry. Key faculty: Suzanne Wickum. CE hours: 16. To register, email optce@uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu.

■ **24-25.** *Primary EyeCare Conference*. Renaissance hotel, Westchester, NY. Hosted by: PSS EyeCare. Key faculty: Deepak Gupta, Mile Brujic, Kimberly Reed. CE hours: 16. To register, email education@psseyecare.com or go to www.psseyecare.com.

■ **24-26.** *Annual Education Conference*. Mystic Marriott Hotel and Spa, Groton, CT. Hosted by: Connecticut Association of Optometrists. CE hours: 18. To register, email Stephanie Bartos at sbartos@cteyes.org, Lynn Sedlak at lsedlak@cteyes.org, call (860) 529-1900 or go to www.cteyes.org.

■ **30-Nov. 1.** *26th Annual Education Conference*. Abe Martin Lodge, Nashville, IN. Hosted by: Fellowship of Christian Optometrists, International. CE hours: 12. To register, email Kelly Frantz at kfrantz@ico.edu or go to www.fcoint.net.

November 2015

■ **6.** *Envision University Presents Assistive Technology*. Envision, Wichita, KS. Hosted by: Envision University. CE hours: 4. To register, email Bonnie Harrell at bonnie.harrell@envisionus.com, call (316) 440-1514 or go to www.envisionuniversity.org.

■ **6-8.** *Fall Congress*. Omni Grove Park Inn, Asheville, NC. Hosted by: North Carolina State Optometric Society. CE hours: 18. To register, email Lauren Godwin at lauren@nceyes.org or call (919) 977-6964.

■ **6-8.** *New Technologies and Treatments in Vision Care*. Sheraton Philadelphia Downtown Hotel, Philadelphia. Hosted by: *Review of Optometry*. Key faculty: Paul Karpecki (meeting chair), Blair Lonsberry, Douglas Devries, Jeffry Gerson. CE hours: 17. To register, email Lois DiDomenico at ReviewMeetings@jobson.com, call (866) 658-1772 or go to www.reviewofoptometry.com.

■ **6-9.** *Conference on Primary EyeCare*. Marriott Hotel, White Plains, NY. Hosted by: PSS EyeCare. CE hours: 16. To register, email Sonia Kumari at education@psseyecare.com, call (203) 415-3087 or go to www.psseyecare.com.

■ **7-8.** *Glaucoma Grand Rounds Program*. Western University College of Optometry Pomona Campus, Pomona, CA. Hosted by: Western University of Health Sciences, College of Optometry. Key faculty: Raymond Maeda, Valerie Wren, Pinakin Davey. CE hours: 16. To register, email Maria Espinosa at mespinosa@westernu.edu, call (909) 706-3493 or go to www.westernu.edu.

■ **8.** *Fall 2015 Education Conference*. Fredericksburg Hospitality House Hotel & Conference Center, Fredericksburg, VA. Hosted by: Virginia Academy of Optometry. Key faculty: Ron Melton, Randall Thomas. CE hours: 4. To register, email John Dresely at jwdod@verizon.net.

■ **13-14**. *2015 WOA Primary Care Symposium*. Country Springs Hotel, Waukesha, WI. Hosted by: Wisconsin Optometric Association. CE hours: 9. To register, email Joleen Breunig at joleen@woa-eyes.org or go to www.woa-eyes.org.

■ **13-15.** *Monterey Symposium 2015*. Monterey Marriott and Conference Center, Monterey, CA. Hosted by: California Optometric Association. CE hours: 60. To register, email Sarah Harbin at sharbin@coavosion.org, call (916) 266-5022 or go to www.montereysymposium.com.

■ **14-15.** *Heart of America Congress*. Kansas City, KS. Hosted

by: OEP Foundation. CE hours: 13. To register, email Theresa Krejci at TheresaKrejciOEP@verizon.net or go to www.oepf.org.

■ **18.** *AAO-NJ Conference.* Jumping Brook Country Club, Neptune, NJ. Hosted by: American Academy of Optometry New Jersey Chapter. CE hours: 2. To register, email Dennis Lyons at dhl2020@aol.com or call (732) 920-0110.

■ **21-22.** *Everything Therapeutic – San Antonio.* Westin Riverwalk Hotel, San Antonio, TX. Hosted by: University of Houston College of Optometry. Key faculty: Bruce Onofrey. CE hours: 16. To register, email optce@uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu/.

■ **22.** *Clinical Chiefs Optometry Update.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: SCCO at Marshall B. Ketchum University. Key faculty: Mark Sawamura. CE hours: 8. To register, email ce@ketchum.edu, call (714) 449-7495 or go to www.ketchum.edu/ce.

December 2015

■ **2-6.** *OEP Clinical Curriculum: VT/Visual Dysfunctions.* Office of Robert Lewis, Phoenix. Hosted by: OEP Foundation. Key faculty: Robert Lewis. CE hours: 35. To register, email Theresa Krejci at TheresaKrejciOEP@verizon.net, call (800) 447-0370 or go to www.oepf.org.

■ **5.** *Retina Update 2015.* Anaheim, CA. Hosted by: *Review of Optometry* and Optometric Retina Society. Key faculty: Brad Sutton, Joseph Pizzimenti. CE hours: 12. To register, email Lois DiDomenico at ReviewMeetings@jobson.com, call (866) 658-1772 or go to www.revoptom.com.

■ **5-6.** *Malinovsky Ocular Disease Seminar.* Bloomington, IN. Hosted by: IU School of Optometry. Key faculty: Todd Peabody, Jeff Perotti, Don Lyon, Tony Van Alstine, Patty Henderson. CE hours: 14. To register, email Cheryl Oldfield at coldfiel@indiana.edu or go to www.opt.indiana.edu/ce/seminars.htm.

■ **5-6.** *32nd Annual Cornea, Contact Lens & Contemporary Vision Care Symposium.* Westin Memorial City Hotel, Houston. Hosted by: University of Houston College of Optometry. Key faculty: Jan Bergmanson. CE hours: 16. To register, email optce@uh.edu, call (713) 743-1900 or go to ce.opt.uh.edu/.

■ **6.** *Annual GP Lens Symposium.* Marshall B. Ketchum University, Fullerton, CA. Hosted by: SCCO at Marshall B. Ketchum University. Key faculty: Barry Weissman, Brooke Messer. CE hours: 8. To register, email ce@ketchum.edu, call (714) 449-7495 or go to www.ketchum.edu/ce. ■

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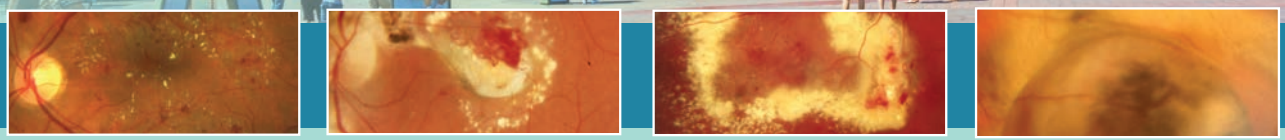

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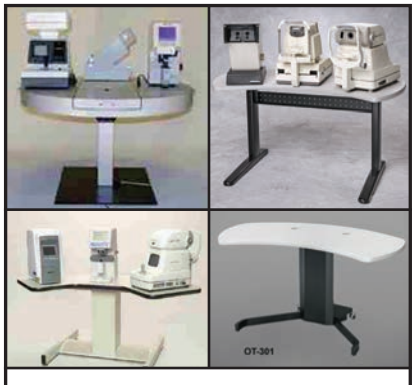

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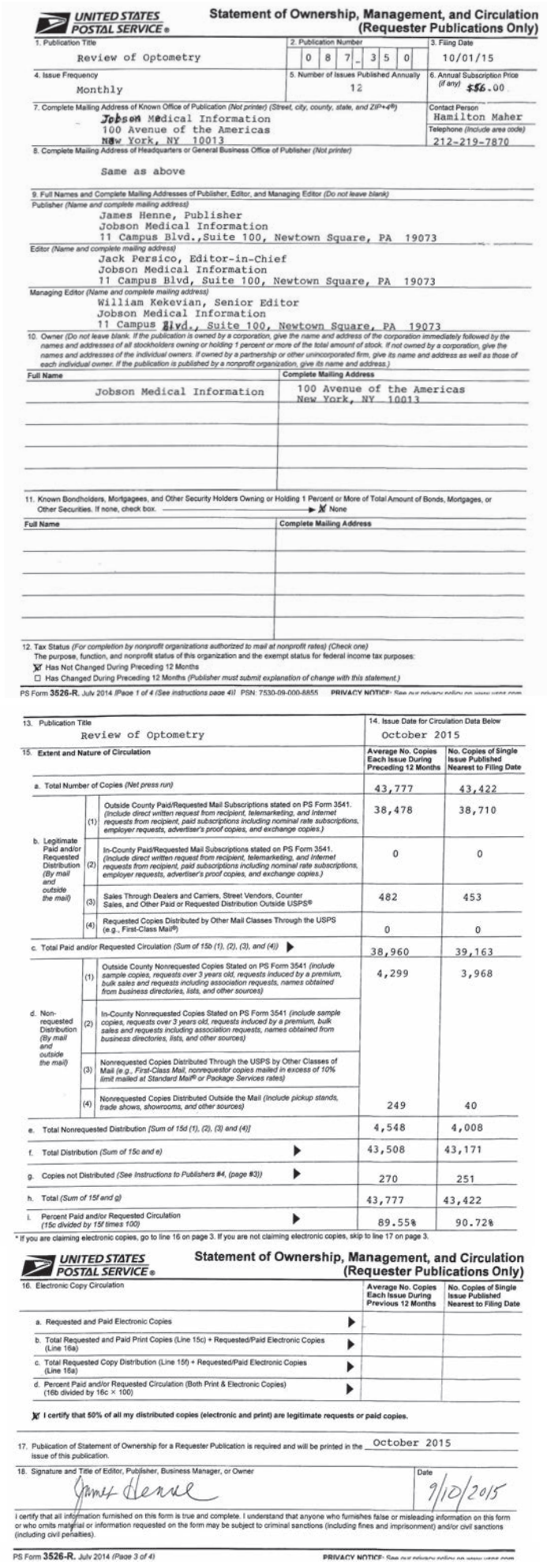

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13. Publication Title	14. Issue Date for Circulation Data Below
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15. Extent and Nature of Circulation			Average No. Copies Each Issue During Preceding 12 Months	No. Copies of Single Issue Published Nearest to Filing Date
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TRAVATAN Z®
(travoprost ophthalmic solution) 0.004%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE
TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION
The recommended dosage is one drop in the affected eye(s) once daily in the evening.
TRAVATAN Z® (travoprost ophthalmic solution) should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the intraocular pressure lowering effect.

Reduction of the intraocular pressure starts approximately 2 hours after the first administration with maximum effect reached after 12 hours.

TRAVATAN Z® Solution may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Pigmentation
Travoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as travoprost 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 travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. 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 TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes
TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intraocular Inflammation
TRAVATAN Z® Solution should be used with caution in patients with active intraocular inflammation (e.g., uveitis) because the inflammation may be exacerbated.

Macular Edema
Macular edema, including cystoid macular edema, has been reported during treatment with travoprost ophthalmic solution. TRAVATAN Z® Solution 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.

Angle-closure, Inflammatory or Neovascular Glaucoma
TRAVATAN Z® Solution has not been evaluated for the treatment of angle-closure, inflammatory or neovascular glaucoma.

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.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS
Clinical Studies Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice. The most common adverse reaction observed in controlled clinical studies with TRAVATAN® (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% was ocular hyperemia which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus. Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN® or TRAVATAN Z® Solutions included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal staining, dry eye, iris discoloration, keratitis, lid margin crusting, ocular inflammation, photophobia, subconjunctival hemorrhage and tearing.

Nonocular adverse reactions reported at an incidence of 1 to 5% in these clinical studies were allergy, angina pectoris, anxiety, arthritis, back pain, bradycardia, bronchitis, chest pain, cold/flu syndrome, depression, dyspepsia, gastrointestinal disorder, headache, hypercholesterolemia, hypertension, hypotension, infection, pain, prostate disorder, sinusitis, urinary incontinence and urinary tract infections.

In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

USE IN SPECIFIC POPULATIONS
Pregnancy
Pregnancy Category C
Teratogenic effects: Travoprost was teratogenic in rats, at an intravenous (IV) dose up to 10 mcg/kg/day (250 times the maximal recommended human ocular dose (MRHOD), evidenced by an increase in the incidence of skeletal malformations as well as external and visceral malformations, such as fused sternebrae, domed head and hydrocephaly. Travoprost was not teratogenic in rats at IV doses up to 3 mcg/kg/day (75 times the MRHOD), or in mice at subcutaneous doses up to 1 mcg/kg/day (25 times the MRHOD). Travoprost produced an increase in post-implantation losses and a decrease in fetal viability in rats at IV doses > 3 mcg/kg/day (75 times the MRHOD) and in mice at subcutaneous doses > 0.3 mcg/kg/day (7.5 times the MRHOD).

In the offspring of female rats that received travoprost subcutaneously from Day 7 of pregnancy to lactation Day 21 at doses of ≥ 0.12 mcg/kg/day (3 times the MRHOD), the incidence of postnatal mortality was increased, and neonatal body weight gain was decreased. Neonatal development was also affected, evidenced by delayed eye opening, pinna detachment and preputial separation, and by decreased motor activity.

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® Solution should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers
A study in lactating rats demonstrated that radiolabeled travoprost and/or its metabolites were excreted in milk. It is not known whether this drug or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when TRAVATAN Z® Solution is administered to a nursing woman.

Pediatric Use
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

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

Hepatic and Renal Impairment
Travoprost ophthalmic solution 0.004% has been studied in patients with hepatic impairment and also in patients with renal impairment. No clinically relevant changes in hematology, blood chemistry, or urinalysis laboratory data were observed in these patients.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Two-year carcinogenicity studies in mice and rats at subcutaneous doses of 10, 30, or 100 mcg/kg/day did not show any evidence of carcinogenic potential. However, at 100 mcg/kg/day, male rats were only treated for 82 weeks, and the maximum tolerated dose (MTD) was not reached in the mouse study. The high dose (100 mcg/kg) corresponds to exposure levels over 400 times the human exposure at the maximum recommended human ocular dose (MRHOD) of 0.04 mcg/kg, based on plasma active drug levels. Travoprost was not mutagenic in the Ames test, mouse micronucleus test or rat chromosome aberration assay. A slight increase in the mutant frequency was observed in one of two mouse lymphoma assays in the presence of rat S-9 activation enzymes.

Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 mcg/kg/day [250 times the maximum recommended human ocular dose of 0.04 mcg/kg/day on a mcg/kg basis (MRHOD)]. At 10 mcg/kg/day, the mean number of corpora lutea was reduced, and the post-implantation losses were increased. These effects were not observed at 3 mcg/kg/day (75 times the MRHOD).

PATIENT COUNSELING INFORMATION
Potential for Pigmentation
Patients should be advised about the potential for increased brown pigmentation of the iris, which may be permanent. Patients should also be informed about the possibility of eyelid skin darkening, which may be reversible after discontinuation of TRAVATAN Z® (travoprost ophthalmic solution) 0.004%.

Potential for Eyelash Changes
Patients should also be informed of the possibility of eyelash and vellus hair changes in the treated eye during treatment with TRAVATAN Z® Solution. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or vellus hairs, and/or direction of eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container
Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice
Patients should also be advised that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician's advice concerning the continued use of TRAVATAN Z® Solution.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Use with Other Ophthalmic Drugs
If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes between applications.

Rx Only
U.S. Patent Nos. 5,631,287; 5,889,052, 6,011,062; 6,235,781; 6,503,497; and 6,849,253

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Diagnostic **Quiz**

Personal Foul

By Andrew S. Gurwood, OD

History

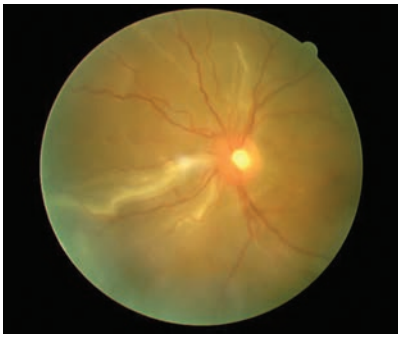
A 32-year-old Caucasian male reported to the office with a chief complaint of vision loss in the left eye following trauma received during a basketball game.

The patient explained that he had been hit around the left eye with an elbow during a game. Immediately follwing the injury he saw an "impressive" flash of light and, ever since then, said he felt as if some of the floor was "missing" or "foggy."

The patient's systemic and ocular histories were unremarkable and he denied exposure to chemicals or allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OU at distance and near. His external examination was normal with no evidence of afferent pupillary defect.



This 32-year-old patient lost vision following trauma received during a basketball game. Can this dilated fundus photo help determine a diagnosis?

His peripheral confrontational visual field exam found distorted and missing floor in the inferior temporal quadrant.

The biomicroscopic examination of the patient's anterior segments found normal structures. Using Goldmann applanation tonometry, his intraocular pressure was measured at 15mm Hg OU.

The pertinent posterior segment findings are shown in the accompanying fundus photograph.

Your Diagnosis

Does this case require any additional tests? What does this patient's history and clinical findings tell you about his likely diagnosies? How would you manage this patient? What's the patient's likely prognosis? To find out, please visit us online at www.reviewofoptometry.com.

Retina Quiz Answers *(from page 134):* 1) b; 2) d; 3) d; 4) a; 5) a.

Next Month in the Mag

In November, *Review of Optometry* takes a look at building a high-volume practice. Topics include:

- *Secrets of the Pros—Build Volume in Contact Lenses, Spectacles and Pharmaceuticals*
Success stories from optometrists who took on more patients and how they learned to manage a high-volume practice.

- *Office Design Principles for Busy Practices*
How the physical layout and other amenities at the practice can affect patient flow and satisfaction.

- *Hiring Another OD to Boost Your Capacity—and Profits*
Bringing in another doctor comes with pros and cons. Learn how to structure the buy-in and divide labor for the best results.

- *10 Staff Management Tips to Boost Productivity*
Know how to delegate, conduct staff meetings, train and provide performance reviews.

Also in this issue:
- *Does Nutrition Influence the Course of Glaucoma?*
- *Ocular Oncology: Diagnostic Principles and Pearls*
- *Annual* Review of Optometry *Income Survey Results*

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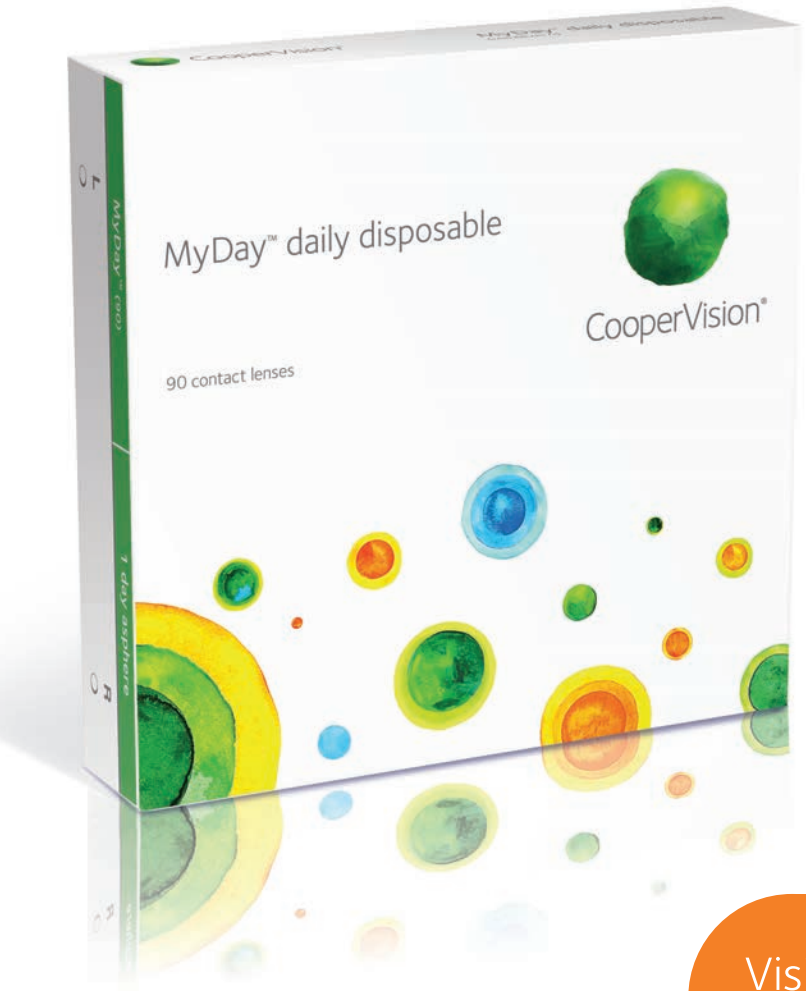

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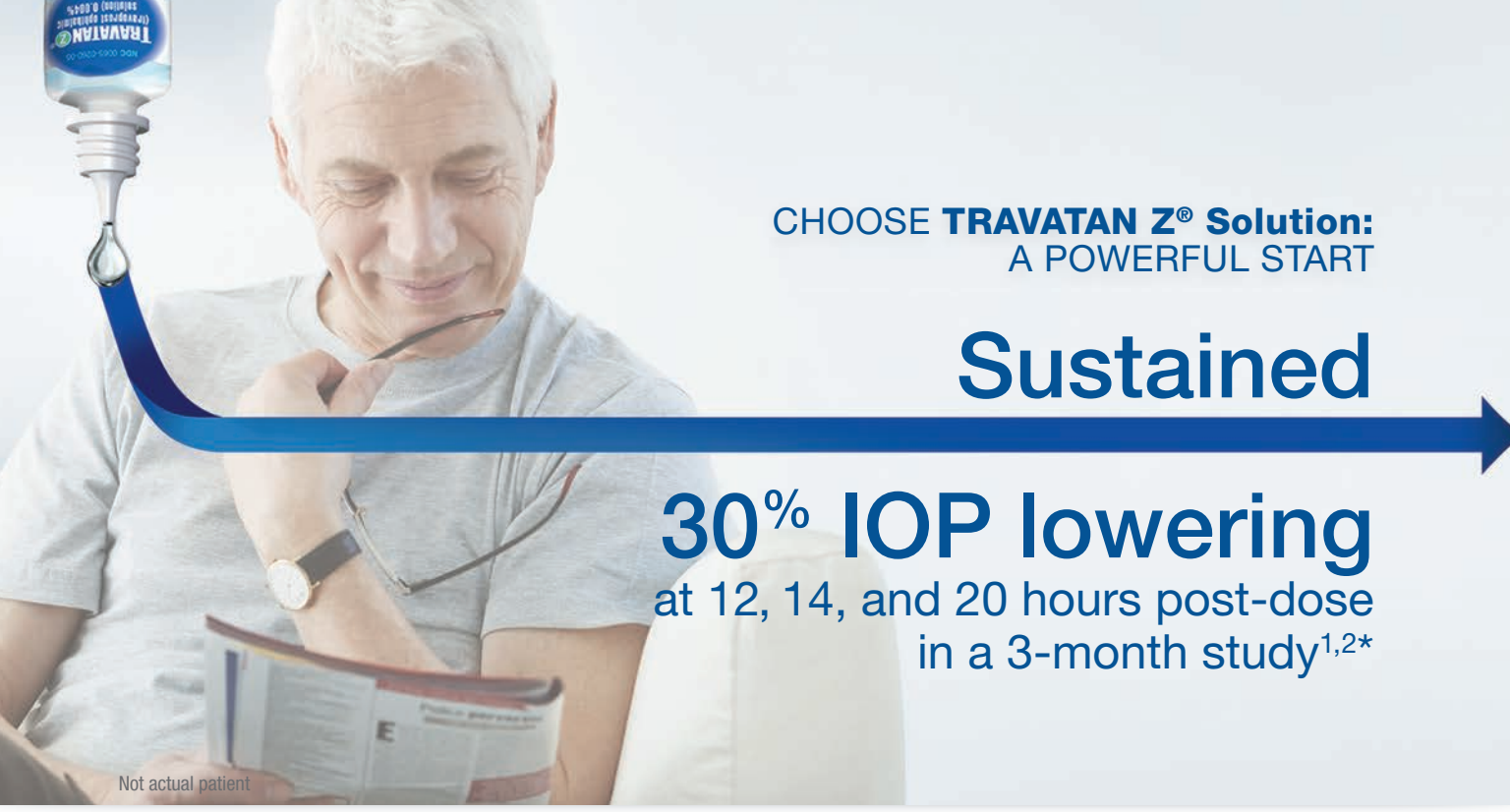

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INDICATIONS AND USAGE

TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration

The recommended dosage is 1 drop in the affected eye(s) once daily in the evening. TRAVATAN Z® Solution should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the IOP-lowering effect.

TRAVATAN Z® Solution may be used concomitantly with other topical ophthalmic drug products to lower IOP. If more than 1 topical ophthalmic drug is being used, the drugs should be administered at least 5 minutes apart.

IMPORTANT SAFETY INFORMATION

Warnings and Precautions

Pigmentation—Travoprost ophthalmic solution has been reported to increase the pigmentation of the iris, periorbital tissue (eyelid), and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. The long-term effects of increased pigmentation are not known. While treatment with TRAVATAN Z® Solution can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes—TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Use With Contact Lenses—Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Adverse Reactions

The most common adverse reaction observed in controlled clinical studies with TRAVATAN Z® Solution was ocular hyperemia, which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain, and pruritus. In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

Use in Specific Populations

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

For additional information about TRAVATAN Z® Solution, please see the brief summary of Prescribing Information on the adjacent page.

*__Study Design:__ Double-masked, randomized, parallel-group, multicenter non-inferiority comparison of the efficacy and safety of travoprost 0.004% preserved with benzalkonium chloride (BAK) to TRAVATAN Z® Solution after 3 months of treatment in patients with open-angle glaucoma or ocular hypertension. Baseline IOPs were 27.0 mm Hg (n=322), 25.5 mm Hg (n=322), and 24.8 mm Hg (n=322) at 8 AM, 10 AM, and 4 PM for TRAVATAN Z® Solution. At the end of Month 3, the TRAVATAN Z® Solution group had mean IOPs (95% CI) of 18.7 mm Hg (-0.4, 0.5), 17.7 mm Hg (-0.4, 0.6), and 17.4 mm Hg (-0.2, 0.8) at 8 AM, 10 AM, and 4 PM, respectively. Statistical equivalent reductions in IOP (95% confidence interval about the treatment differences were entirely within ±1.5 mm Hg) were demonstrated between the treatments at all study visits during the 3 months of treatment.

References: 1. Data on file, 2013. **2.** Lewis RA, Katz GJ, Weiss MJ, et al. Travoprost 0.004% with and without benzalkonium chloride: a comparison of safety and efficacy. *J Glaucoma.* 2007;16(1): 98-103. **3.** Drugs@FDA. FDA Approved Drug Products: TRAVATAN Z page. US Food and Drug Administration website. http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm. Accessed March 31, 2015.

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