

June 15, 2012

REVIEW[®] OF OPTOMETRY

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JOSHUA MARC LAHIFF, OD

A Growing Practice Is Built on Happy Patients

Garnering new patients through **word-of-mouth referrals** is more than a great marketing strategy—it is the key to success that endures the test of time.

Successful practices see more patients

Focusing your practice on getting new patients rather than dollars per patient is a more pragmatic—and preferable—business approach. This is true for any practice. In a 2009 study of independent practices, it was reported that the practices with the greatest gross revenue see three times as many patients as the average practice. Furthermore, there is little difference in the gross revenue per exam between rural and urban settings, small and large practices.¹

Increased patient traffic and improved exam productivity have major impacts on practice revenue. In fact, another 2009 study found that, on average, 52% of a practice's gross revenues come from exam fees alone.² This constitutes nearly 2/3 of gross income.^{1,2}

According to Dr Josh LaHiff, practicing optometrist in Cheyenne, WY, increasing patient traffic is all about providing patients with an outstanding experience: "You want to be able to provide them [patients] with such an experience that they're going to invest more in your clinic." Dr LaHiff should know. He sees at least 30 patients per day. Which is impressive considering there are 28 other eye care professionals in Cheyenne, a town with a population of only 50,000.

Success is built on a happy patient experience

Increasing patient traffic through referrals is often as simple as delivering excellent service and the healthiest products. "To be the best, you have to use the best. If you wow the patient, treat them like gold, that's how you really generate those referrals," says Dr LaHiff.

That's why Dr LaHiff believes the most effective approach to achieving profitable, long-term patient relationships is to "do what's best for the patient, even if it may not be the most profitable option for the doctor initially, because it is what is right, and the profit will come as an annuity in return visits and the happiness of the patient."

High patient satisfaction inside and outside of the practice is the most effective catalyst for generating new patients. This can be especially true for contact lens patients. A survey of 1086 patients found that those who are happy in their contact lenses are nearly 2x more likely to recommend their eye doctor than those who are unhappy in their lenses.²

ACUVUE® Brand—proven to keep patients satisfied

The proven method to achieve satisfaction is to use a product with consistently successful results. Dr LaHiff believes that, "When you use a product that you know is going to work time and time again, it cuts your chair time down and it's easier for you, your staff, and your patients to put them in something that's comfortable."

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According to Dr LaHiff, "The more you use a superior product, the better the experience is going to be for the patient." And that's been a proven strategy for the success of his practice. ■

Joshua Marc LaHiff, OD, is a partner and practicing optometrist at Cheyenne Vision Clinic in Cheyenne, WY, and clinical instructor for the Illinois College of Optometry in Chicago, IL. He received his doctor of optometry degree with honors from the Pacific University College of Optometry in Forest Grove, OR.

Dr LaHiff is a member of numerous associations and serves as a speaker and professional consultant for several medical companies, including VISTAKON® Division of Johnson & Johnson Vision Care, Inc. He was compensated for this article.

GROW



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Reference: 1. Data on file. Johnson & Johnson Vision Care, Inc. 2011.

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IN THE NEWS

WellPoint, Inc., one of the nation's largest health benefits companies, announced that it **will acquire 1-800 CONTACTS**, the largest direct-to-consumer retailer of contact lenses in the U.S. "This acquisition strategically aligns with our efforts to capitalize on new opportunities for growth, and further diversifies the company's revenue stream into the complementary and higher-margin eyewear business," said Angela F. Braly, chair, president and chief executive officer of WellPoint. The acquisition is expected to close in the third quarter of 2012.

W. Lee Ball, Jr., O.D., has been appointed to the Board of Directors of the **American Optometric Foundation (AOF)**. The AOF, an affiliate of the **American Academy of Optometry**, is a philanthropic organization devoted to the advancement of optometric education and research.

Revenue in the "optometrists industry" is expected to increase at an annualized rate of 2.9% to **\$14 billion** during the next five years, according to an industry report from the research firm IBISWorld. "While low disposable income and high unemployment deterred some consumers from seeking eye care, operators maintained growth by reducing prices and catering to favorably shifting demographics. The slowly recovering economy is expected to foster **stronger growth** in 2012," IBISWorld says. Optometrist offices will also face rising **competition** from large chain retailers, such as Walmart. Optometrists are forecasted to focus more on providing the **full scope of medical eye care** as a result.

Equal Compensation for Louisiana O.D.s

New legislation ensures proper reimbursement from participating HMOs, PPOs.

Optometrists in Louisiana should soon be entitled to receive the same reimbursements as ophthalmologists for the provision of medical eye care or vision services from participating insurance companies or health care plans.

This includes compensation for medical and vision services from participating health maintenance organizations (HMOs), preferred provider organizations (PPOs), managed care organizations, plans or contracts of insurance, or any medical hospital service contracts.

These proposals were outlined in Louisiana State Bill 669, which was approved by the Louisiana House and Senate on June 3. At press time, the governor was expected to sign the bill into law.

In addition, SB-669 stipulates:

- An HMO, PPO, managed care organization, plan or contract of insurance, or any medical or hospital service contract shall not impose a co-payment, co-insurance

amount, or any other fee on a covered participant or insured party that is greater than the amount charged for the same service when provided by an allopathic physician or an osteopathic physician.

- It shall be unlawful for an HMO, PPO, managed care organization, or plan or contract of insurance to require a duly licensed optometrist to participate as a provider in another medical or vision care plan or contract as a condition of or requirement for participation by such a duly licensed optometrist as a provider in any medical or vision care plan or contract.

"We're not asking for special treatment," said James Sandefur, O.D., president of the Optometry Association of Louisiana, in his testimony to the legislature. "We just want to be treated fairly. It's not right for health plans to pay optometrists less than ophthalmologists for the same identical services."

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 - Macular hole³
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Puerto Rican Bill Would Allow TPAs

Optometrists in Puerto Rico have tried at least six previous times—unsuccessfully—to gain authority to prescribe therapeutic pharmaceutical agents.

Will the seventh time be the charm?

Much is riding on this newest bill (P.S. 2634), which was submitted to the legislature in mid-May by Sen. Angel Martinez Santiago. Puerto Rico is the only jurisdiction in all of the United States and its territories where optometrists are not permitted to prescribe any topical agents for the treatment of ocular disease.

At stake: “The U.S. Armed Forces have adopted a new policy that only optometrists holding a therapeutic license can practice in their VA hospitals,” says Katerin A. Ortiz, O.D., who is the liaison from the Colegio de Optometras de Puerto Rico (its equivalent of a state association) to the AOA, as



Puerto Rico’s Legislative Assembly is set to vote later this month on a bill that would allow TPAs for its optometrists.

well as the Director of Pediatrics at the InterAmerican University of Puerto Rico School of Optometry.

This change in policy means that “Optometrists with a Puerto Rican license would therefore be given a lower rank than another optometrist with the same degree holding a license from the U.S.,” Dr. Ortiz says.

Last year, private practice optometrist Osvaldo Negrón, of

Bayamon, P.R., posted a petition online to bring attention to the issue and to encourage the commonwealth’s government to allow therapeutic prescriptive authority for optometrists. At last count, the petition had nearly 1,200 electronic signatures.

Now, the current bill would amend the optometric law to allow optometrists in Puerto Rico to prescribe all topical drugs, including glaucoma meds, as long as the optometrist passes the Treatment and Management of Ocular Disease (TMOD) part of optometry boards, Dr. Ortiz says.

The bill is currently in the Senate. It must also be passed in the House of Representatives to be approved. The legislature is expected to make its decision by June 25.

The island commonwealth has a population of about 4 million, including more than 100 ophthalmologists and some 475 optometrists.

Hall of Fame Honors 2012 Inductees

The National Optometry Hall of Fame, administered by Optometry Cares—The AOA Foundation, will induct five new hall of fame members into the elite group during a ceremony at Optometry’s Meeting on Thursday, June 28 at 7:00 p.m.

• **Thomas Lewis, O.D., Ph.D.**, a 1970 graduate of the Pennsylvania College of Optometry, has held various administrative and teaching positions at the college, in-



cluding dean of Academic Affairs.

In 1989, he was named president of PCO, and the college grew to university status under his leadership.

• **Frank Fontana, O.D.**, a 1949 graduate of the Illinois College of Optometry, has been involved with clinical investigations, consulting, writing and lecturing throughout his career, spanning



more than six decades.

“Uncle Frank” has served as a

consulting editor for *Review of Optometry* since 1999, and has been an adjunct assistant professor and a member of the research panel of the Center for Corneal and Contact Lens Research at the University of Missouri at St. Louis College of Optometry.

In 2011, he established the “Dr. Frank & Mrs. Dorris Fontana Optometry” endowment scholarship at the university.

Other 2012 inductees include Kevin Alexander, O.D., Ph.D., James A. Boucher, O.D., and William “Billy” Cochran, O.D.

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Bill Calls for Repeal of SGR Formula

A House bill introduced last month would stop an approximate 30% Medicare pay cut next year by repealing the program's sustainable growth rate (SGR) formula and phase out its fee-for-service payment system.

In addition, the "The Medicare Physician Payment Innovation Act of 2012" states it would stabilize current payment rates to ensure beneficiary access in the near-term, eliminate scheduled SGR cuts, and create incentives for undervalued primary, preventive and coordinated care services.

"With the growing Medicare uncertainty facing all physicians and our patients, the AOA is encouraged by a number of emerging bipartisan proposals aimed at transforming Medicare's broken payment system into one that ensures stable and fair reimbursement, safeguards our full-scope role, and respects our relationship with our patients," says Roger Jordan, O.D., chair of the AOA Federal Relations Committee. "As Congress and the White House dig deeper into the details in the coming weeks and months, the AOA will continue working to lock-in our recent payment and other

gains that fully recognize optometry's essential primary care role in Medicare."

The bill is sponsored by Rep. Allyson Schwartz (D-PA) and Rep. Joe Heck, M.D. (R-NV).

Key points of their bill include:

- **Repeal the sustainable growth rate permanently:** By eliminating the \$300 billion debt to the Medicare program, this provision aims to restore stability and fiscal transparency to the payment system, and attempts to establish a clear path to comprehensive payment reform. The cost of the repeal is expected to be offset from the savings based on the reduction in military operations in Iraq and Afghanistan.

- **Stabilize the current payment system:** 2012 payment levels would be maintained through December 31, 2013. From 2014 to 2017, Medicare would raise rates by an annual 2.5% for primary care, preventive and care-coordination services. Rates for all other physician services would increase by 0.5% annually. The differential represents an attempt to emphasize primary care over procedure-oriented specialty care, according to the bill.

- **Reward clinicians for high-quality, high-value care while disincentivizing fragmented, volume-driven care:** In 2018, new payment models that reward physicians for "high quality, high value care," as opposed to the volume of services, will be introduced. These new models will build on Medicare demonstration projects now underway for accountable care organizations, medical homes and shared savings.

- **Because 2018 is considered a transitional year, Medicare will continue to pay physicians on a fee-for-service basis, but only at 2017 levels. Beginning in 2019, physicians who stick with fee-for-service reimbursement will suffer pay cuts: 2% in 2019, 3% in 2020, 4% in 2021, and 5% in 2022. Fee-for-service rates will remain frozen at 2022 levels in the years beyond. In contrast, physicians participating in the new payment models "will continue to receive stable reimbursement" with the opportunity to earn even more based on their performance."**

At press time, the bill had been referred to the House Energy and Commerce Committee for further consideration.

Call for Eye-catching Office Design Entries!

Do patients admire the earthy tones and plush seating in your waiting room? Is your office decor eye-catching? Then we've got a chance for you to showcase your style. We're now accepting entries for *Review's* 2012 Office Design Contest—the deadline for submissions will be Sept. 15, 2012.

Download an entry form today at www.revoptom.com by clicking "Web Exclusives" on the top left menu. All entries will be presented anonymously to our expert panel of judges and ranked on a scale according to use of space, stylistic appeal and functionality.

Please send all completed entries and high-resolution photos to Colleen Mullarkey, Senior Editor/Senior Web Editor, at cmullarkey@jobson.com. You can also e-mail her with any questions about submission.



A Bright Idea for Nearsightedness

Compact fluorescent bulbs are already saving people money on electricity bills—could these light bulbs help save their vision, too?

That's what researchers at the University of Alabama at Birmingham are hoping, and they've already taken a step in that direction. They found that small increases in daily artificial light slowed the development of nearsightedness by 40% in tree shrews (close relatives of primates).

This study is the first to show that increasing daily fluorescent light levels can slow the development of myopia. The team used a goggle that lets in light but no images to produce myopia in one eye of each tree shrew.

They found that a group exposed to elevated fluorescent light levels for eight hours per day developed 47% less myopia than a

control group exposed to normal indoor lighting, even though the images were neither more nor less blurry.

They are currently experimenting with light levels and times to see if a short, bright light treatment could be effective. "If we can find the best kind of light, treatment period and light level, we'll have the scientific justification to begin studies raising light levels in schools, for instance," says Thomas Norton, Ph.D., who headed up the research team. "Compact fluorescent bulbs use much less electricity than standard light bulbs, and future programs raising light levels will have more impact the less expensive they are."

Sieglwart JT Jr., Ward AH, Norton TT. Moderately elevated fluorescent light levels slow form deprivation and minus lens-induced myopia development in tree shrews. Paper presented at the Association for Research in Vision and Ophthalmology, May 8, 2012: Fort Lauderdale, Fla.



Recent study is the first to show that increasing daily fluorescent light levels can slow the development of myopia.

Glaucoma Worsens in Winter?

Intraocular pressure (IOP) and visual field sensitivity peak in the winter months, especially in people with early glaucoma, according to new data presented at the 2012 ARVO meeting. The seasonal effect was much larger in regions with more extreme seasonal variations in weather.

Stuart K. Gardiner, Ph.D., from the Devers Eye Institute in Portland, Ore. and colleagues studied the trends and found a five times larger effect size in the northern states (e.g., Michigan and Minnesota) compared to the southern states (e.g., Florida and Texas). This finding is important because

factors not related to glaucoma could potentially be hampering the detection of disease progression and IOP changes.

The researchers analyzed IOP measurements and visual fields for seasonal variation collected from 33,873 visits over a median of 12.5 years made by 1,636 participants of the Ocular Hypertension Treatment Study (OHTS).

The 22 participating clinics in the OHTS study were divided into six similar geographic regions: Atlantic, Central, North, Pacific Northwest, Southeast and West. Regions were determined based on size and timing of seasonal changes

in precipitation, temperature and sunlight hours.

Seasonal variations in IOP were found in all six regions; January and February were the peak months, with the size of variation ranging from 0.14mm Hg to 0.39mm Hg. In five of the six regions, statistically significant seasonal variations in visual sensitivity occurred, ranging from 0.04 dB to 0.13 dB.

Researchers believe the findings of the study will help practitioners better understand the disease and reduce clinical test-retest variability. The next step is determining whether there is a causal relationship with climatic change.

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Resveratrol Restores Lost Vision in Wet AMD

A non-prescription nutraceutical agent might help restore lost vision in patients with neovascular age-related macular degeneration (AMD) who are otherwise unresponsive to anti-VEGF therapy, according to a series of case studies presented at this year's annual ARVO meeting in Ft. Lauderdale, Fla.

The research focuses on an oral nutraceutical, Longevinex (Resveratrol Partners, LLC), which delivers high doses of vitamin D and resveratrol—the powerful antioxidant that is found predominantly in the skins of red grapes and other fruits.

Lead researcher, Stuart Richer, O.D., Ph.D., chief of optometry and director of ocular preventative medicine at James A. Lovell Federal Health Care Center in North Chicago, Ill., and associates selected 17 patients with advanced neovascular AMD who did not respond to or were not candidates for laser treatment or anti-VEGF injection.

In 16 of the 17 patients, Dr. Richer's team noted that scotomas began to disappear, glare recovery time was reduced, and both contrast sensitivity and visual acuity improved within three to six weeks of starting Longevinex therapy.

“With our instruments, we documented a more youthful appearance of retinal tissues as well



Photo: National Eye Institute, National Institutes of Health

High supplemental doses of vitamin D and resveratrol could help restore lost vision in patients with neovascular AMD, like this individual.

as improved underlying circulation. There were also other improvements in health observed or measured outside of the eyes that were unanticipated,” Dr. Richer said. “This oral nutraceutical taps into the newly appreciated science of epigenetics, where gene protein-making switches are favorably turns on and off, and suggests that age-related eye problems may not be inevitably progressive and biological age is not necessarily cast in stone.”

Because the nutraceutical is not subject to FDA approval and may be purchased by anyone, Dr. Richer cautions that patients should discuss the potential benefits and risks of Longevinex use with their eye care providers before initiating therapy. ■

Richer SP, Stiles WR, Ulanski L 2nd, Thomas C. Observation of human retinal remodeling in octogenarians with resveratrol+. Poster presented at the Annual Association for Research in Vision & Ophthalmology meeting, Ft. Lauderdale, Fla. May 6-10, 2012.

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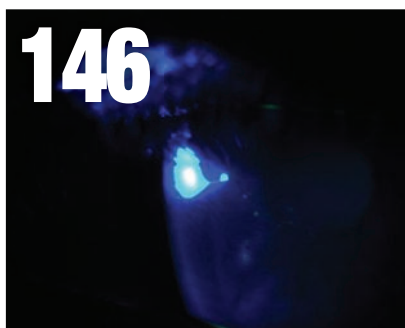
This patient presented with decreased vision in his right eye after waking up from an invasive surgical procedure that involved severe blood loss.

By Trina C. Perkins, O.D., and Susannah B. Marcus-Freeman, O.D.

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TobraDex® ST

(tobramycin/dexamethasone
ophthalmic suspension)
0.3%/0.05%



Indications and Usage: For steroid-responsive inflammatory ocular conditions for which a corticosteroid is indicated and where superficial bacterial ocular infection or a risk of bacterial ocular infection exists.

Ocular steroids are indicated in inflammatory conditions of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe where the inherent risk of steroid use in certain infective conjunctivitis 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

is active against the following common bacterial eye pathogens: Staphylococci, including *S. aureus* and *S. epidermidis* (coagulase-positive and coagulase-negative), including penicillin-resistant isolates. Streptococci, including some Group A and other 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* isolates, *Haemophilus influenzae*, *H. aegyptius*, *Moraxella lacunata*, *Acinetobacter calcoaceticus* and some *Neisseria* species.

Important Safety Information

Contraindications:

- TOBRADEX® ST Suspension, 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.

- Hypersensitivity to any components of the medication

Warnings & 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. If used for 10 days or longer, IOP should be monitored.
- Sensitivity to topically applied aminoglycosides may occur
- Cataracts—use of corticosteroids may result in posterior subcapsular cataract formation
- Delayed healing—the use of steroids after cataract surgery may delay healing and

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- Treats the signs and symptoms associated with acute blepharitis^{1,2}
- Delivers equivalent levels of dexamethasone to the anterior chamber with one-half of the concentration (0.05%) relative to TOBRADEX® (tobramycin/dexamethasone ophthalmic suspension) 0.3%/0.1%^{1,2}

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, steroids may mask infection or enhance existing infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated
- 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
- If product is used in combination with systemic aminoglycoside antibiotics the patient should be monitored for total serum concentration of tobramycin

Please see prescribing information on adjacent page.



TobraDex® ST

(tobramycin/dexamethasone
ophthalmic suspension)
0.3%/0.05%

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use TOBRADEX® ST ophthalmic suspension safely and effectively. See full prescribing information for TOBRADEX® ST.

TOBRADEX® ST (tobramycin / dexamethasone ophthalmic suspension) 0.3%/0.05% Initial U.S. Approval: 1988

INDICATIONS AND USAGE

TOBRADEX® ST is a topical antibiotic 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

DOSAGE AND ADMINISTRATION

- Instill one drop into the conjunctival sac(s) every 4 to 6 hours. (2.1)
- During the initial 24 to 48 hours, dosage may be increased to one drop every 2 hours. (2.1)
- Frequency should be decreased gradually as warranted by improvement in clinical signs, but care should be taken not to discontinue therapy prematurely. (2.1)

DOSAGE FORMS AND STRENGTHS

TOBRADEX® ST ophthalmic suspension contains 3 mg/mL tobramycin and 0.5 mg/mL dexamethasone.

CONTRAINDICATIONS

- TOBRADEX® ST, 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. (4.1)
- Hypersensitivity to any component of the medication (4.2)

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. If this product is used for 10 days or longer, IOP should be monitored. (5.1)
- Sensitivity to topically applied aminoglycosides may occur. (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 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, 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. (5.7)
- If product is used in combination with systemic aminoglycoside antibiotics the patient should be monitored for total serum concentration of tobramycin. (5.8)

ADVERSE REACTIONS

Most common adverse reactions to topical ocular tobramycin are hypersensitivity and localized ocular toxicity, including eye pain, eyelids pruritus, eyelid edema, and conjunctival hyperemia. The reactions due to the steroid component are increases in intraocular pressure with possible development of glaucoma.

To report SUSPECTED ADVERSE REACTIONS, contact Alcon Laboratories, Inc. at 1-800-757-9195 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

Revised: February 2009

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Infiltrative Keratitis and Gram-Negative Bacterial Resistance to PQ-Aldox Lens Care Products

The rate of infiltrative keratitis especially with daily wear silicone hydrogel lenses has been reported with greater frequency.¹⁻⁴ Infiltrative keratitis is associated with several factors¹⁻⁸ including lens care solutions,^{9,10} lens type,¹³ smoking,⁵ and bacterial bioburden.⁵⁻⁸ **Contact lens associated infiltrative keratitis (CLAIK) has been reported at higher rates in particular with polyquaternium (PQ)-Aldox (myristamidopropyl dimethylamine) based Multi-Purpose Solutions (MPS).**^{1-5,9}

Notably, CLAIK has repeatedly been associated with one PQ-Aldox MPS, Opti-Free RepleniSH.^{1-4,11} In one report this solution was being used in 71% of CLAIK cases.³ In another recent study, sponsored by Alcon, **it was reported that patients using Opti-Free RepleniSH were 63% more likely to develop symptomatic corneal infiltrates**, in a univariate analysis, compared to the other 50 possible lens and lens care brand combinations evaluated.¹¹ **Importantly, there has been no demonstrated correlation between transient, solution related corneal staining and inflammatory keratitis.**¹²

Low levels of lens case contamination may occur with any MPS or peroxide system in asymptomatic patients, but gram-negative contamination was reported highest with Opti-Free RepleniSH.¹³ Recent scientific findings in patients using lens care solutions with CLAIK, demonstrate case contamination with certain gram-negative clinical isolates, the predominant species being *Stenotrophomonas maltophilia* and *Achromobacter*.¹⁴ These gram-negative bacteria have also been cultured in the lens cases of patients using PQ-Aldox MPS.^{13,15} **Additional research has shown that these clinical isolates are resistant to a PQ-Aldox MPS and can re-grow during storage in PQ-Aldox MPS in as few as 6 days.**¹⁵⁻¹⁷ Non-Aldox PQ-based MPS, such as those containing PHMB, and peroxide lens care solutions have demonstrated excellent biocidal efficacy against these same clinical isolates.¹⁵⁻¹⁸ Table 1 presents biocidal efficacy against clinical isolates of *Achromobacter* and *Stenotrophomonas* when exposed to PQ-Aldox MPSs and a PHMB-PQ MPS.¹⁹ Lens care solutions

that are ineffective against these clinical isolates may be prone to case contamination and CLAIK may result directly from these bacteria and/or their endotoxins being repeatedly exposed to the ocular surface.¹⁵

Log unit reduction		
	<i>Achromobacter</i> *	<i>Stenotrophomonas</i> *
Biotrue MPS (PHMB-PQ)	2.9	3.5
OPTI-FREE PureMoist (PQ-Aldox)	0.1	1.2
OPTI-FREE RepleniSH (PQ-Aldox)	0.0	1.3
OPTI-FREE Express (PQ-Aldox)	0.2	1.2

Table 1. MPS Biocidal Efficacy Against *Achromobacter* and *Stenotrophomonas* Clinical Isolates Associated with CLAIK¹⁹

Further investigation is warranted to understand the association between infiltrative keratitis events and the use of PQ-Aldox MPS products. **The inefficacy of PQ-Aldox MPS against clinical isolates cultured from CLAIK patients should be considered by eye care practitioners in recommending lens care systems for their patients.**

CLAIK has the potential of creating a significant economic burden on patients²⁰ and may contribute to patients choosing to stop wearing lenses. **Switching patients to MPS products with broad antimicrobial efficacy and proven biocompatibility, along with recommending appropriate lens and lens case cleaning regimens,⁶ may help to prevent CLAIK, minimize risk for future recurrence²¹ or contact lens drop out.**

Biotrue™ MPS from Bausch + Lomb has proven biocompatibility and also demonstrates excellent disinfection efficacy compared to competitive multi-purpose solutions,^{22,23} even against clinical isolates such as *Stenotrophomonas* and *Achromobacter*, which are known to be associated with corneal infiltrative keratitis.

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23. Results of in vitro study following FDA/ISO stand-alone procedure for disinfecting products. Tests against all solutions were modified with organic soil to create a more rigorous test condition. Primary criteria for effective disinfection are defined as a reduction in the number of bacteria by a minimum of 3 logs (99.9%) and a reduction of mold and yeast by a minimum of 1 log (90%) within the recommended disinfection time.

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HL 5784 SL 6999

Our Own Greatest Enemy

My compliments to Drs. Sowka and Kabat on their comments regarding therapeutic practice (“*Build a Therapeutic Practice*,” May 2012). I have often argued that O.D.s can be their own greatest enemy, creating their own mysterious brand of legal guidelines and clinical protocols based on who knows what! This one reminds me of an old favorite: “If I don’t dilate, I won’t be responsible for the periphery.” While I often do not know whether to laugh or cry about these matters, I do know that you guys need to stick with it. Great job!

—Elliot M. Kirstein, O.D.
Cincinnati, Ohio

Mazel Tov, Monty!

I had to comment on Monty Vickers’s exceptional tribute to Irv Borish (“*Borish Built a Better O.D.*,” April 2012). It ranks as one of his finest “Chair-side” columns ever, and captures the essence of this iconic personality in a way nobody else has even approached. Congratulations Monty (or as Irv would have exclaimed, “Mazel tov!”), and I also fondly recall meeting your grandmom Mimi many years ago. I can certainly imagine that comment on the Bible.

—Marc S. Hecker, O.D.
Latham, N.Y.

Be Careful What You Wish For

The debate over including or excluding stand-alone vision plans, as part of the upcoming establishing of state insurance exchanges, is a very important one. The success or failure of many of our practices may ultimately hinge on the outcome of that policy decision. It was with great interest that I read the letter from VSP board director Dr. Tim Jankowski (“*Letters to the Editor*,” February 2012).

Dr. Jankowski expresses several concerns over the possibility of VSP and other stand-alone plans being excluded from the exchanges. He laments the possibility of being discriminated against, compared to medical doctors, and not being allowed to be on the provider panels just because of the way they are set up, thus losing access to caring for his patients. He wants a guarantee of being included on the plan panels. He “can’t risk losing those patients.”

Well, Doctor, welcome to the club. We VSP-excluded doctors are used to it. How ironic that he and

For Dr. Jankowski and his VSP colleagues to portray themselves in this light as being discriminated against and potentially losing business is the ultimate display of hypocrisy.

other VSP policy makers should be in this precarious position while all along they have been perpetrating the worst kind of discrimination—discrimination against their own based only on the doctor’s mode of practice. Now they can appreciate how we excluded doctors feel and are affected when an employer changes vision plans and we are then arbitrarily prohibited from caring for our patients due to VSP’s prejudicial anti-competitive policies enforced under the guise of “private” O.D.s providing better patient care. More likely they are concerned that the VSP member would prefer to be serviced weeknights or Saturday afternoon, or even Sunday when the private doc is closed. For Dr. Jankowski and his VSP colleagues to portray themselves in this light as being discriminated against and potentially losing business is the ultimate display of hypocrisy.

Furthermore, I disagree with his other main argument. Most of the medical plans will likely have associated well vision plans with such plans as Davis, OptumHealth, OptiCare, etc.—plans, by the way, that do not discriminate against providers. As long as these well vision plans are available, there is no reason to contend that usage will drop off.

I do agree with Dr. Jankowski that the possibilities of O.D.s being excluded from the medical panels are real and potentially extremely damaging. He makes reference to the Harkin Amendment. He again laments at the fact that, while apparently providing for parity amongst different categories of health care providers, it does not require a plan to contract with every provider, therefore potentially leaving him locked out of a plan. Does that sound familiar?

I would encourage Dr. Jankowski and any other VSP board member or provider to lobby for the Harkin Amendment to be changed to address his concern. If that could be done, I will be the first to offer my sincere appreciation for the effort that would then also result in myself and the thousands of other excluded VSP doctors to then be required to be allowed on the VSP panel. Beware what you wish for. You might get it. ■

—Name withheld by request



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What Makes a Great Doctor?

Patients may feel safer selecting a know-it-all with a lengthy CV, but your studies and achievements will only go so far in providing truly excellent care.

By Amy Hellem, Editor-in-Chief

Clinical science is a cornerstone of eye care, but by no means is it more important than a thoughtful understanding of your patients' conditions. Empathy is king in optometric care because you are at the forefront of primary care; you are often the one to diagnose and follow patients. While patients may demand a surgeon who graduated from the highest-ranking medical school or an M.D. with the most accolades, they need something different from their O.D.

Surgery doesn't require a relationship in the same way that primary care and comanagement do. Optometry is altogether different in

that way. As optometrists, you are charged with guiding your patients through often difficult and emotional journeys. Sure, they need you to be well educated and current; but practically speaking, what will really stand out is how personable, attentive and considerate you are, particularly when you are delivering bad news.

In this year's annual Retina Report, you will meet Dottie Copoulos, a 79-year-old former geriatric nurse from Peabody, Mass. (see "Through the Eyes of a Patient," page 40). Ten years ago, Mrs. Copoulos was informed that, like her mother before her, she had age-related macular degeneration.

Having witnessed her mom's struggles, Mrs. Copoulos knew she had the disease too, so the actual diagnosis didn't come as a surprise. What she didn't expect, however, was the manner in which her ophthalmologist delivered this bad news. I won't spoil it for you by repeating it here—you can check out the full story for that—but I will say that her doctor was extremely callous and turned bad news into an unforgettable, devastating blow.

"Through the Eyes of a Patient" is an important story that is worth reading. While on one hand, eye care practitioners know more about vision loss than anyone else, they often get so wrapped up in the science that they forget to consider what the patient is experiencing from a real-world perspective. For

the patient, it's not just another exam. Patients are psychologically and emotionally invested in their ocular health and they want to believe that you are too, which is why your approach is so important—even with less than pleasant patients.

Also in this article, Marc Gannon, O.D., founder and director of the Low Vision Institute in Fort Lauderdale, Fla., offers tips on how to appropriately inform patients of bad news (see page 41). The bottom line: You want the patient to leave with the perception that you are their advocate, not their adversary.

Clinical knowledge is important and can't be underestimated. But, it's not everything.

Patient education, proper communication and empathy are critical—especially when you are managing vision loss. In these cases especially, striving to relate to the patient and see things from his or her point-of-view can even trump clinical science. ■

Can't wait till next month to hear more from *Review*?

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on recent articles and news with colleagues, as well as stay up-to-date on some of eye care's more off-the-wall tidbits that are floating around in the social media space.

Amy Hellem
Editor-in-Chief

VARILUX PHYSIO ENHANCED™ LENS TECHNOLOGY—

W.A.V.E. TECHNOLOGY 2™ RECOGNIZED BY THE AAO FOR IMPROVED LOW-LIGHT VISION

Varilux® is the only spectacle lens brand to have its technology recognized by the AAO, not once but twice

At the annual meeting of the American Academy of Ophthalmology in October 2010, Dr. Marguerite McDonald presented a poster on Varilux Physio Enhanced™ lenses with W.A.V.E. Technology 2™—a progressive addition lens (PAL) design that incorporates pupil size modeling data for improved low-light vision.

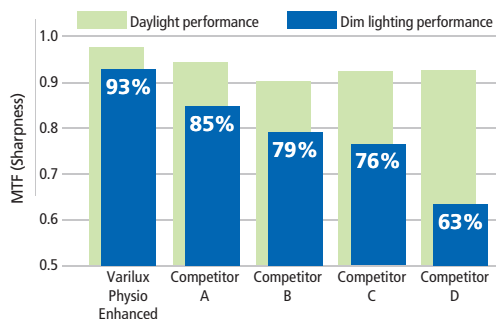
Statistically Significant Preference for PAL that Uses Pupil Size Data to Optimize Wavefront

PAL wearers face a common problem: decreased acuity in dim lighting conditions. Subjects in a double-masked, randomized, non-dispensing wearer test preferred Varilux Physio Enhanced lenses in both standard (71%) and dim (82%) lighting conditions.

Wearer Studies Confirm Optical Bench Test Findings

These wearer-comparison outcomes corroborate optical bench test results that showed the Varilux Physio Enhanced lens exhibited reduced wavefront aberration levels and improved contrast function when compared to four other progressive lenses of identical prescription and material.

Comparison of Visual Sharpness in Low-light vs Bright-light Conditions

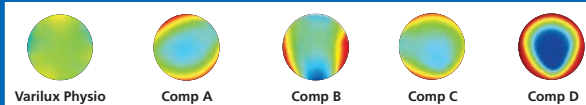


The Varilux Physio Enhanced lens maintained better contrast sensitivity in both low-light and bright-light conditions as indicated by a higher modulation transfer function (MTF).

These tests showed Varilux Physio Enhanced offered:

- The lowest level of aberration in the portion of the lens utilized
- The highest modulation transfer function in dim and standard lighting

Comparison of Wavefront Aberration Levels



The Varilux Physio Enhanced lens exhibited lower aberration levels than designs that do not account for the effect of pupil size variation on progressive lens performance.

Conclusions

Cumulative results from a double-masked wearer study and optical bench testing indicate that the Varilux Physio Enhanced lens, which is designed to account for the natural changes in pupil size that occur in response to lighting levels and the individual's accommodative state, provides improved vision in low-light conditions when compared to progressive lenses that do not incorporate pupil size modeling data into their design.

The data show that the Varilux Physio Enhanced lens:

- Is globally preferred, with preference achieving statistical significance in dim light situations
- Has fewer aberrations than other progressive designs tested
- Preserves 93% of image sharpness in dim light for improved contrast sensitivity

To read the complete poster, please go to www.variluxusa.com

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Got Any Plans for Summer?

No, I'm not talking about vision plans vs. medical plans. I'm talking about pools, BBQs, mosquitoes and sweaty underpants. **By Montgomery Vickers, O.D.**

Ah, summer! I love summer and all that it brings. This is the time for relaxing, for sunglasses and contact lenses, for itching and burning, for generic vs. brand names, for ABO vs. AOS, for Liberals vs. Conservatives, for global warming vs. polar bears, for gin vs. tonic.

What should the slightly-above-average optometric physician do to make summer even more exciting and fruitful? Well, if you follow these 12 easy steps, your summer will be as lovely as mine:

1. Take Mom to lunch. My own mother, still playing tennis at age 88, took care of her mother, who lived to age 99 and 11 months. Daily, Mom made sure that Mimi, as we called her, was happy and fed. On Sundays, we would all get up and walk to church. (Yes, walk! Yes, church!) And after church, my Mom's sister would take Mimi for lunch. We would walk home and find Mimi on the porch. In the time it took for us to tell the preacher he did a good job and walk home, Mimi's weekly lunch with Aunt Jo would be over and Mimi would be waiting on the porch. Then, the whole next week, as Mom served breakfast, lunch and dinner, all Mimi would talk about is that one super speedy lunch. That's why, come Hades or high water, I make sure I take Mom to lunch once every summer...to give her something to talk about for the rest of the year.

2. Buy new technology. This goes without saying, doctors!

Advances in medical technology are astounding us all, so we owe it to our patients to invest wisely in new technologies. That's why I just bought an electric banjo. I'm almost certain I am the only optometrist in my town with a new electric banjo.

3. Write a love letter to your most significant other. My accountant was so pleased when he got his!

4. Take a walk. Get outside. Unlimber those limbs. I did. That's why I have an appointment with my orthopedic surgeon next month.

5. Ride a bike to work. See you at the orthopedic surgeon's office next month.

6. Eat MUFAs. Be careful who you tell this to, as only Dr. Oz knows what this stands for and he might even punch you out if you do not enunciate it properly.

7. Take Fridays off. Yes, *all* Fridays. You mean you still see patients on Fridays? Gosh, I thought I was being facetious.

8. Write a letter to patients who haven't been in for 10 years. Inform them you are sorry they were not there when all active patients received gold watches. There's always next year....

9. Eat more of the wonderful fresh and healthy foods that are only available in summer, like corn on the cob, fresh berries, and three-patty bacon cheeseburgers.

10. Get your eyes examined. This does not mean "Physician, Refract Thyself." Call a trusted colleague, set up an appointment, have pre-testing, get in the chair, and steal eyedrop samples while you are dilating. (Tip: Bring your own dilating drops—he'll give you 12 drops of atropine so you can't work for two weeks.)

11. Arrange for all of your frame reps to come simultaneously for a Trunk Sale and schedule it to be held at your "trusted" colleague's office at 1 p.m. on a Tuesday.

12. Lend your beach house to an optometrist from St. Albans, West Virginia, whose first name is not Tom, John or Nathan. Make your summer special! ■

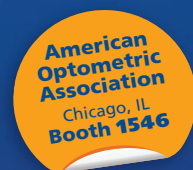




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The Calm Before the Storm

Currently, we're enjoying a lull in the stormy economy. But now is also the time to prepare. **By John Rumpakis, O.D., M.B.A., Clinical Coding Editor**

It's Sunday morning and I find myself sitting in an airline lounge at La Guardia Airport. I'm looking out at the vista of the Manhattan skyline after a glorious weekend in New York City. As crazy as it may sound, I'm struck by the calm... The city was full of life, as always, but calm. The weather was uncharacteristically calm—mid-70s and no humidity. All would appear to be blue skies and smooth sailing ahead...

But then, I look to the forecast—tropical storms approaching, high winds and heavy thunderstorms coming.

As metaphoric as it may be, that too is the state of optometry right now. People are uncharacteristically calm. After a couple of very hard years, business sentiment is on the upswing after what were very good first and second quarters for most of the O.D.s I have spoken to.

Dark Skies Ahead

Unfortunately, as business gets better, complacency gets worse. I have been the beneficiary of many e-mails over the past few years from you, thanking me for helping to introduce the benefits of medical eye care and proper medical coding to your practices. Some of you have gone so far as to say that, if it wasn't for doing this, your practices may not have survived the recent recessionary cycle.

While I believe that the optometric practice of today (and of the future) incorporates "total patient



care," and not just refractive or medical eye care, we need to stay vigilant about what else the future is going to bring. (Notice that I said "is" and not "may.")

Things are always calmest before the storm. This month, the Supreme Court will decide the fate of health care reform. But make no mistake about it—no matter what the court may decide, the behaviors, policies and attitudes that have formed over the last couple of years are here to stay.

Anticipate an Audit

Third-party audits of your practice are going to happen. It is not a question of *if*, but *when*; and you need to be prepared. In recent announcements, about 10 contractors for the Centers for Medicare & Medicaid Services (CMS) have indicated that they will be stepping up their audits—Medicare Comprehensive Error Rate Testing (CERT) audits and Medicare Recovery Audit Contractor (RAC) audits.

A couple of CMS contractors have specifically targeted outpatient-based ophthalmic claims; more specifically, they're targeting 920XX office visits as well as

many of the procedures that we perform on a daily basis, such as fundus photography, anterior segment photography and visual fields. The documentation of these services is of concern.

It has to be complete, must clearly demonstrate the medical necessity of the procedure performed, and should fall within the commonly accepted standards of practice.

CMS and other medical carriers are not the only entities that have found that conducting audits have improved their fiscal bottom line. Refractive carriers are also increasing the number of audits of optometric providers.

And the economics of these situations are significant. It's not unusual for an optometric audit by a refractive or medical carrier to easily be in the hundreds of thousands of dollars.

So, don't be lulled into complacency by the relative, recent calm within our profession and industry. Yes, things in the economy are slowly getting better, but the winds of change are still blowing strong and threaten our viability in ways we have yet to fully understand. ■

Please send your comments to CodingAbstract@gmail.com.

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References: **1.** Based on a post-launch evaluation in which 88 eye care practitioners refit over 400 patients in AIR OPTIX® AQUA contact lenses. Alcon data on file, 2011. **2.** Nash W, Gabriel M, Mowrey-McKee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci.* 2010;87:E-abstract 105110. **3.** Compared to HEMA contact lenses; based on the ratio of lens oxygen transmissibilities; Alcon data on file, 2010. **4.** Dumbleton K, Richter D, Woods C, et al. Compliance with contact lens replacement in Canada and the United States. *Optom Vis Sci.* 2010;87(2):131-139. **5.** Compared to 2-week replacement lenses; based on self-reported lens replacement time and third-party industry pricing information; Alcon data on file, 2012.

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MGD:

Getting to the Root Cause of Dry Eye

Eye care professionals are shifting their focus to the role of the meibomian glands in dry eye, and away from traditional inflammation-driven models of disease etiology.

By **Caroline A. Blackie, O.D. Ph.D., and Donald R. Korb, O.D.**

Meibomian gland dysfunction (MGD) is now considered the leading cause of dry eye.¹ This is according to the published findings of the prestigious 2011 International Workshop on MGD.¹ Conceptually, this is a substantial shift in our thinking, leading us away from the traditional inflammation-driven models of dry eye disease etiology. A review of the literature reveals that our unwavering focus on inflammation as the driving force behind dry eye, in retrospect, has been a distraction for some time.

While identifying and managing ocular surface inflammation is critical, we will never fully understand dry eye if we remain fixated on treating inflammation alone. If we turn our attention to what is causing the inflammation, we can open our minds to the recent paradigm shift—that MGD is currently thought to be the leading cause of dry eye.¹ With this new foundational thinking, our approach to diagnosis and management must necessarily undergo



The Meibomian Gland Evaluator (MGE, TearScience Inc.) in use during the diagnostic evaluation of habitual meibomian gland function.

a makeover. To begin, we will review MGD and how to evaluate meibomian gland health.

Changes in Diagnosis

The historical and narrow concept of MGD as a hypersecretory disorder with obvious signs

of infection and inflammation has been expanded to include the much more commonly encountered obstructive hyposecretory and often non-obvious form of MGD. This form of MGD results in inadequate lipid production for the formation of the oily layer of

the tears.¹ Obstructive MGD is the most common form of MGD resulting in dry eye.²

In the recent past, a clinical diagnosis of MGD has been based primarily on overt signs of morphological changes of the lid margins and meibomian gland orifices. For example, these include pouting (an elevated internal plug of solidified secretions, which may be expressed from the orifice with pressure); capping (a dome over the orifices possibly of solidified oil that may or may not be epithelialized); reduction in number of the orifices and loss of definition of the orifice cuffs; erythematous, irregular, thickened lid margins, with or without telangiectasia surrounding orifices; and serration of the lid margin.³

Conversely, non-obvious and early obstructive MGD presents a more tricky diagnosis, given that very few or even none of the aforementioned obvious clinical signs are observable with a slit lamp. We now believe that there is only one way to diagnose MGD under these circumstances, and that is to test the functionality of the meibomian glands directly.⁴

If the glands are not functional and the patient is symptomatic, regardless of the appearance of the lids and ocular surface, this should direct our treatment towards rehabilitating the glands. This holds true even in the relatively rare case of dry eye that may have originated as true aqueous deficient dry eye (ADDE).

Meibomian Gland Functionality

What is the best way to assess meibomian gland function? The brute-force digital methods of the past have been effective at diagnosing the old medical model



The LipiFlow (TearScience Inc.) during a 12-minute, in-office dry eye treatment. It simultaneously applies heat and pulsatile pressure to the eyelids.

form of infective MGD, where the glands are full of turbid thick secretions that can only be expressed with force. There are many ways to grade these secretions and, while important, they do little to inform us of the habitual function of the glands on a daily basis. What we really want to know is whether the glands release functional oil during everyday blinking.

A recent innovation by TearScience Inc. known as the Meibomian Gland Evaluator now offers a metric to standardize the amount of force applied to the glands during diagnostic testing and to mimic the force experienced by the glands during a deliberate blink.⁵ Because this force is much less than the force of the typical digital manipulation of the glands used in the past, the amounts of oil that are expressed are very small and require careful inspection.

Once we have gained some knowledge of the patient's meibomian gland function through evaluation and other more familiar testing (e.g., tear film break-up time, symptom report, ocular surface staining/staining the lid wiper, Schirmer testing, etc.),

we can proceed with a treatment plan. From the work of Anthony Bron, M.D., F.R.C.Ophth., we know that while dry eye can be academically classified as ADDE or evaporative dry eye (EDE) in nature, both forms become virtually indistinguishable as the disease progresses, rendering attempts to make the distinction fairly meaningless.⁶

Even if we are able to clearly and predictably distinguish ADDE from EDE, dry eye presents such a clinical conundrum with tremendously high treatment failure that the accepted clinical approach to therapy remains one of "use whatever works." Options are: tear replacement or supplementation, ocular lubricants, anti-inflammatory therapy, immune modulators, lid therapy, environmental adjustments (including humidity goggles) and meibomian gland rehabilitation. All of these are intended to improve, maintain or protect the health of the ocular surface, regardless of the disease etiology.

So, which treatment path is likely to give us the highest probability of short- and long-term success? Because MGD currently is understood to be the leading

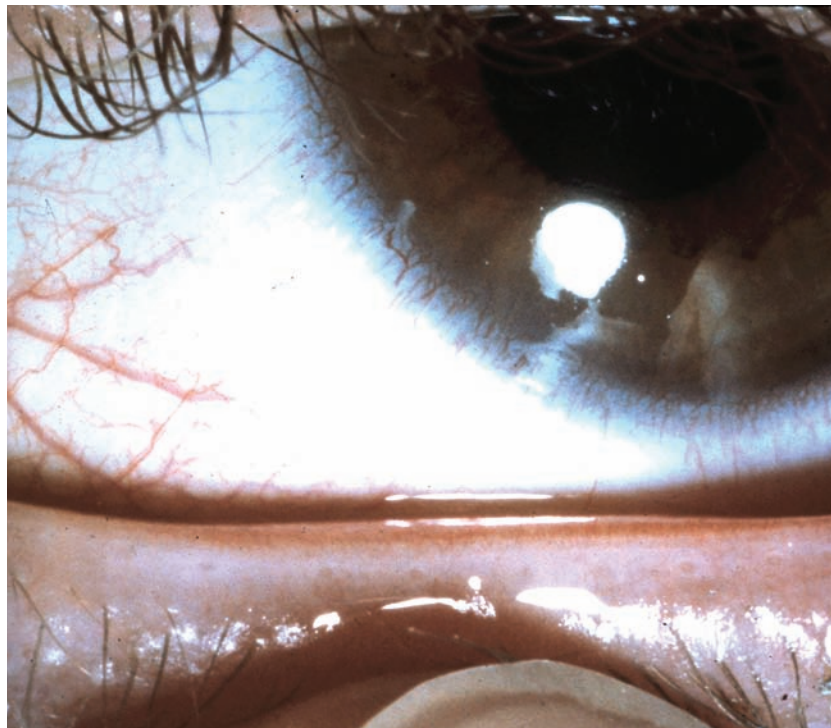
cause of dry eye, it makes sense to start there; but all categories of treatments will be mentioned as they all, potentially, serve a role in our clinics.

Treatment for MGD and Dry Eye

Treatment of MGD is designed to restore the normal flow of meibomian gland secretions, thereby increasing the likelihood of a healthy lipid layer and consequently reducing tear evaporation and enhancing tear film stability. This outcome is achieved primarily through removal of material obstructing the ducts of the glands. The four main treatment approaches for MGD are as follows: 1) physical expression to remove obstruction material; 2) application of heat to the eyelids to soften and preferably liquefy solidified and obstructive meibomian gland contents; 3) lid scrubs to relieve external meibomian gland orifice blockage due to epithelial overgrowth; and 4) medications to mitigate infection and inflammation of the eyelids and also to possibly improve the lipid profile of the glands.

Let's look at each treatment approach in turn:

• **Physical expression.** Physical expression of meibomian glands for therapeutic purposes is an in-office procedure that has at least an 80-year history and can be supplemented by the patient performing self-expression at home.⁷ The reported techniques vary from gently massaging the lids against the eyeball to forceful squeezing of the lids either against each other or between a rigid object on the inner lid surface and a finger, thumb or rigid object (glass rod, cotton swab, metal paddle, etc.) on the outer lid



A normal healthy eyelid. Upon gentle pressure over the glands, the gland orifices release clear liquid oil.

surface.⁸ The purpose of the rigid object on the inner lid surface is to protect the eyeball from forces transferred through the eyelid, and also to offer stable resistance to increase the amount of force that can be applied to the glands.

The amount of force needed to express obstructed glands can be significant. Unfortunately, the pain frequently is also significant and typically this is the limiting factor in the overall efficacy of the technique.⁸ A recent study reported that just 7% of the patients could tolerate the pain resulting from adequate therapeutic physical expression of the glands.⁸

Regardless of the method of meibomian gland physical expression, the goal is to evacuate the obstruction and other meibomian gland material from the gland, thereby creating a new environment that offers the potential for

normal gland secretion. If physical expression is a viable option, it should be continued until the dysfunction is resolved.⁷

• **Heat therapy.** The application of heat to the eyelids has traditionally been implemented by using warm compresses. However, many innovative devices have emerged with the goal of providing a regulated elevated temperature to the eyelids. These tools, in the forms of a face-mask, goggle or similar device, have been designed to more effectively heat the lids and increase convenience for the patient, possibly improving compliance.^{9,10}

These devices are not subject to FDA approval and typically are home therapies that patients would purchase for themselves. While difficult to manage from a compliance and safety perspective, the at-home therapies can be effective

Upgrading Your Contact Lens Patients



By Douglas C. Clark, O.D.

CHANGE. IT'S A DREADED WORD IN SO MANY optometric offices. Some patients, staff and colleagues do everything they can to resist it. Implementing change does take time and effort, but adopting new technology is undeniably beneficial for our patients and our practices.

Change is Good

I am still amazed at the number of new contact lens patients who come into my office wearing HEMA soft contact lenses. We're not offering our patients the best possible care if we continue to fit them in outdated materials. Optometrists who don't stay current with the latest products and technologies risk being perceived as outdated by patients, colleagues and worse yet—former patients.

Our practice has been very successful in transitioning patients from their older HEMA contact lenses into Alcon's AIR OPTIX® AQUA contact lenses. It takes only a few moments of discussing the improved oxygen transmission and longer wearing times of this silicone hydrogel lens to get patients to admit to limited wearing time and reduced comfort with their current HEMA lenses. My technicians are trained to have already started this discussion once they see a patient wearing older contact lens materials. Remind techs to keep their explanations short, with emphasis on patient benefits.

If a patient expresses any resistance to change, expect some objections. Many times, the objection is related to finances and occasionally, the patient will have had a previous bad experience with changing product several years ago. You may have difficulty changing all of these patients, but if you ask the right questions, you will find that they aren't as fond of their old lenses as they initially professed to be. Use this as an opportunity to educate them on the benefits of changing.

Making the Right Choice

Many good lenses exist from which we can choose to fit our patients, but certain characteristics are more important than others when deciding which silicone hydrogel contact lens is most suitable for patients. For example, deposit resistance, moisture retention, wettability and breathability are all features that I pay attention to in a lens, and AIR OPTIX® AQUA contact lenses perform well along these dimensions.

Thanks to a plasma surface-treatment, AIR OPTIX® AQUA contact lenses resist deposits better than other major SiHy contact lens brands.¹ And the engineering has helped to minimize the rate of lens dehydration,² for comfort all day long.

The lens's high oxygen transmission reduces the possibility of neovascularization, which patients recognize as whiter,

healthier eyes, while practitioners appreciate the knowledge that the lens will be less likely to result in hypoxia. This is especially comforting, seeing that no matter what patients tell you—especially young adults—they *will* occasionally sleep in their lenses.*

AIR OPTIX® AQUA contact lenses were also designed to have a wetting angle of only 26°.³ Along with the superior deposit resistance,¹ this feature helps give patients the comfort they demand and expect in a monthly replacement contact lens. AIR OPTIX® AQUA is my lens of choice because I know it will work the first time in 9 out of 10 patients.

Keeping with the Times

The Ford Model T was an excellent car at its time, but you don't see many of them on the road today. If you could drive either a Model T or a tripped out V8, which one would you choose? That said, why would you continue fitting your patients in older technology contact lenses?

So, the next time you walk into the exam room and find that your patient is wearing an antiquated contact lens, take that little bit of time to offer them a superior product, such as AIR OPTIX® AQUA contact lenses. You can be confident that your patients will enjoy the benefits of whiter eyes, improved comfort and crisp, clear vision.⁴ Furthermore, not only will your expertise be elevated in their eyes, but they will also see your office as being on the cutting-edge.

Dr. Clark practices at Pelham Eye Care in Pelham, Ala. He is a past president of the Southern Council of Optometrists, a committee member of the American Optometric Association and a board of trustee member of The Southern College of Optometry.

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*AIR OPTIX® AQUA (lotrafilcon B) contact lenses: High oxygen transmissible lenses. Dk/t = 138 @ -3.00D. Other factors may impact eye health.

Important information for AIR OPTIX® AQUA (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness. Risk of serious eye problems (ie, corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

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A lid with MGD (obvious signs are telangiectasia, lid margin thickening and posterior blepharitis). Diagnosis can be made by observation of the lid margin. Dysfunctional glands release a thick opaque material upon forceful expression.

and should be recommended for patients that are willing and able to use them safely.⁹ It is important to note that the efficiency of all techniques where heat is applied to the outer lid surfaces are limited by the diffusion of heat through the lid tissue and tarsal plate, and also the vascular effect of the blood flow transporting the heat away from the lids.

When we prescribe home therapy in the form of warm compresses or similar heating methods, it is critical to inform the patient not to directly place pressure over the cornea and also to avoid any eye rubbing following

heat therapy. Recent studies have indicated that home heat therapy can raise the corneal temperature. This alone, or in combination with physical manipulation of the cornea through massage or eye rubbing, can lead to transient visual distortions or even keratoconus in apparently normal eyes.^{11,12}

The use of intense pulsed light (IPL) therapy has been suggested as an off-label treatment for MGD. However, there are currently no peer-reviewed studies published on this technique, and therefore little is known about the precise mechanism of action for the treatment of MGD or the safety risks of

repeated treatment. We will know more when safety and efficacy have been investigated.

• *Simultaneous heat and expression.* As of August 2011, there is one FDA-approved treatment for EDE involving the simultaneous application of heat and pulsatile pressure to the eyelids. The treatment device is called the LipiFlow, manufactured by TearScience Inc.¹³ It is being used in academic centers and private practices in the United States, Canada and Europe, and has been shown to improve both meibomian gland functionality and reduce dry eye symptoms in multicenter studies.¹³⁻¹⁵

The LipiFlow treatment is unique and significantly more effective than any other available heating method.¹³ The heat is applied to the inner lid surface directly over the glands, thus avoiding the heat loss incurred with conventional methods as the heat diffuses through the outer lid tissue and the tarsal plate prior to reaching the glands. The lids are sandwiched between the heater on the inner lid surfaces and a pulsatile pressure component on the outer lid surfaces, which allows for expression of the gland contents of upper and lower eyelids simultaneously during the 12-minute heating phase. This is in contrast to the conventional therapeutic gland expression that can only occur after the heating phase. In addition, because the gland expression occurs during heating, there is no pain experienced during the administration of the treatment.^{13,14}

Although there is no single melting point for solidified meibomian secretion because the chemistry and viscosity of the solidified secretion itself are

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variable, we know that solidified secretions from severely obstructed glands have a considerably higher melting point than those from apparently normal, unobstructed glands.^{16,17} So, it can be assumed that higher temperatures, provided safety is maintained, are superior for the treatment of more severely obstructed glands.

• **Lid scrubs.** Lid scrubs, although primarily suggested for anterior blepharitis, can help

remove crusts and inspissated secretions blocking the gland orifices. Lid scrubs are performed on the lid margin, over the meibomian gland orifices to prevent epithelial overgrowth or obstructive material from sealing the orifice. Use of lid scrubs and lid massage also have been shown to improve tear film break-up time, and lid hygiene also helps and is the universal treatment for any associated staphylococcal or seborrheic blepharitis. Therefore, use of lid

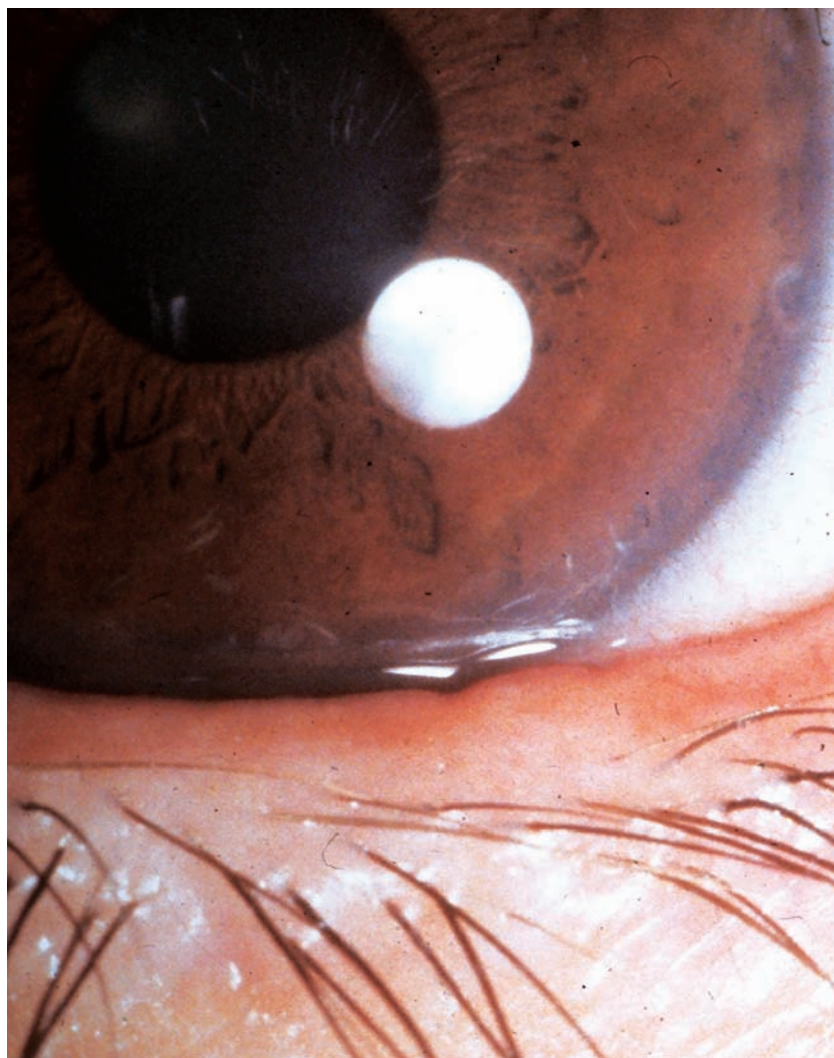
scrubs has been suggested as part of the daily routine of heat therapy and lid massage in patients with MGD.⁹

• **Intraductal probing.** An alternative to the more conventional heat and expression is physical probing of each individual meibomian gland orifice. A single study reported short-term relief of symptoms and reduction in the inflammatory signs of obstructive MGD as a result of intraductal gland probing.¹⁸ Future investigations will expand our knowledge of the safety and efficacy of this treatment in the short and long term.

• **Medication.** Antibiotics—particularly the tetracyclines, including doxycycline, tetracycline and minocycline—continue to find a place in the modern management of MGD. Tetracyclines decrease the secretion of bacterial lipases that are known to break down the normal meibum lipids into inflammatory free fatty acid fragments.¹⁹ In addition, tetracyclines are said to have anti-collagenase and anti-matrix metalloproteinase (MMP) properties.^{20,21}

Success also has been reported with minocycline in decreasing a branch chain fatty acid to its normal values, resulting in an increase in tear film break-up time following two months of minocycline therapy in patients with MGD.²² Other studies have shown the benefits of doxycycline over tetracycline.²³ Although the involved mechanisms may not be fully understood, systemic tetracyclines appear to improve the lipid profile of meibomian gland secretion.⁹

Similar to the anti-inflammatory properties of tetracyclines, macrolides also have anti-inflammatory and anti-MMP activity.²⁴ The topical drug treatment efficacy of azithromycin (1% and



A “quiet” lid margin with MGD. The only visible sign is lid margin serration. The MGD is not obvious. This patient is highly symptomatic with significant obstructive MGD. Diagnosis is made upon meibomian gland expression.

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1.5%) has shown some success in patients with MGD.^{25,26}

While there is evidence that antibiotics may improve patient symptoms and meibomian gland lipid secretion quality, there is no evidence that antibiotics can relieve meibomian gland obstruction. This suggests that the improvements may not be in functional restoration of obstructed meibomian glands, but in better quality of meibomian gland secretions. If the latter is correct, antibiotic therapy may be very beneficial if administered in combination with, or immediately after, treatment to remove the meibomian gland obstruction (physical expression, heat therapy, etc.).

The use of topical cyclosporine, an immunosuppressant, in ADDE (especially with ocular surface inflammation) has been established.²⁷ There is also some supporting evidence for its role in MGD in the form of improvement in objective signs and sometimes, also symptoms.^{27,28} Similar to the use of antibiotics, once gland obstructions have been removed via heat therapy and physical expression, topical cyclosporine may then help alleviate any associated inflammatory components.

The administration of topical corticosteroids to suppress the inflammatory response associated with dry eye has been shown to be effective in the relief of dry eye signs; reduction in ocular surface staining; reduction of lid wiper epitheliopathy and dry eye symptoms.²⁹⁻³² The use of steroids usually is temporary or pulsatile due to the potential risks of chronic use. Additionally, the obstruction would first have to be removed from the glands, in conjunction with steroid use, to increase the likelihood of treatment success.

Topical androgens continue to be investigated for the treatment of dry eye and MGD but further research is required to elucidate their role in the clinic.^{33,34}

• **Diet.** Dietary alteration and supplementation has been gathering support in recent years. Because patients with MGD have an altered lipid composition, changing the dietary lipid intake may affect the lipid composition of the meibomian glands. As such, omega-3 supplements have been recommended as treatment for dry eye and MGD.³⁵ There is also recent evidence that omega-3 consumption may significantly reduce certain conjunctival inflammatory markers.³⁶ Thus, published data provide some evidence to recommend dietary alterations and supplementation of omega-3; however, further research is required to fully quantify or evaluate these effects.⁹

• **Tear supplementation.** Tear supplementation remains a mainstay in the management of dry eye. Tear substitutes perform many important roles, such as improving the optical quality of the tear film, alleviating dry eye symptoms, facilitating adequate spreading of meibomian gland secretions, and increasing lubrication between the eyelid wiper and the ocular surface, to name just few. Not surprisingly, the newer tear substitutes that incorporate lipid emulsions demonstrate significant improvements in tear stability, patient comfort and lipid layer thickness.³⁷⁻³⁹

One can also apply ointment to the lid margins, providing a reservoir of lipid on the eyelid to replenish the tear film lipids upon blinking. The only issue with this is that the ointment typically results in an annoying transient

blurring of vision and is therefore best applied at night.⁴⁰

• **Environmental modifications.** Environmental modifications will have a significant effect on patient symptoms because exposure to conditions of low relative humidity and temperature-controlled heated or air-conditioned environments increase evaporative tear loss.

This aspect of patient education regarding dry eye management is probably underemphasized. The tempering of our living and working conditions, where possible, to increase the relative humidity can dramatically reduce symptoms and the need for tear supplements. The successful use of moisture goggles demonstrate this effect.^{9,41}

In conclusion, the past several years have seen a remarkable paradigm shift in our emphasis from an inflammatory-driven, aqueous deficient model of dry eye to one of lipid-deficient evaporative dry eye as a sequela of MGD. Thus, our focus now targets the eyelids and meibomian glands rather than the lacrimal gland output. Our experience confirms the conclusion of the prestigious MGD workshop that MGD is likely the leading cause of dry eye throughout the world. Emerging therapies for dry eye will further reflect this thinking. In the meantime, we can sharpen our observational skills as they pertain to the eyelids, the lid margins and the meibomian glands themselves. ■

Dr. Blackie is an optometrist in Boston and is the clinical research scientist for TearScience Inc., in Morrisville, N.C. Dr. Korb is in private practice at Korb Associates, Boston, and is the co-founder and chief technical officer of TearScience Inc.

Both authors have disclosed

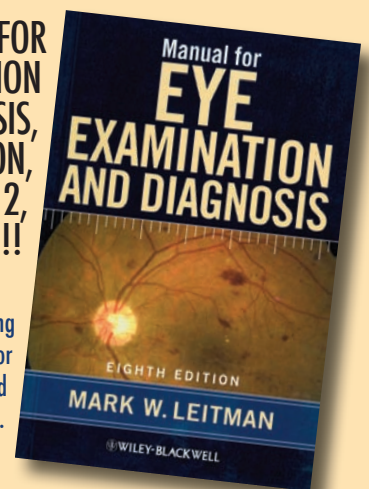
that they receive financial support from Korb Associates and TearScience Inc. However, neither author solicited exposure. Rather, they were invited by Review to discuss this technology based on their experience.

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Through the Eyes of a Patient

Science is key, but don't disregard the importance of bedside manner—especially at the time of diagnosis. **By Jane Cole, Contributing Editor**

In 2002, Dottie Copoulos knew something was wrong. The then 69-year-old former geriatric nurse from Peabody, Mass., recognized that her vision symptoms were getting worse.

She was already aware that she had floaters. But now, when she inspected her flowers from her backyard deck, the avid gardener couldn't distinguish her roses from her clematis plants.

Mrs. Copoulos, whose mother suffered from advanced age-related macular degeneration, realized she was losing her central vision. But she still was not prepared to hear her ophthalmologist's diagnosis as she anxiously sat in his exam chair a decade ago.

"I always liked my doctor. He had good credentials and was considered the best eye doctor in the area," Mrs. Copoulos recalls. "But I will never forget how he told me I had AMD. He said, 'I've got good news and bad news. The bad news is that your eyes progressed to the point that I have to report you to the Division of the Blind and you will be hearing from the DMV.



Photos: Mark Lorenz

In addition to her regular job, Dottie Copoulos wrote a weekly health care column in the local newspaper. Now with advanced AMD, she can't even read the newspaper.

You won't be able to drive anymore, Dottie. But the good news is that you are going to get a rebate on your taxes and you won't have to pay excise tax on your car anymore."

Mrs. Copoulos, a normally coop-

erative and pleasant patient, says this time she erupted.

"I said, 'What are you talking about? You are telling me I can't drive but I won't have to pay excise tax on my car?' He was naming off all the benefits I would receive from

Break Bad News the Right Way

Informing a patient about a devastating eye disease is a delicate but critical factor in the diagnosis.

The best way to break bad news? "Be direct and straightforward," says Marc Gannon, O.D., founder and director of the Low Vision Institute in Fort Lauderdale, Fla.

"First, you need to let the patient know what the condition is," he says. "Second, you need to define and explain the disease. Let them know the ramifications and the actions they can take so they are not left without hope."

Simply giving the patient a brochure to read when they get home is not appropriate, especially since many patients with eye disease have already lost their ability to read.

Also, you obviously don't want to tell a patient that there is nothing that can be done.

"Be honest and give them a good working knowledge on what they can do to preserve their vision and the options available to them," Dr. Gannon adds.

Macular degeneration is the most common cause of vision loss in older adults, and it is important to reinforce to the patient that there is life after the diagnosis, he says.

"I hope by the time they leave the office, the patient is equipped with an understanding of their condition and [the knowledge] that it is not a hopeless situation at all," Dr. Gannon says. "Life isn't over, and they can learn how to compensate in the areas of their lives they wish to preserve."

being blind," she says. "I got so upset, I walked out. My doctor's nurse called me at home later and apologized. She said my doctor just got back in the office after back surgery and was in a lot of pain. I told her that he should have waited another day to come back to work then."

Even though deep down she knew she had AMD, hearing the diagnosis—and the way in which it was delivered—was still jarring.

"It's like hearing you have cancer," she says. "When you hear those words, you're never prepared for anything like that." (See "Break Bad News the Right Way," above.)

Today, Mrs. Copoulos joins the estimated two million Americans over age 50 who have advanced AMD.

And, while science and clinical research continue to make strides in finding better treatments, patients like Mrs. Copoulos learn to adapt and carry on with their day-to-day lives as their vision continues to worsen.

Here, she shares her first-hand experience on what it is like to live with advanced AMD.

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Making Adjustments

For three decades, Mrs. Copoulos ran the nursing divisions of several large Massachusetts convalescent homes. She raised three children, was a regular participant in the Harvard Nurses' Health Study and wrote a weekly health care column, "Ask Dottie," in the local newspaper.

When Mrs. Copoulos did carve out time for herself, she was a voracious reader. Since her AMD has progressed to both eyes, the now 79-year-old Mrs. Copoulos can't read books anymore.

"I go through three books on CD a week. I have a player in my bedroom and a portable one I carry around with me. I just love it," she says.

Mrs. Copoulos tries to compensate for what she has lost due to AMD and keeps a positive attitude. In addition to losing her ability to read, she can't drive anymore and has to rely on others for rides to get around to appointments, lunches or trips to the grocery store. And, seemingly small—but yet once important—routines have also fallen by the wayside.



Dottie Copoulos' husband painted thick white lines along the edges of their outside steps so she can see them better when she goes in and out of their home.

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Mrs. Copoulos has the jars in her spice rack memorized. If someone comes over to lend a hand in the kitchen, the spices always get rearranged. Then she has to figure out which spice is which by using her sense of smell.



“I used to love to put on eye shadow, but now, when I’m through, I look like a clown. And, forget about tweezing,” she says with a laugh. “Things I used to do for personal appearance and normal morning routines have changed for me. But what can you do?”

Mrs. Copoulos admits she loves a good sale, but she can’t go to stores alone anymore. With AMD, numbers on the tags are too hard to read, especially if the price contains any circular number such as a three, a six or an eight.

“With macular degeneration, you lose part of the figure, so the threes look like eights and the sixes look like threes. I have to always have someone with me. You lose your independence,” Mrs. Copoulos explains.

In fact, losing her independence has been the hardest part of coping with AMD, she says. “I hate being dependent on people for certain things. That’s my personality, it might not bother other people as much, but it bothers me. I used to

be able to paint the whole house by myself, but now I can’t do it anymore.”

Mrs. Copoulos has no shortage of friends and family who want to help, but sometimes the help backfires. Because she now has only peripheral vision, Mrs. Copoulos arranges everything in her house precisely so she will know exactly where it is when she needs to use it, including her spices in her spice rack. When someone comes over to visit and insists on lending a hand in the kitchen, the spice rack gets rearranged; this may seem trivial for most, but not for Mrs. Copoulos.

“It’s annoying. I put all the spices I use the most in the front and the ones I use for baking I put separately,” she says. “A lot of my girlfriends and sisters come here and they want to help me but I say, ‘Don’t help me,’ because it just confuses me. I’ve messed up many meals because of my spice rack. If things are where I put them, I do well.”

Making change at the checkout line or leaving tips in restaurants or at the airport are no easy tasks either, so Mrs. Copoulos has to carefully arrange her wallet in denominations. She learned this the hard way years ago after tipping a porter \$50 instead of \$5.

“That was one happy porter,” she says.

Mrs. Copoulos continues to be active, even though she may be moving at a slower pace. She still goes to the movies with friends although now she can only hear the sound, and she has kept her sense of humor intact.

“My husband Alex is legally deaf, and I’m legally blind,” she says. “Alex will be driving us down the highway, and he’ll say ‘I told you to watch for Exit 16 and we just missed it.’ I tell him, ‘If I could see Exit 16, I would be driving.’”

Although her children have encouraged her to move into a one-level house, Mrs. Copoulos is reluctant, as she has 50 years of history and memories in her Pea-

body home. So, she makes do. Mrs. Copoulos stays on one floor of the house so she doesn't need to navigate the stairs, and her husband, Alex, painted a thick white line along the borders of their outside steps so Mrs. Copoulos can see them better when she goes in and out of their home.

"I am afraid to do a lot of walking. If you trip, you can't see where you are falling," Mrs. Copoulos says. "I walk very slowly, and it's not because I'm in pain; it's because of my fear of falling. I hate the white cane. I won't use it yet, so when I walk I always need to have someone with me."

Visual Aids and Studies

When Mrs. Copoulos was first diagnosed with AMD, she says the Division of the Blind was extremely supportive. A social worker paid her a visit at her home and her doctor provided her with visual aids including a large illuminated magnifier and thick telescopic glasses that she used to watch television. But now, as her AMD has progressed, she just listens to the sound coming from the set.

Even before she had floaters, the former geriatric nurse read about the AREDS study and began to take the recommended formulation of vitamins and minerals, knowing her family history of AMD. Now Mrs. Copoulos encourages her own three children to take ocular vitamins in an effort to potentially prevent the onset of vision loss, since studies have indicated AMD can be inherited genetically.

Mrs. Copoulos has also participated in several AMD studies in Boston during the past decade, including a controlled study where she took a pill for one year. "I'm not sure if I was in the placebo group or not, but whichever group I was in, it didn't work. That's life though. As a geriatric nurse, I knew what I was getting into."

Although Mrs. Copoulos's vision continues to decline, she is realistic but still hopeful that one day science will find a cure.

"I read about these new studies and miracles, but those success stories are a very small percent. But, whatever comes around that is new, I am willing to try it," she says.

Mrs. Copoulos has had to adapt her lifestyle to cope with AMD, but she says that she hasn't let it take over her life.

"It takes me longer to do things and I am more cautious," she says. "But, I won't let it stop me from doing anything. It might take me longer, but I will get there." ■

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LVC: No Longer Out of Bounds for Athletes

With today's cutting-edge refractive procedures, more of our athletic patients are now able to enjoy the visual benefits of laser vision correction. **By Derek N. Cunningham, O.D.**

Competitive athletes' visual demands are much more specific than those of our typical patients. Athletes are regularly subjected to extreme heat or cold, rain, wind, snow, perspiration and physical contact—all while traveling at a high rate of speed and/or focusing on rapidly moving objects.

Even more challenging, many athletes are expected to adapt to dynamic lighting conditions seamlessly, as many baseball, football and soccer players start a game in the late afternoon in the direct sunlight and finish playing in artificial light under a nighttime sky. When combined, these factors create endless potential for vision-related problems.

Here, we'll review the primary reasons why your athletic patients likely will ask you if laser vision correction (LVC) is right for them. Additionally, we'll discuss a host of individual pre- and postoperative considerations that you will have to address.

Athletes Want LVC

LVC is becoming increasingly more common in athletes. Many

athletes prefer laser treatments to other forms of vision correction for a host of reasons, including:

- Superior postoperative visual acuity, which is necessary for faster reaction time and optimum depth perception.

- Spectacles and contact lenses (regardless of aspheric design) may reduce contrast sensitivity under different lighting conditions.¹

- Training and competition often takes place in extreme environments. Resultant perspiration and the use of protective headgear can make spectacle wear virtually impossible.

- A greater likelihood for contact lens intolerance caused by a decreased blink rate, disruption of the natural tear film secondary to increased perspiration, and excessive tear evaporation resulting from prolonged exposure to extreme weather conditions.

One question that we are often asked: "Does the type of LVC matter for certain athletes?" Although there is no definitive answer, there are different benefits to certain procedures.

The postoperative differences for various laser procedures range

from safety concerns and postoperative recovery times to in-game performance issues. Contrast sensitivity is one of our most influential postoperative variables, and can be considered a more sensitive assessment of visual function than even visual acuity. For this reason, we place considerable emphasis on the correction of higher-order aberrations (HOA). And, no matter what, we will address each patient's specific visual demands with respect to the sports he or she participates in when determining the optimal refractive procedure.

Primary Considerations for LVC

Without question, eye care clinicians must consider the potential ocular safety (or lack thereof) associated with a particular sport. Obviously, some athletes have a much higher incidence of eye injuries than others. For example, basketball players routinely experience ocular injuries that involve finger- and fingernail-related corneal trauma. This type of injury can become considerably more serious if associated with corneal flap complications. Other athletes, such

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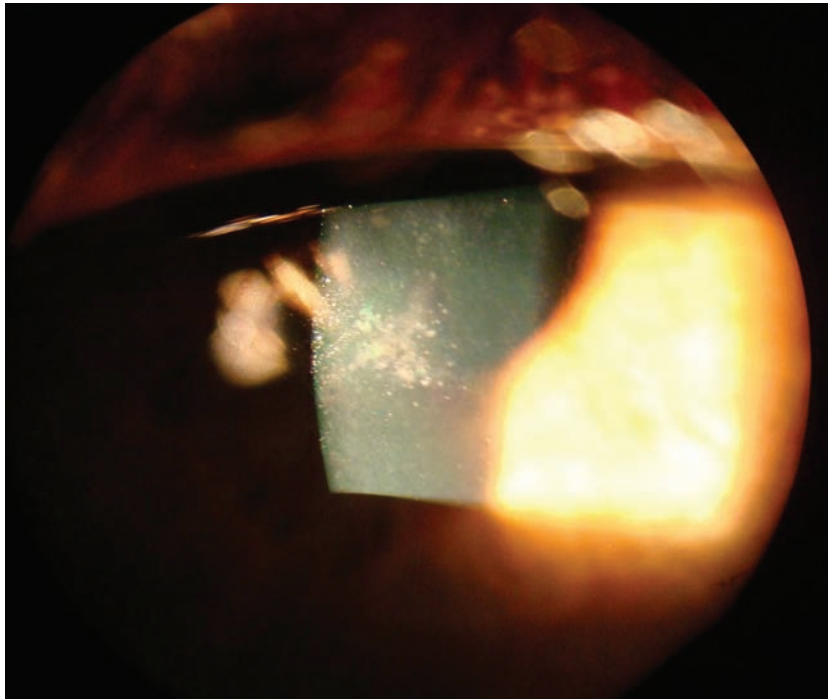
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A golfer presented with decreased vision and light sensitivity one day after rubbing his eye. He underwent LASIK 13 months earlier. Notice the significant inflammation located at the LASIK flap interface.

as volleyball players, usually suffer blunt trauma to the entire orbit, which poses much less of a specific corneal concern.

Here's a review of some of the most important considerations we must account for when recommending LVC to athletes:

- **Flap stability.** Traditional microkeratome-cut corneal flaps generally are relatively stable. But, with the advent of the femtosecond-created flaps, our concerns of long-term stability largely have been alleviated. With sufficient healing time, we do not consider blunt trauma to the facial area (e.g., secondary to boxing) or major concussive forces (e.g., secondary to football) to be deterrents for creating laser-assisted flaps.

Also, femtosecond-created flaps have been shown to yield better contrast sensitivity, a faster visual recovery and improved quality of

vision compared to mechanically-created flaps.²

Extensive testing has confirmed the stability of femtosecond-created corneal flaps.³ So, we do not consider the flap a variable in the surgical decision process unless there is a significant risk of digital trauma to the eye. One study indicated that the amount of blunt force needed to dislodge a laser-cut flap typically was enough to dislocate the lens and cause iris separation from its sclera/ciliary attachment.³ This suggests that flap dislocation is a rare, relatively minor problem, considering the excessive force the athlete would have to encounter.

Additionally, laser-cut flaps were shown to be stable following testing with variably angled, direct air streams in excess of 400mph.³ Although, flap stability was shown to be the same at both days one

and nine postoperatively, we still err on the side of caution (one week) before permitting our athletes to resume contact sports.

- **Recovery time.** Although individuals who undergo either custom surface ablation or LASIK with femtosecond-cut flaps exhibit similar postoperative results at the three- to six-month follow-up, studies show a much faster recovery of optimum vision in patients with laser-created flaps.⁴ For athletes who currently are in season or cannot sacrifice a prolonged off-season visual recovery period, LASIK with femtosecond-cut corneal flaps is a much better option than surface ablation.

- **Type of sport.** The two major sports in which we consider flap integrity to be of fundamental concern are basketball and wrestling. Basketball players and wrestlers have a disproportionately high incidence of digital cornea insult, and mechanical trauma to the flap can negatively impact the chance for good visual recovery.

Furthermore, the growing popularity of mixed martial arts and ultimate fighting has kept our concerns about finger-related eye trauma fairly high. For these athletes, we will not create a corneal flap of any kind.

Additionally, we often choose not to create a flap in swimmers. Instead, we recommend surface ablation procedures. Why? Because swimming goggles often exhibit constant pressure on the globe when worn, as well as create a vacuum suction during both application and removal—both of which could dislodge the flap.

- **Existing refractive error.** Although some studies have shown similar postoperative uncorrected visual acuity between conventional and wavefront-guided LASIK

(wLASIK) in patients with existing refractive error, these reports also show that wavefront-guided treatments have produced significantly better outcomes for contrast sensitivity and glare under mesopic conditions, as well as fewer subjective complaints.⁵ Some of the accuracy is due to flap architecture (e.g., femtosecond-created flaps have a planar configuration whereas microkeratome-cut flaps are meniscus-shaped and may produce some unwanted optical power).

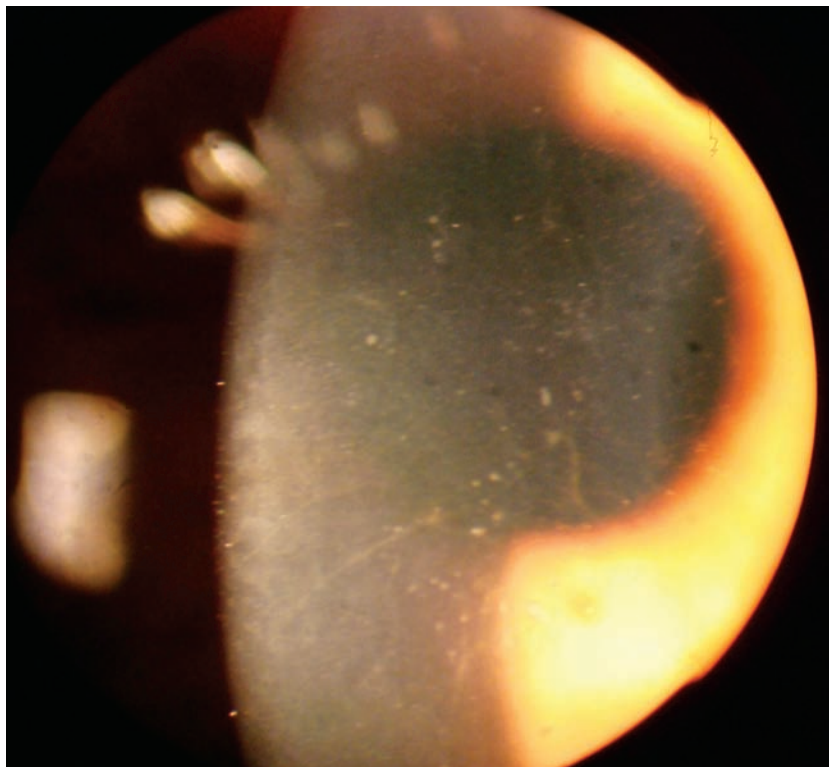
Understanding the importance of contrast sensitivity to visual performance in athletes, we always recommend wavefront-guided treatments—if possible.

- **Enhancement procedures.**

Assuming that there are no physiological contraindications, we will enhance an athlete based on best-corrected visual acuity and symptoms. This includes very small residual refractive error. Understanding the necessity of exceptional vision, we are more likely to enhance a very small residual refractive error in an athlete than in a normal patient.

To illustrate this point, our medical director, Steven Dell, M.D., recently performed an enhancement procedure on a Major League Baseball player. The athlete had an uncorrected visual acuity of 20/20-O.S. This was important to address because he bats right-handed, which means that flawless visual acuity in his left eye is essential for hitting the ball. In a typical patient, the risk/reward ratio would not justify an enhancement procedure for 20/20- visual acuity. But, in this particular case, the enhancement was justified and the patient was extremely satisfied.

We usually use wavefront-guided surface ablations to enhance our athletes (although this is techni-



After one week of aggressive topical corticosteroid therapy, the golfer's interface inflammation was almost completely resolved.

cally off-label). Wavefront-guided enhancements have been shown to reduce HOAs and improve low contrast sensitivity, whereas conventional enhancements have been shown to increase HOAs and decrease high contrast sensitivity.^{6,7}

- **Postoperative dry eye.** As always, significant preoperative dry eye is a contraindication to LVC, but postoperative dry eye often is unavoidable. Because dry eye can increase HOAs—partially negating some of the benefits of wavefront-guided treatments—we treat any postoperative dry eye very aggressively.^{8,9} This includes identifying any sign or symptoms of mild dry eye during the preoperative work-up, and then starting any necessary treatments from weeks to months in advance.

Our threshold for deficient tear volume or mild blepharitis is very

low. Low tear volume may be treated with artificial tears (with or without punctal occlusion) or topical cyclosporine eye drops. There is mounting evidence that nutritional therapy may increase tear production and quality, so this is a routine suggestion of ours.¹⁰ Although moderate to severe blepharitis is a contraindication for refractive surgery, patients with mild blepharitis are operated on routinely. While mild blepharitis ultimately carries the same risks of more severe cases, we are genuinely concerned about the athlete's visual performance as well.

Poor or altered meibum production will lead to early tear film break-up time (TFBUT), evaporative dry eye and loss of surface tension that is required to maintain optimum tear meniscus. Preoperative treatment options for

LVC in 'Airborne Athletes'

Previously, LASIK had not been authorized for aviators in the U.S. Navy and U.S. Air Force because of concerns regarding postoperative vision quality. A pivotal study conducted by Steven Schallhorn, M.D., and associates indicated that patients who had femtosecond-cut flap wLASIK for myopia correction experienced significantly better visual performance while driving at night than those who underwent non-wLASIK with mechanical keratome-cut corneal flaps.¹² These results were instrumental in the U.S. military's decision to permit wLASIK in aviators and astronauts.¹²

The U.S. military and NASA have conducted extensive research on pilots in extreme conditions. Parameters studied included postoperative contrast sensitivity, visual acuity, return to flight time, postoperative flight performance, hypoxia and high-altitude flap stability. The researchers noted that, in all parameters studied, wLASIK with femtosecond laser-created flaps proved to be equal or superior to any other form of vision correction studied.¹³ With this extensive body of research, our clinicians are now comfortable recommending the aforementioned treatment option to all pilots that we treat.

At this time, every branch of the U.S. military permits custom wLASIK with a laser-created flap for its pilots. The only exception is that Navy pilots (and prospective pilots) must agree to enter a study before undergoing the procedure.

blepharitis include oral tetracycline analogues, topical macrolide antibiotic drops, topical combination steroid/antibiotic drops and nutritional support. We are cautious about the use of warm compresses and lid scrubs during both the preoperative and acute postoperative phases, because heat and mechanical stimulation can exacerbate inflammation. Instead, we may use warm compresses and lid scrubs for long-term blepharitis management after the surgery.

When the tear film evaporates, it does so in a non-uniform fashion (which is evident upon watching TFBUT with fluorescein staining). This is important to understand, considering that the largest index of refractive change in the human eye occurs at the air/tear film interface.¹¹ So, this process can have a significant impact on any athlete's ability to perceive images clearly.

With thinner corneal flaps and shallower ablation depths, surgeons no longer sever as many corneal nerves as they used to. This has yielded far fewer cases of persistent

dry eye in athletes. Nonetheless, a large armamentarium of treatment options is required before considering refractive surgery on an elite, professional athlete.

In addition to more aggressive therapies, artificial tears and omega-3 fatty acids are mainstays of our preoperative treatment that we often continue for several months into the postoperative period. There is also mounting speculative research that supplementation with macular carotenoids may have a beneficial effect on vision following laser surgery. There has yet to be repeatable and well-controlled data to support this hypothesis, but we are very interested in the future results.

At this time, the only two laser vision procedures that our practice offers athletes are wavefront-guided surface ablation and femtosecond-created flap wLASIK. We believe that these two procedures give our athletes the best possible postoperative vision as well as the fewest potential complications.

Due to decreased recovery time and improved patient safety, we perform significantly more wLASIK procedures than surface ablations on our athletes.

In general, however, you must pay particular attention to both preoperative and postoperative dry eye concerns. Do not forget to educate athletes on the importance of artificial tear use during the postoperative period. Also, be sure to inform athletes that enhancement procedures may be more common due to their specific visual requirements. ■

Dr. Cunningham is the director of research and optometry at Dell Laser Consultants, in Austin, Texas.

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Cataract Prevention: A Race Against Time?

Even moderate delay or prevention of age-related cataracts could put a significant dent in vision loss and health care costs.

By Marc D. Myers, O.D., and Andrew S. Gurwood, O.D.

Although many clinicians consider cataract surgery to be an economical procedure, cost containment is always a concern in an unstable economy. Cataract is one of the leading causes of reversible vision loss in the world, and ranks as the most common age-related eye disease in America.¹⁻³ With nearly three million cataract surgeries performed in the United States each year, it is estimated that the total annual cost of cataract management exceeds six billion dollars.^{3,4}

If researchers were able to successfully develop even moderate avenues of prevention, the cost savings could amount to tens of millions of dollars. So, the search is on for methods to delay the onset of clinically significant cataract formation. Currently, considerable research is being conducted to evaluate such modalities.

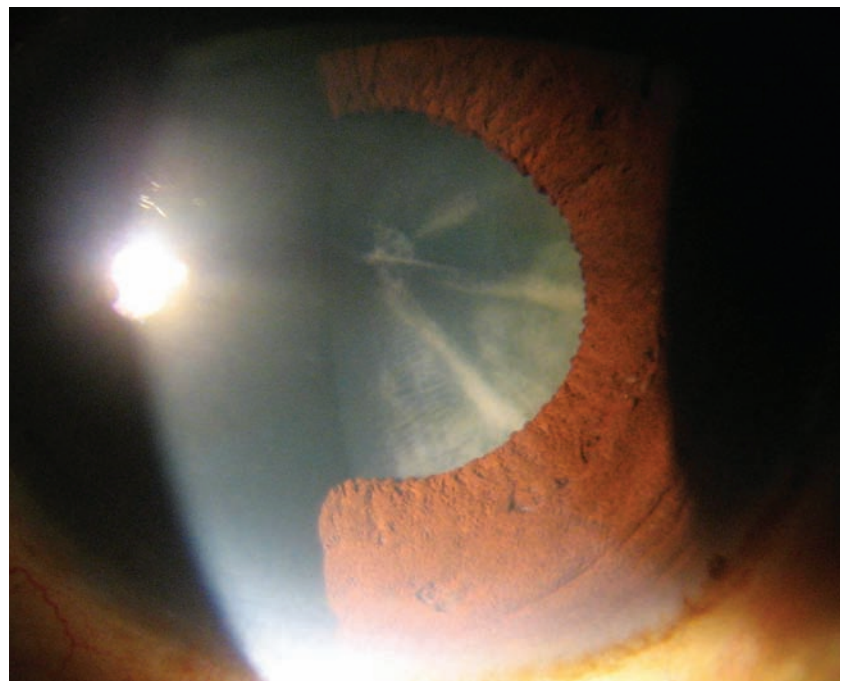
In this article, we will look at risk factors for age-related cataracts (ARCs), its pathophysiology and some noteworthy research regarding new and potential treatments.

Risk Factors and Pathophysiology

So, what causes a cataract? More to the point: Can any of those causes be modified or

removed in order to prevent cataract formation?

Intrinsic or non-modifiable risk factors include age, gender, race, family history of cataracts, myopic



Cortical cataracts—characterized by white, wedge-like opacities that start in the periphery of the lens—form due to the disruption of fiber cell membranes, followed by the disintegration of the damaged fiber cell's cytoplasmic contents.

refractive error and the size of the crystalline lens.⁵⁻⁸ Major environmental risk factors include, but are not limited to, UV exposure, living in warmer ambient temperatures, nutrition and supplement usage, systemic disease and its medical management, increased body mass index, and smoking.⁵⁻⁸ The most common forms of ARCs include nuclear sclerotic, cortical spoke and posterior subcapsular (PSC) lens opacities.¹⁻⁶

- **Nuclear sclerosis** is considered the end-product of insoluble lens protein aggregation.^{8,9} Although the process is not completely understood, these changes in the lens proteins are due to increased protein oxidation as well as modifications of the crystalline proteins.^{8,9}

As protein oxidation increases, insoluble proteins accumulate and aggregation occurs, causing the process of aging of the lens—in which the lens nucleus becomes more rigid, light scattering occurs and lens coloration increases.^{8,9}

- **Cortical cataracts** form due to the disruption of fiber cell membranes, followed by the disintegration of the damaged fiber cell's cytoplasmic contents.⁶⁻⁹ Different than nuclear opacification, cortical cataract formation occurs whenever alterations develop in the cortical fibers.

This type of cataract formation is not necessarily a consequence of aging. Unlike nuclear cataracts—which evolve uniformly throughout the center of the lens—cortical lens changes begin in small clusters of cortical fiber cells near the equator of the lens and progress as stressors dictate.⁶⁻⁹

- **PSC opacities**, perhaps the least understood type of ARC, occur less often than nuclear or cortical opacities.⁸⁻¹⁰ Postmortem lens examination has revealed that

Significant Studies of Vitamins/Minerals in ARC

- **The Age-Related Eye Disease Study (AREDS)**, sponsored by the National Eye Institute, is the most highly publicized study to investigate the use of oral supplements for eye care.³⁶ Researchers used a high-dose formulation of antioxidants and zinc to determine if these oral supplements could alter the clinical course of AMD and/or the progression of lens opacities.³⁶

After five years of supplement usage, the opacity component of the study revealed that the AREDS supplement formulation did not slow the development or progression of age-related lens opacification.³⁶

- **AREDS 2** has been designed to determine if modifying the formulation—by adding macular xanthophylls (lutein and zeaxanthin) and/or long-chain omega-3 fatty acids (DHA) and eicosapentaenoic acid (EPA)—would impact the progression to advanced AMD in high-risk patients, or if the supplement could influence the progression of ARC.⁴⁷ In addition, the AREDS 2 formulation includes reduced zinc and/or no beta-carotene.⁴⁷ The outcome of AREDS 2 is currently pending.

- **The Roche European American Cataract Trial (REACT)** was a multicenter, double-masked, placebo-controlled study that monitored cataract progression over a four-year period.⁴⁸ A total of 297 adult American and English patients were randomized to receive a placebo or an oral multivitamin containing beta-carotene, vitamin C and vitamin E. Cataract severity was documented with serial digital retroillumination imagery of the lens.

Progression was quantified by image analysis assessing increased area of lens opacity. In the American patient population, the combination of micronutrients produced a slight deceleration in the progression of ARC after a three-year period. In the English population, no statistically significant benefit was observed.⁴⁸

- **The Linxian Cataract Study** looked at subjects from a rural Chinese population, ages 45 to 74, in a large trial to determine if a multivitamin supplement could affect the risk of ARC.⁴⁹ Subjects received one of four multivitamin/mineral combinations. The possible combinations included retinol/zinc, riboflavin/niacin, ascorbic acid/molybdenum and selenium/alpha-tocopherol/beta-carotene. Subjects in the control group received a placebo.

In the 65- to 74-year-old patient group, who were randomized to the retinol/zinc preparation, a statistically significant 36% reduction in nuclear cataracts was observed.⁴⁹ No statistical significance was observed in the other age groups, other combination groups or in the development of cortical spoke or PSC cataracts.⁴⁹

- **The Beaver Dam Eye Study** was an investigation of the relationship between multivitamin supplement use and the five-year incidence of nuclear, cortical and posterior subcapsular cataract.⁴¹ The subjects reported the type, dosage and duration of supplement used at baseline and logged their supplement usage to the end of the five-year trial period.⁴¹ When compared to nonusers, those reporting the use of vitamin C or E showed a 60% lower five-year risk of nuclear or cortical cataract formation.⁵⁰

The authors concluded that the evidence supported an association between supplement use and benefit, but also acknowledged the limitations of the study's design, its inability to measure lifestyle differences and the inability of the study to determine the specifics of other daily nutrients consumed.⁵⁰

the cellular changes appearing secondary to PSC are streams of cells migrating from the equator to the posterior pole.⁸⁻¹⁰

These cells are swollen with eosinophilic cytoplasm, suggesting

that they have somehow failed to elongate.⁸⁻¹⁰

The abnormal conglomeration reduces the transparency of the lens close to the anterior convergence point (or nodal point) of the eye,

interfering with the convergent light of near objects.

Are Vitamin Supplements the Answer?

Dietary antioxidants, such as vitamin C (ascorbate), vitamin E (tocopherol), beta-carotene and omega-3 fatty acids, have been included in human epidemiologic, animal and laboratory-based studies that investigate ARC formation (see “*Significant Studies of Vitamins/Minerals in ARC*,” page 53).¹¹

Carotenoids, specifically the xanthophylls xanthin and zeaxanthin that have been found in the crystalline lens, also are being studied to assess their ability to influence cataractogenesis.¹²

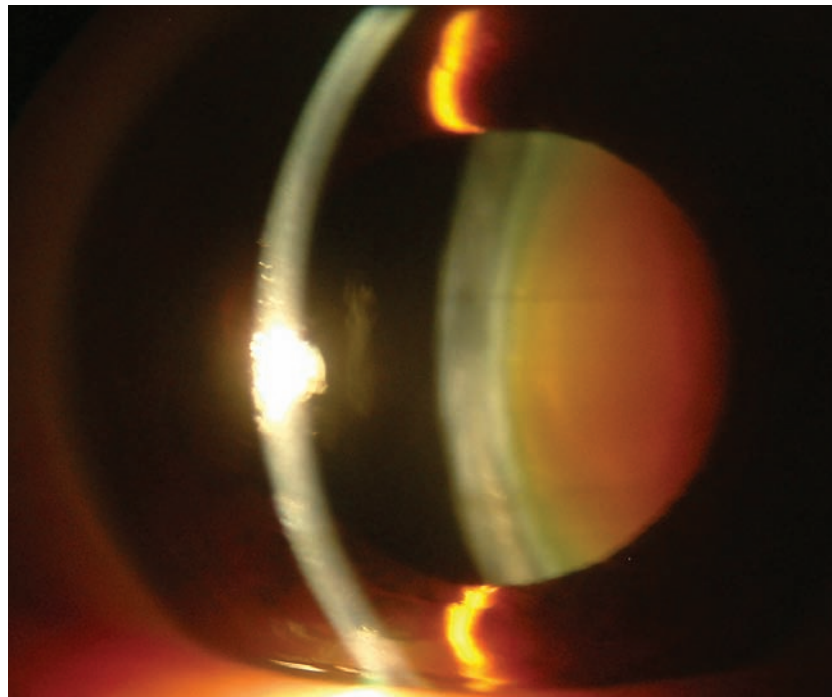
Vitamin C

Vitamin C is present in high concentrations in the aqueous humor and the human lens.¹³ It prevents aggregation of crystallin by inhibiting disulphide cross-linking; by acting as a scavenger of reactive oxygen species; and by sequestering unfolded lens proteins.^{13,14} Mixed conclusions have been reported with respect to the potential benefit of a vitamin C supplement and

Mixed conclusions have been reported with respect to the potential benefit of a vitamin C supplement and reduced cataract incidence.

reduced cataract incidence.¹⁵⁻²⁰

One investigation monitored 24,593 Swedish women who were administered a questionnaire regarding lifestyle factors and supplement use.¹⁵ Following eight years of monitoring, researchers concluded that the use of vitamin C supplements may actually be asso-



With nuclear sclerosis, the lens nucleus becomes more rigid, light scattering occurs, and lens coloration increases.

ciated with a higher risk of ARC formation.¹⁵

In contrast, some studies identified dietary vitamin C as having a protective role that may lower the risk of ARC formation.^{16,17} In addition, a recent study examined cataract formation revealing a strong association with vitamin C depletion and lens opacification.¹⁸ In separate investigations assessing the impact of long-term vitamin and carotenoid

use, vitamin C was found to have an association with reduced risk of cortical cataract formation in a cohort of non-diabetic women.¹⁹

A later report from the same cohort of U.S. women discovered that subjects with the highest intake of vitamin C had a reduced prevalence of nuclear opacities.²⁰

In the Baltimore Longitudinal Study on Aging, researchers assessed plasma levels of ascorbate in 660 subjects and discovered that recommended plasma levels were not associated with risk of nuclear or cortical lens opacities.²¹ In phase II of the Physician Health Study, male physicians from the United States were randomized to use vitamin C or placebo—this large study demonstrated that long-term use of vitamin C had no overall effect on cataract occurrence.²¹ Taking these data as a whole, it appears that vitamin C neither prevents nor provokes cataractogenesis.¹³⁻²²

Vitamin E

Vitamin E, specifically alpha-tocopherol, is a lipid-soluble antioxidant that is concentrated in lens fibers and membranes.²³ The coenzyme is believed to inhibit cataract formation by reducing photoperoxidation of lens lipids and by

stabilizing lens cell membranes; however, studies have shown vitamin E supplementation does not necessarily reduce the incidence of ARC.^{23,24}

As was the case with vitamin C, phase II of the Physician Health Study found that long-term vitamin E supplementation had no overall effect on cataract occurrence.²² Additional large clinical trials that reported similar results include the Alpha-Tocopherol, Beta-Carotene (ATBC) Cancer Prevention Study; the Vitamin E, Cataract, and Age-Related Macular Degeneration (VECAT) Study; and the Women's Health Study (WHS).²⁵⁻²⁷

Similarly, investigators in Melbourne, Australia, determined that a daily dose of 500 international units of vitamin E used over a four-year period did not reduce the

incidence of progression of ARC.²⁸ Also, a randomized, double-masked, placebo-controlled trial using 600 international units of vitamin E on an every-other-day dosing schedule vs. a placebo for 9.7 years showed no benefit in daily use of vitamin E in cataract prevention.²⁹

In short, the majority of evidence suggests that vitamin E does not delay the onset of cataracts.

Beta-carotene

Beta-carotene, a fat-soluble compound and member of the carotenoid family, is a naturally occurring component of fruits, grains, oils and vegetables.³⁰ Common foods that contain beta-carotene include green plants, carrots, sweet potatoes, squash, spinach, apricots and green peppers.

In its natural form, beta-carotene is a precursor (inactive form) to vitamin A.³⁰ Beta-carotene produces retinol—a fat-soluble form of vitamin A.

Beta-carotene has been the focus of investigations due to its effect as an antioxidant at low partial pressures. Low partial pressures are known to exist within the crystalline lens, making the substance worthy of investigating as a possible cataract suppressant. The obvious advantage is that this compound is obtainable naturally in fruits and vegetables.^{30,31}

WHS tested the benefits of beta-carotene (50mg on alternate days) on the development of ARC formation in 18,405 eligible patients.³¹ After more than two years of follow up, researchers concluded that beta-carotene had neither a large


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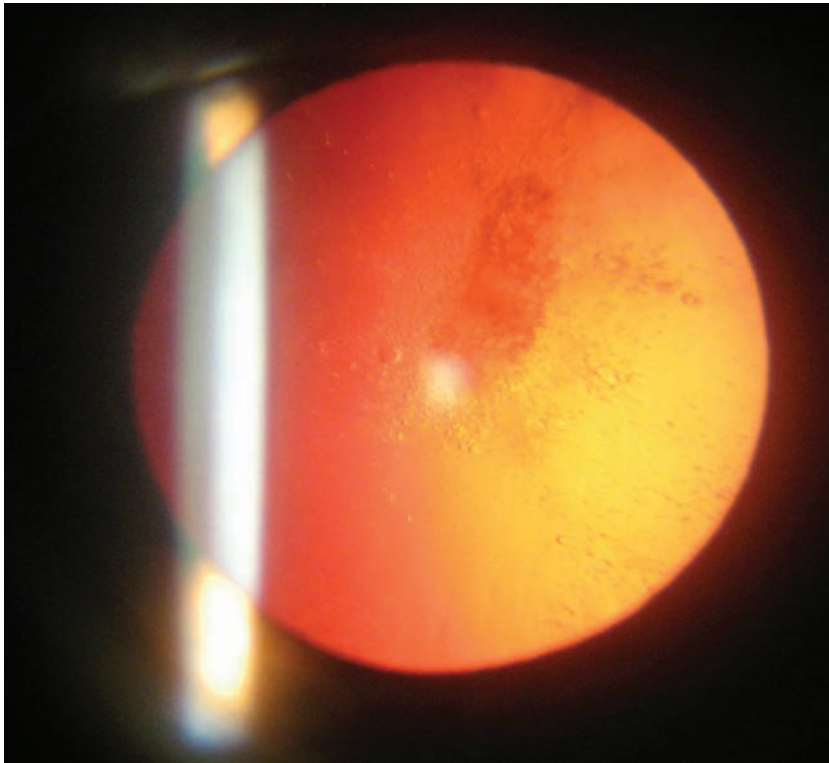
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Posterior subcapsular cataracts start as small, opaque areas that usually form near the back of the lens.

benefit nor harmful effect on cataractogenesis.³¹

A similarly designed study, which included more than 22,000 American male physicians, concluded after 12 years of beta-carotene supplementation, that no overall benefit or harm on cataract development was identified.³²

Omega-3 Fatty Acids

Omega-3 polyunsaturated fatty acids are fats that may be found in marine and plant oils. In humans, during late fetal and early neonatal life, the brain and eye are primarily enriched with the omega-3 fatty acid docosahexaenoic acid (DHA).³³ High levels of DHA are present within the outer segments of the retina's photoreceptor cells.

Recent literature has identified that the dietary intake of omega-3 fatty acids may reduce the risk of

early age-related macular degeneration (AMD).^{34,35} The Age-Related Eye Disease Study is ongoing and is investigating if omega-3 fatty acids, along with other nutritional supplements, may aid in the prevention or decrease the rate of progression of AMD.³⁶

A systematic literature review in 2005 investigated the effect that omega-3 fatty acids have on human eye health.³⁷ It was specifically designed to examine whether omega-3 fatty acids could prevent, slow the rate of progression or decrease the rate of ARC surgery in an aging population. Data revealed no statistically significant association between the intake of foods or oils containing omega-3 fatty acid and a decreased incidence of ARC.³⁷

In our review of the literature that has emerged since that report,

we found no peer-reviewed medical or scientific studies that investigated stand-alone, omega-3 fatty acid supplementation and its influence on ARC. We did, however, discover a paucity of information discussing the potential benefits of omega-3 fatty acid intake and cataract formation as we explored commercial products. However, the majority of this information appears in non-peer reviewed medical literature.

Other Proposed Treatments

Compared to proteins, lipids make up only a small component of the lens structure. Lipid peroxidation (LPO) does, however, lead to the disintegration of lens fiber plasma membrane.³⁸ One proposed treatment strategy to prevent the formation of cataractous opacities is to prevent the activation of LPO end products by administering antioxidants that have a strong affinity to LPO.^{2,38} Carnosine is a histidine-containing compound that occurs naturally in several human tissues, particularly muscle.

One study reported that carnosine is a potent lipid peroxidase mimetic that possesses powerful antioxidant properties.³⁹

N-acetylcarnosine (NAC) is a pro-drug of carnosine that has been developed for clinical use as an eye drop. Marketed as Can-C (1% N-acetylcarnosine, Innovative Vision Products), the topical solution is supplied as a cellulose-based compound that can be purchased without a physician's prescription.³⁹⁻⁴¹ In a randomized, double blind, placebo-controlled clinical trial of NAC, Mark Babizhayev, Ph.D., and colleagues reported improvement in best corrected visual acuity, glare sensitivity and increased lens transmissivity.⁴¹

Our literature search of peer-

BECAUSE inflammation HAPPENS



Make DUREZOL® Emulsion your steroid for post-op care.

Unique molecular design optimizes potency and penetration^{1,4}

Covered on more than 82% of national formularies⁵

IMPORTANT SAFETY INFORMATION:

Indications and Usage: DUREZOL® Emulsion is a topical corticosteroid that is indicated for the treatment of inflammation and pain associated with ocular surgery.

Dosage and Administration: Instill one drop into the conjunctival sac of the affected eye(s) 4 times daily beginning 24 hours after surgery and continuing throughout the first 2 weeks of the postoperative period, followed by 2 times daily for a week and then a taper based on the response.

Contraindications: DUREZOL® Emulsion, as with other ophthalmic corticosteroids, is contraindicated in most active 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. If this product is used for 10 days or longer, IOP 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 beyond 28 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.

- **Bacterial infections** – 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 infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.
- **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.

Adverse Events: Ocular adverse reactions occurring in 5-15% of subjects in clinical studies with DUREZOL® Emulsion included corneal edema, ciliary and conjunctival hyperemia, eye pain, photophobia, posterior capsule opacification, anterior chamber cells, anterior chamber flare, conjunctival edema, and blepharitis.

Please see full prescribing information on adjacent page.

Alcon®

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DUR11500JAD

U.S. Patent No. 6,114,319

DUREZOL®
(difluprednate ophthalmic emulsion) 0.05%

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all of the information needed to use Durezol[®] safely and effectively. See full prescribing information for Durezol.

DUREZOL[®] (difluprednate ophthalmic emulsion) 0.05%
Initial U.S. approval: 2008

INDICATIONS AND USAGE

Durezol is a topical corticosteroid that is indicated for the treatment of inflammation and pain associated with ocular surgery. (1)

DOSAGE AND ADMINISTRATION

Instill one drop into the conjunctival sac of the affected eye(s) 4 times daily beginning 24 hours after surgery and continuing throughout the first 2 weeks of the postoperative period, followed by 2 times daily for a week and then a taper based on the response. (2)

DOSAGE FORMS AND STRENGTHS

Durezol contains 0.05% difluprednate, as a sterile preserved ophthalmic emulsion for topical ophthalmic use only. (3)

CONTRAINDICATIONS

Durezol, 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. (4)

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. If this product is used for 10 days or longer, IOP should be monitored. (5.1)
- Cataracts - Use of corticosteroids may result in posterior subcapsular cataract formation. (5.2)
- 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 beyond 28 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. (5.3)
- Bacterial infections - 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 infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated. (5.4)
- 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). (5.5)
- 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. (5.6)

To report SUSPECTED ADVERSE REACTIONS, contact Alcon Laboratories, Inc. at 1-800-757-9195 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

See 17 for PATIENT COUNSELING INFORMATION.

Revised date: March 2010

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FULL PRESCRIBING INFORMATION

1 INDICATIONS AND USAGE

Durezol (difluprednate ophthalmic emulsion) 0.05%, a topical corticosteroid, is indicated for the treatment of inflammation and pain associated with ocular surgery.

2 DOSAGE AND ADMINISTRATION

Instill one drop into the conjunctival sac of the affected eye(s) 4 times daily beginning 24 hours after surgery and continuing throughout the first 2 weeks of the postoperative period, followed by 2 times daily for a week and then a taper based on the response.

3 DOSAGE STRENGTHS

Durezol contains 0.05% difluprednate as a sterile preserved emulsion for topical ophthalmic administration.

4 CONTRAINDICATIONS

The use of Durezol, as with other ophthalmic corticosteroids, is contraindicated in most active 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 disease of ocular structures.

5 WARNINGS AND PRECAUTIONS

5.1 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 beyond 28 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.

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, 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 culture should be taken when appropriate.

5.7 Topical ophthalmic use only

Durezol is not indicated for intraocular administration.

6 ADVERSE REACTIONS

Adverse 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 subjects in clinical studies with Durezol included corneal edema, ciliary and conjunctival hyperemia, eye pain, photophobia, posterior capsule opacification, anterior chamber cells, anterior chamber flare, conjunctival edema, and blepharitis. Other ocular adverse reactions occurring in 1–5% of subjects included reduced visual acuity, punctate keratitis, eye inflammation, and iritis. Ocular adverse events occurring in < 1% of subjects included application site discomfort or irritation, corneal pigmentation and striae, episcleeritis, eye pruritus, eyelid irritation and crusting, foreign body sensation, increased lacrimation, macular edema, scleral hyperemia, and uveitis. Most of these events may have been the consequence of the surgical procedure.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Teratogenic Effects
Pregnancy Category C. Difluprednate has been shown to be embryotoxic (decrease in embryonic body weight and a delay in embryonic ossification) and teratogenic (cleft palate and skeletal) anomalies when administered subcutaneously to rabbits during organogenesis at a dose of 1–10 µg/kg/day. The no-observed-effect-level (NOEL) for these effects was 1 µg/kg/day, and 10 µg/kg/day was considered to be a teratogenic dose that was concurrently found in the toxic dose range for fetuses and pregnant females. Treatment of rats with 10 µg/kg/day subcutaneously during organogenesis did not result in any reproductive toxicity, nor was it maternally toxic. At 100 µg/kg/day after subcutaneous administration in rats, there was a decrease in fetal weights and delay in ossification, and effects on weight gain in the pregnant females. It is difficult to extrapolate these doses of difluprednate to maximum daily human doses of Durezol, since Durezol is administered topically with minimal systemic absorption, and difluprednate blood levels were not measured in the reproductive animal studies. However, since use of difluprednate during human pregnancy has not been evaluated and cannot rule out the possibility of harm, Durezol should be used during pregnancy only if the potential benefit justifies the potential risk to the embryo or 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 breast milk. Systemically administered corticosteroids appear in human milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. Caution should be exercised when Durezol is administered to a nursing woman.

8.4 Pediatric Use

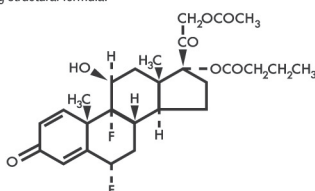
Safety and effectiveness in pediatric patients has not been established.

8.5 Geriatric Use

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

11 DESCRIPTION

Durezol (difluprednate ophthalmic emulsion) 0.05% is a sterile, topical anti-inflammatory corticosteroid for ophthalmic use. The chemical name is 6α,9-difluoro-11β,17,21-trihydroxyprogna-1,4-diene-3,20-dione 21-acetate 17-butyrate (CAS number 23674-86-4). Difluprednate is represented by the following structural formula:



Difluprednate has a molecular weight of 508.56, and the empirical formula is C₂₇H₃₄F₂O₇. Each mL contains: ACTIVE: difluprednate 0.5 mg (0.05%); INACTIVE: boric acid, castor oil, glycerin, polysorbate 80, purified water, sodium acetate, sodium EDTA, sodium hydroxide (to adjust the pH to 5.2 to 5.8). The emulsion is essentially isotonic with a tonicity of 304 to 411 mOsm/kg. PRESERVATIVE: sorbic acid 0.1%.

12 CLINICAL PHARMACOLOGY

12.1 Mechanism of Action

Corticosteroids inhibit the inflammatory response to a variety of inciting agents that may delay or slow healing. They inhibit edema, fibrin deposition, capillary dilation, leukocyte migration, capillary proliferation, fibroblast proliferation, deposition of collagen, and scar formation associated with inflammation. There is no generally accepted explanation for the mechanism of action of ocular corticosteroids. However, corticosteroids are thought to act by the induction of phospholipase A2 inhibitory proteins, collectively called lipocortins. It is postulated that these proteins control the biosynthesis of potent mediators of inflammation such as prostaglandins and leukotrienes by inhibiting the release of their common precursor arachidonic acid. Arachidonic acid is released from membrane phospholipids by phospholipase A₂. Difluprednate is structurally similar to other corticosteroids.

12.3 Pharmacokinetics

Difluprednate undergoes deacetylation in vivo to 6α,9-difluoroprednisolone 17-butyrate (DFB), an active metabolite of difluprednate. Clinical pharmacokinetic studies of difluprednate after repeat ocular instillation of 2 drops of difluprednate (0.01% or 0.05%) QID for 7 days showed that DFB levels in blood were below the quantification limit (50 ng/mL) at all time points for all subjects, indicating the systemic absorption of difluprednate after ocular instillation of Durezol is limited.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, and Impairment of Fertility

Difluprednate was not genotoxic in vitro in the Ames test, and in cultured mammalian cells CHL/IU (a fibroblastic cell line derived from the lungs of newborn female Chinese hamsters). An in vivo micronucleus test of difluprednate in mice was also negative. Treatment of male and female rats with subcutaneous difluprednate up to 10 µg/kg/day prior to and during mating did not impair fertility in either gender. Long term studies have not been conducted to evaluate the carcinogenic potential of difluprednate.

13.2 Animal Toxicology and/or Pharmacology

In multiple studies performed in rodents and non-rodents, subchronic and chronic toxicity tests of difluprednate showed systemic effects such as suppression of body weight gain; a decrease in lymphocyte count; atrophy of the lymphatic glands and adrenal gland; and for local effects, thinning of the skin; all of which were due to the pharmacologic action of the molecule and are well known glucocorticosteroid effects. Most, if not all of these effects were reversible after drug withdrawal. The NOEL for the subchronic and chronic toxicity tests were consistent between species and ranged from 1–1.25 µg/kg/day.

14 CLINICAL STUDIES

14.1 Postoperative Ocular Inflammation and Pain

Clinical efficacy was evaluated in 2 randomized, double-masked, placebo-controlled trials in which subjects with an anterior chamber cell grade ≥ 2* (a cell count of 11 or higher) after cataract surgery were assigned to Durezol or placebo (vehicle) following surgery. One drop of Durezol or vehicle was self instilled either 2 (BID) or 4 (QID) times per day for 14 days, beginning the day after surgery. The presence of complete clearing (a cell count of 0) was assessed 8 and 15 days post-surgery using a slit lamp binocular microscope. In the intent-to-treat analyses of both studies, a significant benefit was seen in the QID Durezol-treated group in ocular inflammation and reduction of pain when compared with placebo. The consolidated clinical trial results are provided below.

Ocular Inflammation and Pain Endpoints (Studies Pooled)

	Durezol QID (n = 107)		Vehicle (n = 220)	
	8	15	8	15
Anterior Chamber cell clearing (% subjects)	24 (22%)*	44 (41%)*	17 (7%)	25 (11%)
Pain free (% subjects)	62 (58%)*	67 (63%)*	59 (27%)	76 (35%)

*Statistically significantly better than vehicle, p<0.01

16 HOW SUPPLIED/STORAGE AND HANDLING

Durezol (difluprednate ophthalmic emulsion) 0.05% is a sterile, aqueous topical ophthalmic emulsion supplied in an opaque plastic bottle with a controlled drop tip and a pink cap in the following size: 5 mL in a 5 mL bottle (NDC 42826-601-05).

Storage

Store at 15–25°C (59–77°F). Do not freeze. Protect from light. When not in use keep the bottles in the protective carton.

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 emulsion. If pain develops or if redness, itching, or inflammation becomes aggravated, the patient should be advised to consult a physician. As with all ophthalmic preparations containing a preservative, patients should be advised not to wear contact lenses when using Durezol.

Revised: March 2010

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reviewed publications relating to NAC found that virtually all information on the subject had been published by Dr. Babizhayev and his colleagues. Interestingly, the publishers are not only the research scientists who discovered and patented the formulation of NAC, but they also have disclosed interests in Innovative Vision Products, the manufacturer of Can-C. With this in mind, one may recognize the potential bias that may exist, leaving the scientific and medical community with an interest in further evidenced-based research investigating the efficacy of NAC.

We located a study from the University of Sydney that investigated the efficacy of NAC with respect to protecting the crystalline lens from oxidation.⁴² The investigators, using mass spectrometry, concluded that there was no evidence suggesting that NAC had any significant direct effect on reducing the levels of oxidation within lens crystallins.⁴² Currently, a systematic review is underway in the United Kingdom that will investigate the effectiveness of carnosine as a cataract-reversing and preventive agent.⁴³ This information is pending and may shed more light on NAC as a possible medical therapy for delaying visually significant cataract formation.⁴³

Although research has been limited to animal studies, eye drops containing disulfiram (DSF) are being considered for their anti-cataract effect.⁴⁴ DSF is a powerful antioxidant that scavenges reactive oxygen species. Due to its poor water solubility, DSF must be combined with methylcellulose

to improve solubility, stability and bioavailability. Thus far, animal models have resulted in delaying cataract development in hereditary, as well as steroid-induced cataractogenesis.⁴⁴⁻⁴⁶

Most cataracts are due to natural, slowly progressive changes in lenticular physiology. This fact alone poses a challenge to those who are designing trials to reduce, delay or reverse the effects of ARC.

To date, no human trial has conclusively displayed clinically significant, reliable data that any one

For now, the most effective strategies used to prevent cataract formation or delay progression include avoiding dehydration and illness, improving nutrition, staying tobacco-free, and limiting sun exposure.

treatment or combination of treatments can produce a delay in onset, reduction in progression, or reversal of signs or symptoms of ARC.

For now, the most effective strategies used to prevent cataract formation or delay progression include avoiding dehydration and illness, improving nutrition, staying tobacco-free, and limiting sun exposure. ■

Dr. Myers is senior staff optometrist at the Coatesville Veterans Affairs Hospital in Pennsylvania. Dr. Gurwood is professor of clinical sciences at The Eye Institute of the Pennsylvania College of Optometry at Salus University in Elkins Park, Pa. He also authors Review's "Diagnostic Quiz."

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Brief Summary of Prescribing Information

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ATON Pharma, a Division of Valeant Pharmaceuticals North America LLC
Madison, NJ 07940

Rx Only

LACRISERT® (hydroxypropyl cellulose) OPHTHALMIC INSERT

DESCRIPTION

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

Each LACRISERT is 5 mg of hydroxypropyl cellulose. LACRISERT contains no preservatives or other ingredients. It is about 1.27 mm in diameter by about 3.5 mm long. LACRISERT is supplied in packages of 60 units, together with illustrated instructions and a special applicator for removing LACRISERT from the unit dose blister and inserting it into the eye.

INDICATIONS AND USAGE

LACRISERT is indicated in patients with moderate to severe dry eye syndromes, including keratoconjunctivitis sicca. LACRISERT is indicated especially in patients who remain symptomatic after an adequate trial of therapy with artificial tear solutions. LACRISERT is also indicated for patients with exposure keratitis, decreased corneal sensitivity, and recurrent corneal erosions.

CONTRAINDICATIONS

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

WARNINGS

Instructions for inserting and removing LACRISERT should be carefully followed.

PRECAUTIONS

General

If improperly placed, LACRISERT may result in corneal abrasion.

Information for Patients

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

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

Drug Interactions

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

Carcinogenesis, Mutagenesis, Impairment of Fertility

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

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

ADVERSE REACTIONS

The following adverse reactions have been reported in patients treated with LACRISERT, but were in most instances mild and transient: transient blurring of vision, ocular discomfort or irritation, matting or stickiness of eyelashes, photophobia, hypersensitivity, edema of the eyelids, and hyperemia.

DOSAGE AND ADMINISTRATION

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

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

Issued June 2007

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LACRISERT® is indicated in patients with moderate to severe Dry Eye syndromes, including keratoconjunctivitis sicca. LACRISERT® is indicated especially in patients who remain symptomatic after an adequate trial of therapy with artificial tear solutions. LACRISERT® is also indicated for patients with exposure keratitis, decreased corneal sensitivity, and recurrent corneal erosions.

Important Safety Information

LACRISERT® is contraindicated in patients who are hypersensitive to hydroxypropyl cellulose. Instructions for inserting and removing LACRISERT® should be carefully followed. If improperly placed, LACRISERT® may result in corneal abrasion. Because LACRISERT® may cause transient blurred vision, patients should be instructed to exercise caution when driving or operating machinery. Patients should be cautioned against rubbing the eye(s) containing LACRISERT®.

The following adverse reactions have been reported, but were in most instances, mild and temporary: transient blurring of vision, ocular discomfort or irritation, matting or stickiness of eyelashes, photophobia, hypersensitivity, eyelid edema, and hyperemia.

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

* In most patients, one LACRISERT® placed into each eye once daily is effective in providing all-day symptom relief. Some patients may require twice-daily use for optimal results.

References: 1. Koffler BH, McDonald M, Nelinson D, Improved signs and symptoms and quality of life with dry eye syndrome: hydroxypropyl cellulose ophthalmic insert patient registry. *Eye Contact Lens*. 2010;3:170-176.
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Prostate Surgery Precipitates 'Shock-Induced' Vision Loss

This patient presented with decreased vision in his right eye after waking up from an invasive surgical procedure that involved severe blood loss.

By **Trina C. Perkins, O.D.**, and **Susannah B. Marcus-Freeman, O.D.**

Non-arteritic anterior ischemic optic neuropathy (NAION) is a common condition that causes sudden, painless vision loss in older individuals. One possible etiology of NAION is acute blood loss that results in hypotension, anemia and associated optic nerve ischemia. The optic neuropathy that results from this sequence of events has been termed shock-induced anterior ischemic optic neuropathy (SIAION).¹

Although rare, SIAION has been documented in patients who have suffered acute blood loss as well as a dramatic change in blood pressure secondary to systemic surgery.

Here, we present the case of a patient who developed SIAION after undergoing complicated radical retropubic prostatectomy. Concurrently, the patient was found to have bilateral disc swelling, and was subsequently diagnosed with a frontal lobe meningioma. It is possible that the optic nerve swelling might have

either precipitated or contributed to the development of AION.

History

A 61-year-old white male presented to the eye clinic complaining of a "purple haze" over the superior half of his vision O.D. after waking from a radical retropubic prostatectomy two weeks earlier. The patient also reported that he saw bilateral flashing lights when his eyes were closed. The symptoms had not changed since initial onset.

His medical history was significant for sleep apnea, colonic polyps, mixed hyperlipidemia, hypertension, allergic rhinitis and prostate cancer.

His preoperative blood pressure measured 154/94mm Hg, with a hemoglobin count of 15.2g/dL and a hematocrit volume of 45%. After surgery, his blood pressure measured 92/52mm Hg, his hemoglobin count was 9.1g/dL, and his hematocrit volume was 27%. The patient required six pints of A-negative blood due to significant periopera-

tive and postoperative bleeding.

At the time of the patient's first visit, his medical therapy consisted of belladonna/opium suppository (for severe pain from spasms of the urinary tract), ciprofloxacin, clonidine, diphenhydramine, docusate, hydrochlorothiazide, lisinopril, metoprolol, oxybutynin chloride, oxycodone/acetaminophen and promethazine injections.

Diagnostic Data

His best-corrected visual acuity was 20/20 O.D. and O.S. Confrontation visual fields revealed a superior defect in the right eye. Additionally, we noted a 2+ relative afferent pupillary defect (RAPD) O.D.

His intraocular pressure was 15mm Hg O.U. Slit-lamp examination of the anterior segment was unremarkable. Fundus examination revealed an edematous, pale optic nerve in the right eye with one inferior splinter hemorrhage (*figure 1*). The left optic nerve appeared congested with indistinct

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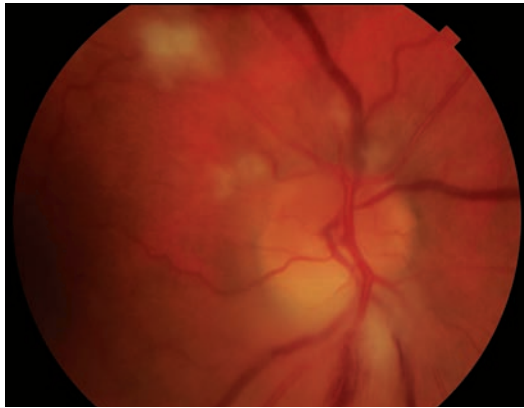
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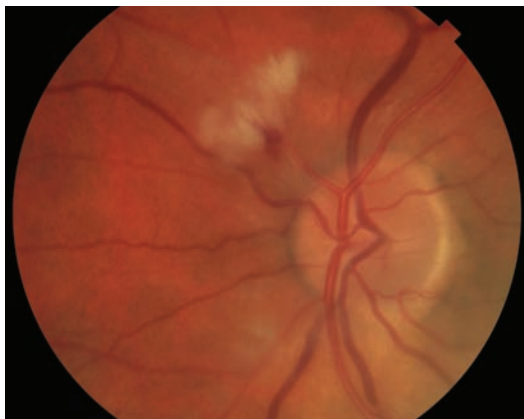
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1. Fundus examination of the right eye revealed an edematous optic nerve with inferior pallor. We documented cotton-wool spots in the peripapillary region and one inferior splinter hemorrhage.



2. The left optic nerve appeared congested with indistinct margins. There were a few peripapillary cotton-wool spots as well as one small adjacent hemorrhage.

margins (*figure 2*). Cotton-wool spots were present in the peripapillary region of both eyes (O.D. > O.S.). Humphrey visual field testing revealed a complete superior altitudinal defect in the right eye, as well as blind spot enlargement in the left eye (*figure 3*).

The pale, swollen optic nerve, RAPD and superior altitudinal visual field defect in the right eye were indicative of ischemic optic neuropathy, which was likely related to blood loss sustained during surgery. The cause of the

left optic nerve swelling was not as clear, so we ordered an MRI.

The MRI revealed a right frontal lobe lesion with marked surrounding edema (*figures 4 and 5*). The lesion measured approximately 1.5cm x 1.7cm x 1.8cm, with a large area of surrounding edema that extended back to compress the fluid space surrounding the right optic nerve (*figures 6 and 7*). We noted significant mass effect, with a right-to-left midline shift of approximately 5mm.

Diagnosis

The patient's optic nerve appearance, in conjunction with his medical history and symptoms, led us to a diagnosis of SIAION in the right eye. Based on the clinical appearance and the presence of an enlarged blind spot on visual field testing, we determined papilledema to be the cause of disc swelling in the left eye. Papilledema

likely contributed to the development of ischemic optic neuropathy in his right eye.

Treatment and Follow-up

Neurology started the patient on dexamethasone for edema and levetiracetam for seizure prophylaxis. The patient was scheduled for a right frontal craniotomy the following week.

Right frontal craniotomy with resection of a 2.5cm x 2.0cm x 1.3cm pink/tan nodule was performed successfully. Biopsy of the

mass revealed a psammomatous-type meningioma. The patient informed his neurologist that his vision was unchanged following the procedure.

Two weeks after the craniotomy, the patient was examined in the eye clinic and exhibited similar diagnostic findings to those obtained at the initial visit. Humphrey visual field testing showed a persistent—but stable—complete superior altitudinal defect in the right eye, with a full field in the left eye.

Three months after his initial visit, the patient's best-corrected visual acuity remained 20/20 O.U. The superior defect in the right eye was stable on confrontation fields testing, as was the 2+ RAPD. Fundus examination showed that the bilateral optic disc edema, hemorrhages and cotton-wool spots had resolved. Additionally, the right optic nerve appeared pale inferiorly while the left optic nerve looked healthy and pink.

The patient was in good spirits and reported an excellent recovery from the craniotomy. Although the condition was stable at this visit, the patient was considered to be at risk for AION in the fellow eye secondary to a small cup-to-disc ratio. We advised the patient to avoid taking antihypertensive medications at bedtime, to continue daily aspirin therapy as prescribed by his primary care provider, and to schedule a follow-up appointment in six months.

Discussion

NAION is an acute ischemic disorder of the optic nerve that occurs at the most anterior portion of the optic nerve.² This painless event is characterized by sudden vision loss and visual field defects that vary in both severity and pattern. It is one of the most common non-reversible

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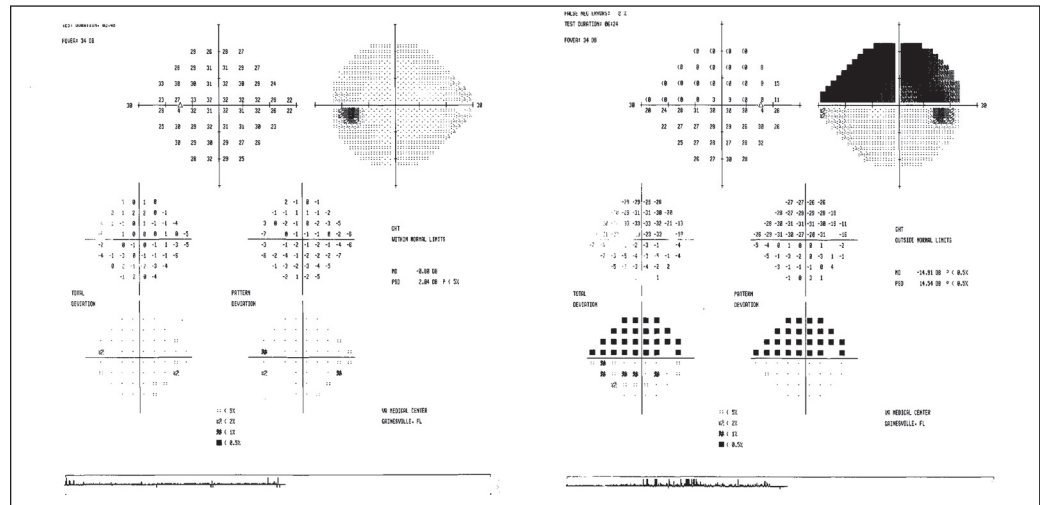


Case Report

causes of vision loss in middle-aged and elderly individuals; in 14.7% of patients, the second eye becomes involved within about five years.^{3,4}

Clinically, patients with NAION present with a visual acuity that ranges from better than 20/20 to no light perception.⁵ Usually, patients also exhibit a RAPD unless the disease is bilateral and symmetric (which is rare). Visual field defects in these patients vary—with superior and inferior arcuates being the most prevalent—and often remain relatively stable after six months.⁶ Initial funduscopy appearance always includes optic disc edema, which may be more prominent in one region (sectoral). Frequently, splinter hemorrhages are seen at the disc margin.⁷ Cotton-wool spots may be present in the peripapillary region of the affected eye. The initial presentation is followed by a gradual transition from optic disc edema to pallor.

• **Risk factors.** Multiple predisposing systemic and ocular risk factors have been identified in the development of NAION. Systemic risk factors include diabetes, collagen vascular diseases, malignant hypertension, hypotension, hyperlipidemia, arteriosclerosis, massive recurrent systemic hemorrhages, migraine and other vasospastic disorders, hematologic disorders, cardiac valvular disease, defective cardiovascular autoregulation, type

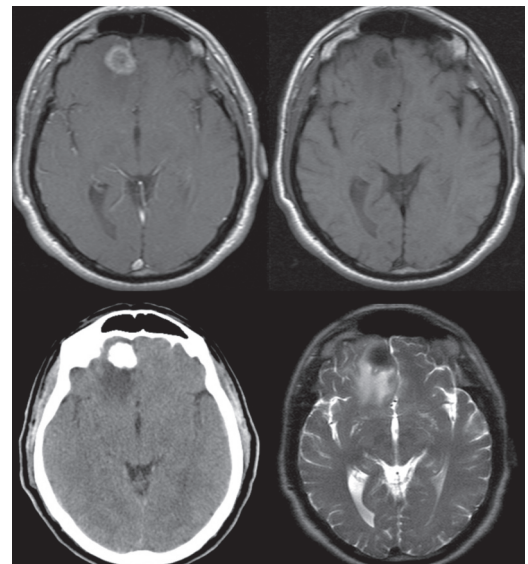


3. Humphrey visual field testing revealed a complete superior altitudinal defect in the right eye and slight blind spot enlargement in the left eye.

A behavior pattern, sleep apnea and internal carotid artery disease.^{6,8-10}

Ocular conditions that have been associated with NAION include an absent or small cup in the optic disc, optic disc drusen, elevated intraocular pressure, marked optic disc edema due to any cause, location of the watershed zone of the posterior ciliary arteries within the optic disc and vascular disorders located in the nutrient vessels of the optic nerve head.¹¹⁻¹⁵

Another cause of NAION is severe blood loss secondary to systemic, non-ocular surgical procedures or significant trauma that results in systemic hypotension and anemia.¹ In such cases, the condition is often referred to as SIAION. Perioperative risk factors for the development of SIAION include prolonged surgical duration, acute systemic hypotension, anemia due to blood loss or prone positioning.¹⁶ Rarely,



4. Axial images of the tumor with different imaging strategies. T1-weighted MRI with Gadolinium (top left). T1-weighted MRI without contrast (top right). Non-contrast CT scan (bottom left). T2-weighted MRI (bottom right).

SIAION may involve both optic nerves, resulting in bilateral vision loss.⁹ Underlying systemic and ocular risk factors may further predispose a patient to the development of SIAION either during or after surgery.

• **Incidence and etiology.** Incidence of SIAION varies with type of systemic surgery and/or surgical location site. One retrospective

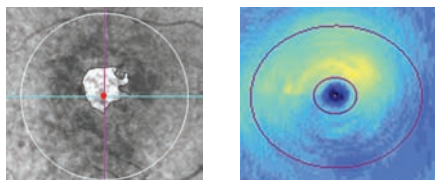
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5. Coronal T2-weighted MRI showing frontal lobe edema and mid-line shift.

study indicated that one in 125,234 non-cardiac surgeries resulted in prolonged vision loss without direct surgical trauma to the orbit or cerebral tissues.¹⁷ This incidence is relatively low when compared to a study of cardiac surgeries in which 22 of 312 patients developed visual field loss or a reduction in visual acuity.¹⁸

NAION is caused by transient nonperfusion or hypoperfusion of the paraoptic branches of the posterior ciliary artery circulation within the optic nerve head.^{19,20} Blood flow within the optic nerve head is dependent upon perfusion pressure (mean blood pressure minus intraocular pressure) divided by the resistance to blood flow. Resistance to blood flow is determined by the state and caliber of the blood vessels as well as the viscosity of the individual's blood.¹⁰

Blood flow within the optic nerve head vessels normally is autoregulated, with a goal of relatively constant blood flow during changes in perfusion pressure.^{15,21} Autoregulation operates over a critical

range of perfusion pressures. If the perfusion pressure falls outside this critical range, autoregulation breaks down and hypoperfusion or transient nonperfusion may result in NAION.¹⁰ Decreased perfusion pressure can be caused by a marked reduction in mean blood pressure secondary to shock, nocturnal hypotension, severe internal carotid artery disease, or ophthalmic artery stenosis or occlusion.²² An

increase in intraocular pressure or a combination of decreased mean blood pressure and increased intraocular pressure also can reduce perfusion pressure.²³

Chronic arterial hypertension is thought to cause derangement of the blood flow autoregulation mechanism. This occurs when the critical range is shifted to a higher level in an attempt to compensate for the higher mean blood pressure. This shift permits sufficient tolerance for hypertension but results in decreased tolerance for hypotension, resulting in an autoregulatory breakdown in the event of sudden blood pressure drop. This, in turn, leads to decreased blood flow to the optic nerve, resulting in ischemia and possible NAION.¹⁵

The aforementioned scenario often occurs during nocturnal arterial hypotension in patients who are using intensive antihypertensive medication, and is thought to be the pathogenesis of the common NAION symptom of vision loss upon waking.¹⁵

Less commonly, massive blood

loss or shock can be the cause of a sudden blood pressure drop.³ A 24% to 46% decrease in postoperative blood pressure from preoperative levels that occurs within 15 minutes to two hours after the surgical procedure has been reported in patients who developed SIAION.²⁴

In the case of massive blood loss, a decrease in hemoglobin creates a lack of oxygen-carrying capacity in the blood, which further compounds ischemia of the optic nerve.⁹ Anemia and low plasma proteins after hemorrhage are thought to create low oncotic pressure, which results in ischemia and vessel wall damage.⁹ A small, retrospective study of six patients with acute blood loss who subsequently developed SIAION showed significant anemia with hemoglobin levels less than 8g/dL (reference range: 13.9g/dL to 18g/dL) from 30 minutes to 72 hours after surgery.²⁴

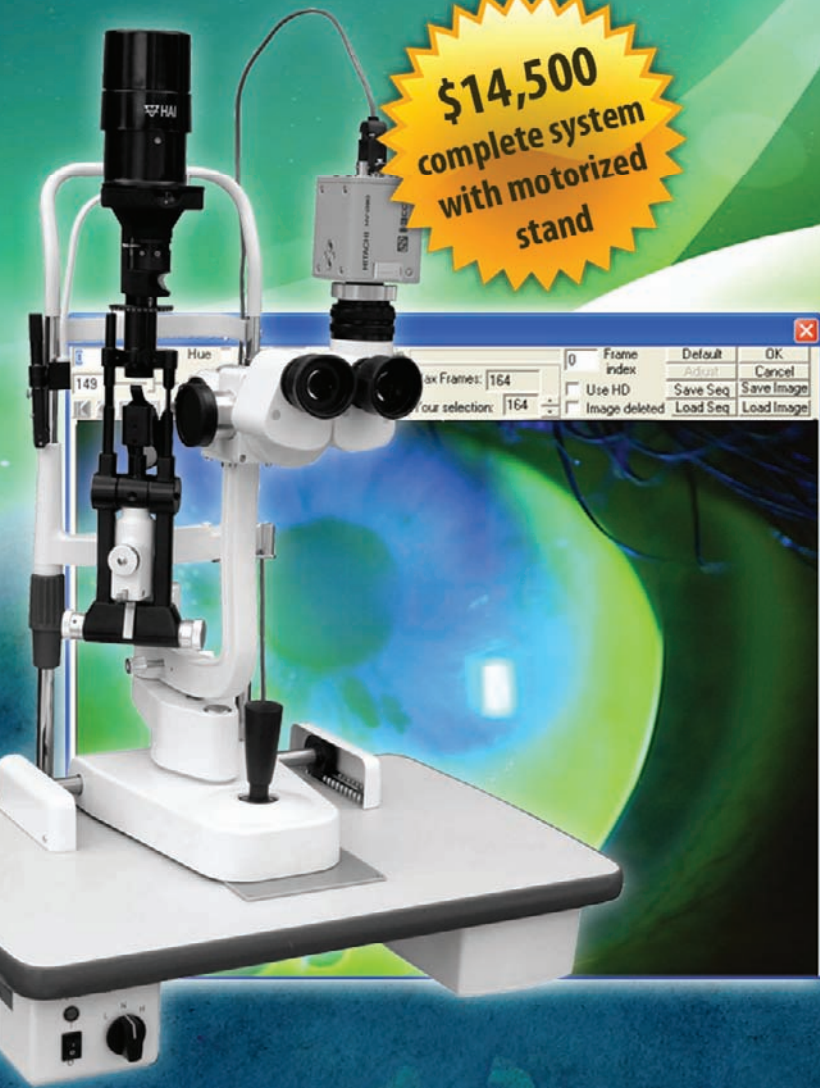
Blood flow resistance can be affected by vascular changes in the small blood vessels within the optic nerve head and/or the arteries that feed optic nerve circulation. These vascular changes may be caused by vasospasm, arteriosclerosis, atherosclerosis, vasculitis, drug-induced vasoconstriction/dilation, or other cardiovascular and systemic diseases.¹⁰ Studies also have shown that swollen axons in the restricted space within the optic disc produce secondary vascular changes by compressing the capillaries and other fine vessels located along the nerve fiber bundles.²⁵

• **Management strategies.** Currently, there is no known treatment for NAION—although many treatments have been recommended. Therapeutic attempts to improve visual acuity or prevent contralateral eye involvement range from noninvasive topical and oral

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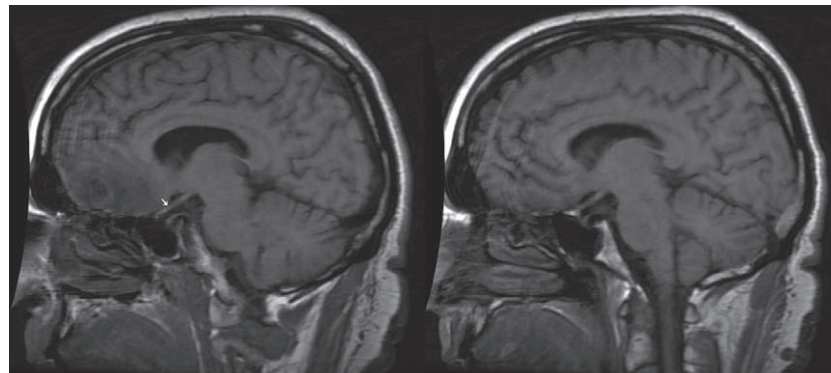
Case Report

therapies to optic nerve decompression surgery. Aspirin therapy, although often suggested to prevent fellow eye involvement, was found to have no effect on the second eye five to eight years after initial occurrence.^{4,26} Brimonidine tartrate also has been studied for its potential neuroprotective effect. However, clinical trials have failed to yield clinical efficacy in humans.²⁷

Even the most well known study related to NAION—the Ischemic Optic Neuropathy Decompression Trial—eventually showed no visual benefit from interventional treatments and was abandoned early due to poor outcomes.⁶ Indeed, subjects in the control group fared better than those in the surgery group.

Another invasive therapy is anti-VEGF injection. In one study of three patients who received an intravitreal ranibizumab injection one to two days after the onset of NAION, the researchers documented a reduction in optic nerve swelling, but no functional improvement.²⁸

Additionally, a case control study of 696 eyes showed promising results when oral corticosteroid therapy was initiated after the onset of NAION.²⁹ Subjects were treated with 80mg of oral prednisone for two weeks, which was then tapered every five days over a two- to three-month period. Oral steroid treatment yielded greater improvement in visual acuity and visual field testing when compared to the control group over a three- to nine-month period. Significant change in visual acuity was quantified as three lines on a Snellen chart (0.3 logMAR). Visual fields improved in 40.1% of the treated group compared to 24.5% of the untreated group. Improvement was more substantial in subjects whose entering



6. Sagittal T1-weighted MRI image (left) shows edema extending posteriorly from the tumor and impinging superiorly on the right optic nerve (see arrow). The left eye shows preservation of the space located superior to the optic nerve.

visual acuities measured 20/70 or worse.^{29,30}

• **Papilledema and Foster-Kennedy syndrome.** Papilledema refers to bilateral optic disc swelling secondary to elevated intracranial pressure. Some of the more common causes of increased intracranial pressure include space-occupying lesions, aqueductal stenosis that produces hydrocephalus, pseudotumor cerebri, subdural or epidural hematomas, subarachnoid hemorrhage, arteriovenous malformations, meningitis, encephalitis and intracranial venous sinus thrombosis.³¹

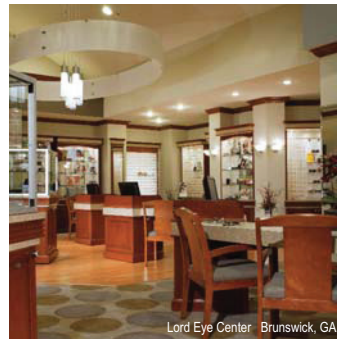
In our patient's case, a space-occupying psammomatous meningioma was suspected to be causing increased intracranial pressure, which led to disc swelling in the left eye. This finding would also cause the symptom of flashing lights with no acute vision loss (as in the right eye), as well as the presence of an enlarged blind spot in the left visual field. This type of benign meningioma is classified by dense epithelial clusters of tumor cells that form numerous corpuscles (which resemble the layers of an onion), leading to calcification.³²

Although the pathogenesis of papilledema is still unclear, multiple

theories have been suggested. These include, but are not limited to, direct infiltration of the optic nerve head by cerebrospinal fluid, turbulence of prelaminar optic nerve tissue, glial edema, disturbance of hydrostatic pressure in the nerve tissue and blood stream, and compression of the central retinal vein with elevated venous pressure.³³⁻³⁷ However, it is clear that axoplasmic transport is impaired with papilledema.³⁸ As with all forms of optic disc swelling, there is blockage of both slow and fast axoplasmic transport, with subsequent axoplasmic backflow.³⁹

Optic disc swelling occurs in steps, with the earliest sign being blurring of the disc margin due to swelling of the axons in the peripapillary nerve fiber layer.^{13,40} Blurring begins in the lower pole, then includes the upper pole and nasal border, with the temporal margin blurring last.⁴¹ Hyperemia occurs next, and is caused by dilation of the capillaries located within the disc.^{40,41} Also, loss of spontaneous venous pulse (if present) occurs at this stage.⁴² Further, disc elevation is seen in advanced cases of disc swelling, starting in the periphery and moving centrally.⁴³ Other common features of papilledema

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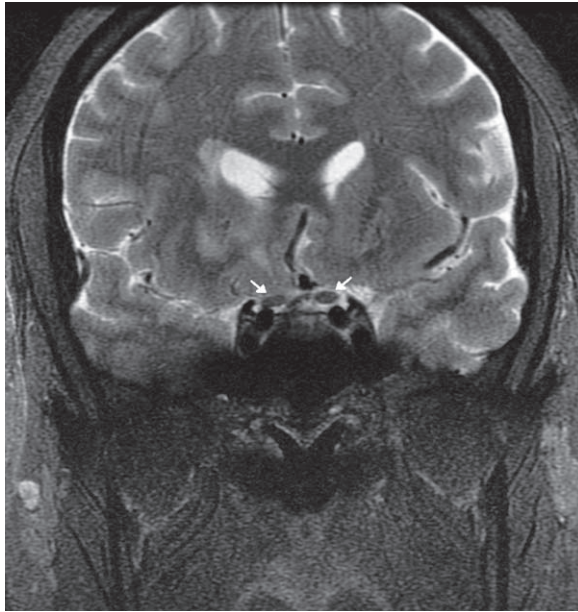
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7. Coronal T2-weighted MRI showing the right optic nerve slightly compressed/distorted due to mass effect from the edema surrounding and extending from the tumor (see arrows).

include radially oriented splinter hemorrhages in the peripapillary nerve fiber layer, exudates and cotton-wool spots.⁴³

Papilledema typically does not result in vision loss in its early stages. However, if left untreated, visual field defects may occur—with enlargement of the physiological blind spot being the earliest (and often the only) defect. The defects can progress, and eventually might lead to severe visual field constriction or central vision loss.⁴³ Transient visual obscurations, including photopsia, have also been reported by patients with papilledema.^{44,45} The cause and clinical significance of this phenomenon remains unknown.⁴⁶

Our patient reported this unusual symptom on presentation, specifically noticing the flashing lights when he had his eyes closed. Although uncommon, superimposed ischemic optic neuropathy can result from severely swollen nerves. This particular phenomenon

has been seen in patients with underlying papilledema and hypertension who subsequently suffered acute hypotension.⁴⁷ This might further explain our patient's clinical presentation.⁴⁷

No discussion on papilledema would be complete without mention of Foster-Kennedy syndrome (FKS)—a condition that is defined by the presence of papilledema in one eye and optic disc atrophy in the fellow eye secondary to an intracranial mass.

The incidence of FKS has been shown to be less than 1% in conjunction with intracranial tumors.⁴⁸

When a patient presents with these clinical signs in the absence of a compressive tumor, the condition is often termed pseudo-Foster-Kennedy syndrome (PFKS). Mimicking conditions include bilateral non-simultaneous optic neuritis or ischemic optic neuropathy, papillitis with an old optic neuropathy, and papilledema with an old optic neuropathy.⁴⁹

However, our patient experienced what appeared to be AION in one eye and optic disc swelling in the fellow eye—a presentation inconsistent with either FKS or PFKS.

SIAION is a rare but potentially visually devastating consequence of surgical or traumatic blood loss that may be exacerbated by an underlying systemic disease. In this case, our middle-aged patient

with pre-existing hypertension and hyperlipidemia underwent a complicated, non-ocular surgical procedure and experienced significant blood loss that resulted in systemic hypotension and anemia. As a result, he developed an acute and permanent loss of vision in his right eye due to SIAION. Unknown to the patient or the medical professionals who were initially involved in his care, he also had a right frontal lobe meningioma.

Upon initial fundus examination, we observed bilateral asymmetric disc edema with splinter hemorrhages and cotton-wool spots. The disc edema in the non-symptomatic eye led to the discovery of the meningioma on MRI. We believe that the edema surrounding the frontal lobe meningioma likely played a role in predisposing this patient to NAION in the right eye, as there is evidence that axonal swelling from optic disc edema caused increased blood flow resistance that led to further ischemia.

Whether the optic disc swelling in the fellow eye represented papilledema or atypical/mild SIAION remains unknown. The lack of acute SIAION symptomatology, the perception of light flashes and the presence of an enlarged blind spot on visual field testing suggest papilledema as the likely cause of optic disc swelling in the left eye. This finding ultimately led to further investigation and subsequent discovery of the meningioma.

Regardless of the role papilledema may have played, this case taught us that patients can—and sometimes do—suffer simultaneous, coincidental ocular disorders. Even though SIAION was the obvious suspect in our patient, this case reminds us that every patient with bilateral disc edema requires neuroimaging. ■

Dr. Perkins is a staff optometrist at The Villages VA Outpatient Clinic in Florida. Dr. Marcus-Freeman is a staff optometrist and optometry residency coordinator at the Malcom Randall VA Medical Center.

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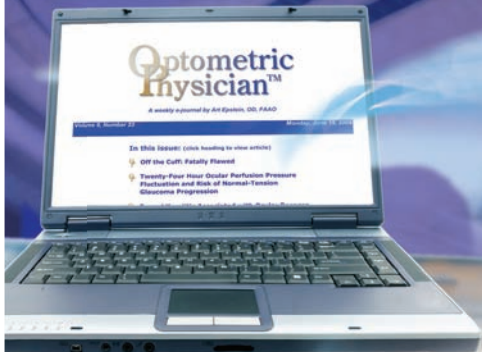
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PAULA R. NEWSOME, OD, FAAO

Building a Thriving Practice

How daily disposable lenses can help your practice grow

More optometrists are moving toward daily disposable lenses and learning that what's healthier for their patients is also healthier for their practice.

Creating a daily disposable practice is achievable

Transforming your contact lens practice to focus on daily disposable lenses can seem like a daunting prospect, but as Paula Newsome, OD and President of Advantage Vision Center in Charlotte, NC, can attest, the conversion was simple: "When we decided to actively engage in daily disposables, we first looked at who we thought could most benefit from the lens. Then we started seeing the health benefits for all our patients. From a health perspective, putting on a fresh lens every day is the absolute best option for our patients." With health as a starting point, every decision made thereafter became easier.

Address the "cost" concern

Dr Newsome admits that she was initially concerned with how patients would react to the cost: "I was thinking in my mind that it was very cost prohibitive." What she found, however, was quite different. She discovered that the leading daily disposable, 1-DAY ACUVUE® MOIST® Brand Contact Lenses, cost less than a dollar per eye, per day. "I think that's a very reasonable amount if you're talking about somebody's sight," she said. And when she presents the health benefits of daily disposables up front, her patients agree that their eye health and their vision are worth the cost.

Create a competitive advantage

Dr Newsome has seen that giving patients the option of daily disposable lenses has given her a competitive advantage: "Promoting daily disposable contact lenses has been a win-win for our practice. The experience my patients have in the lenses has certainly made our practice stand out in the market." A benefit to standing out in a competitive market is building a reputation and increasing word-of-mouth referrals. Increased referrals are not only a benefit to her practice, but an assessment of her care and a true indicator of happy patients. "Patients are excited, enthusiastic, and they

go back to their work and share the good news with others," she says. To date, there has been a 20 percent growth in her daily disposable contact lens business from the same time last year.

Advice to fellow ODs

Dr Newsome has some simple advice for fellow ODs who are considering building a daily disposable practice.

- I. Engage your staff;** as they have a lot of contact with patients, it is important to get them involved in the process
- II. Invite a sales rep** to conduct a lunch and learn to educate the staff
- III. Utilize point-of-purchase and educational materials** to support daily disposables

"I would tell fellow ODs to make sure you include daily disposable lenses in your armamentarium," suggests Dr Newsome. Daily disposable contact lenses are optimal for patients for a number of reasons, including health, convenience, and compliance. There are many benefits to a practice as well. Daily disposable patients have better recall and are less likely to go to other places to purchase contact lenses, according to Dr Newsome.

"I talk to all of my contact lens patients about daily disposables because I believe in it that strongly." Her philosophy is "change is good" and, in her practice, it is paying off. Dr Newsome concludes, "The health benefit that we provide for patients, in addition to the economic benefit to the practice, is a win-win for everyone." ■

Paula R. Newsome, OD, is President of the Advantage Vision Center in Charlotte, NC. She received her doctorate in optometry from the University of Alabama at Birmingham. Dr Newsome is a member of the North Carolina Optometric Society and the American Optometric Society. She is also actively involved in the National Optometric Society, the Piedmont Optometric Society, the Charlotte Medical Society, and is a Fellow of the American Academy of Optometry.

Dr Newsome is a member of numerous associations and serves as a speaker and professional consultant for several medical companies, including VISTAKON® Division of Johnson & Johnson Vision Care, Inc. She was compensated for this article.

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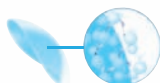
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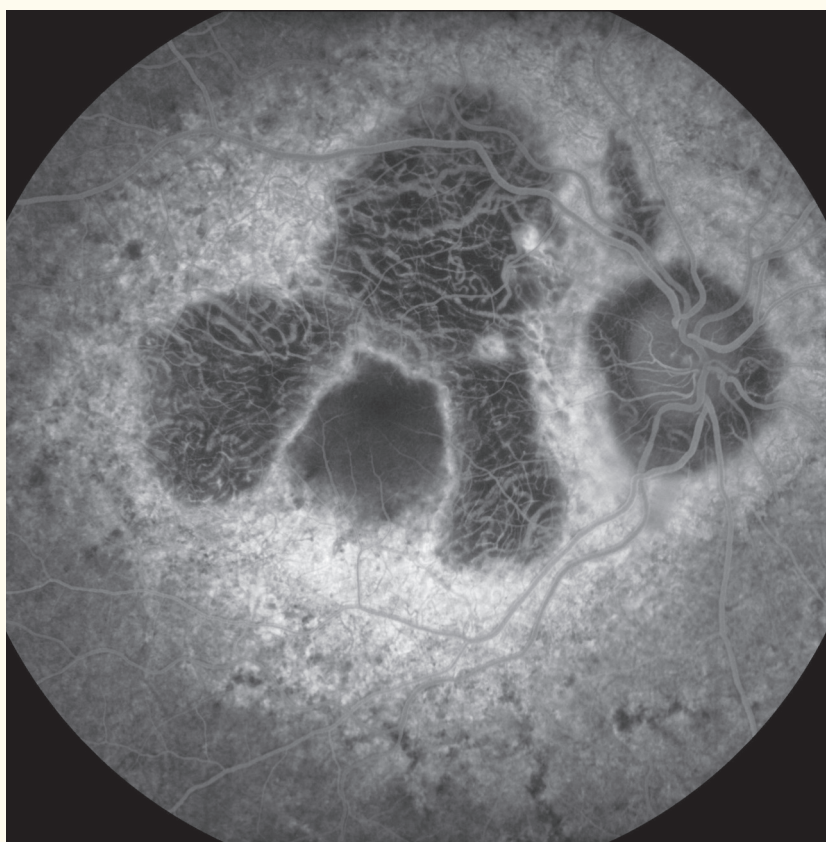
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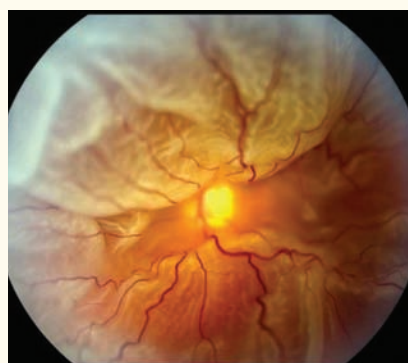
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My Favorite Retinal Photos

An ophthalmic photographer handpicks his favorite photos of retinal diagnoses. Have you seen many of these? **By Jason Calhoun**



Alpha-methylacyl-CoA racemase deficiency.



360-degree retinal detachment.

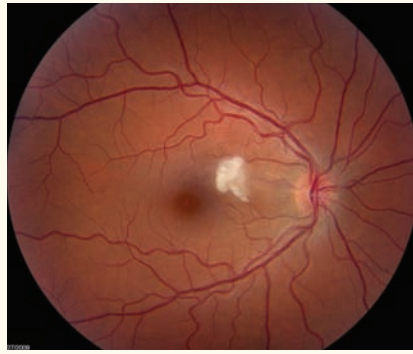


Best disease, also called vitelliform macular dystrophy.

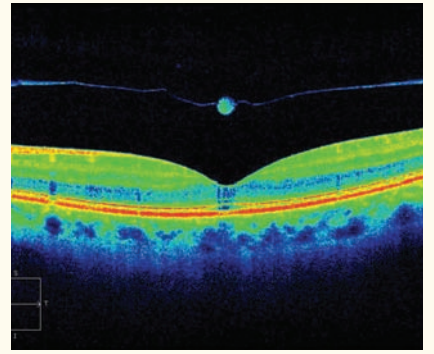
Mr. Calhoun is an ophthalmic photographer/technician at the Mayo Clinic in Jacksonville, Fla.



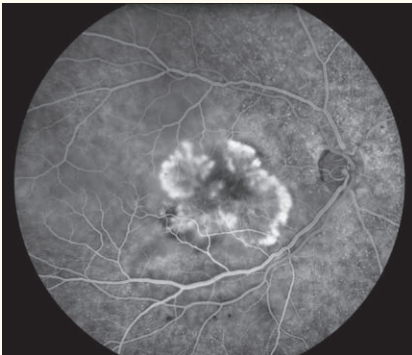
Central retinal artery occlusion.



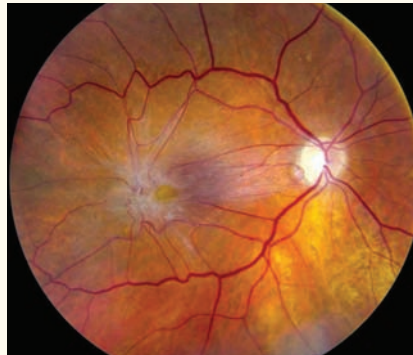
Cotton-wool spot.



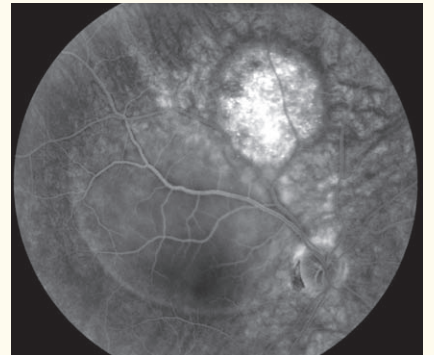
HD-OCT of posterior vitreous detachment with operculum.



Wet age-related macular degeneration.



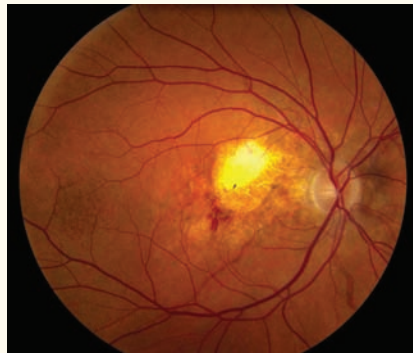
Macular pucker.



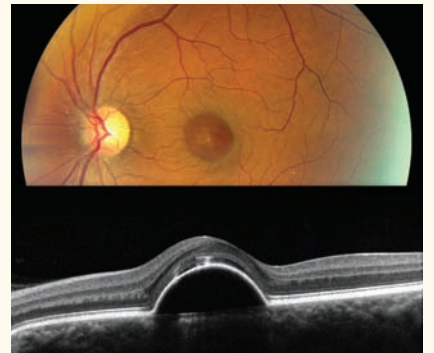
Melanoma with subretinal fluid.



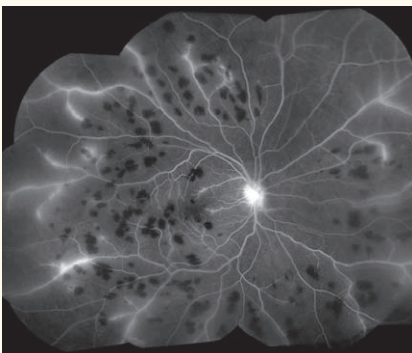
Myelinated nerve fiber layer.



Pseudoxanthoma elasticum with choroidal neovascularization.



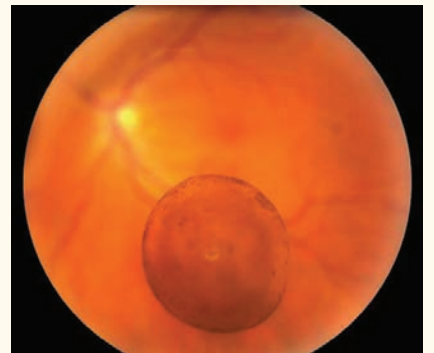
Retinal pigment epithelial detachment.



Fluorescein angiogram of retinal vasculitis due to lupus.



Subhyaloid hemorrhage.



Vitreous cyst.

16 Essential Low Vision Tips and Tricks for Every O.D.

Low vision is not just for specialists. Here are a number of easy ways you can help these visually impaired patients. **By Cheryl G. Murphy, O.D., Contributing Editor**

Every eye doctor should be equipped with the basic items and knowledge to help a low vision patient in need in the office. Here is a list of simple tips, tools and techniques you can put into practice right away.

Diagnostic Items for Doctors

- *Trial lens frame and lenses.*

“Giving someone a high add that is only trialed in the phoropter is a big risk. You would not be giving the patient a ‘real world’ feel for where they would have to hold the reading material to get it to the right focal distance,” says Kristin Protosow, O.D., a state-certified low vision specialist, at Eye Vision Associates in Ronkonkoma, N.Y. So, be sure your office has a trial lens frame set, she says.

“Also, trial framing the near prescription allows you to test lighting conditions, tints, magnifiers and other devices used in conjunction with the appropriate reading add, and it allows for eccentric viewing,” she says.

- *Low vision acuity chart*

(Designs for Vision/Feinbloom chart or a Lea Numbers Low



Not every O.D. needs to be a low vision specialist, but every O.D.'s office should be equipped with the basics, such as a trial frame set, says Kristin Protosow, O.D.

Vision chart). “It’s important to be able to get an accurate measure of acuity on a low vision patient, as it will help you determine how much magnification the patient might need. And the testing can be done at a closer distance, without the patient leaving the exam chair,” says Michael Fischer, O.D., chief of optometry service at the Northport Veterans Affairs Medical Center, in Northport, N.Y., and a consulting low vision clinician at Lighthouse International.

“Additionally, it can make the initial testing a more positive experience

for low vision patients with significantly reduced acuity—they might only see the big E on your projector chart, but they will be able to read many more targets on a low vision chart,” he says.

- *Mars contrast sensitivity test.*

“Contrast sensitivity function can be a much better predictor of visual function than high-contrast letter charts,” says Kevin Houston, O.D., a low vision expert who works in the vision rehabilitation laboratory at the Schepens Eye Research Institute at Massachusetts Eye and Ear. “I use the Mars test, which is held

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Low Vision

by hand at near and can easily be administered by a technician.”

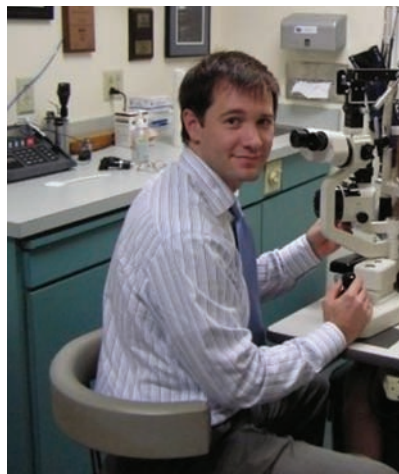
- **Minnesota reading card.** “A continuous text reading card, such as the MN Read, will often pick up problems not evident with traditional single letter testing,” Dr. Houston says.

Simple Suggestions for Patients

- **Tactile stickers.** “Adding tactile stickers to appliances and devices can assist low vision patients in using their washing machine or microwave with ease by feeling the markers,” Dr. Protosow says. Touch Dots are an example of these stickers, and can be purchased online.

- **Improve lighting and contrast in the home.** “Good lighting that is specific to the task is important,” Dr. Protosow says. “Many people like the new LED lights on the handheld magnifiers and full-spectrum lights for reading because they are bright and give even illumination.”

Even simple, direct lighting with



Low vision rehabilitation involves more than providing a few low vision devices. Encourage patients to join a support group, says Kevin Houston, O.D.

a gooseneck lamp will help patients with low vision, Dr. Houston says.

- **Talking watches and clocks.**

“Even when patients really want to use their vision as much as possible, when they are tired or have a bad day, it can be too taxing. Sometimes the simplest device, like a talking watch, can really help,” Dr. Protosow says.

- **Enhance contrast at the dinner table.** “Different color plates, cups, placemats, etc., can provide a lot of help to a low vision patient,” Dr. Fischer says. For example, “don’t put the coffee in a brown mug—put it in a white mug so you can see the level as you pour. Have your white meat chicken with rice on a dark plate so you can see it, and put it on a white placemat so you see the edges of the plate,” he says.

- **Recommend large-print devices.** “Encourage patients to use large-number clocks and watches, big-button phones, large-print checks, and the like,” Dr. Fischer says.

- **Join a support group,** Dr. Houston says. Visit www.macular.org/sgroups or www.earsforeyes.info/ears/tblStateslist.php to recommend one in your area.

Prescribe Helpful Devices

“If you have a vision rehab clinic nearby, I suggest referring patients if they have complaints, or if their better eye has best-corrected vision less than 20/30 or has visual field constriction beyond 50°,” Dr. Houston says. (See “Criteria for Low Vision Referral,” at left.)

But if you don’t have a low vision clinic nearby—or even if you do—all optometrists should be comfortable providing “entry level” low vision rehabilitation care. This includes:

- **Adds greater than +2.50.** “The combination of a gooseneck lamp and a +3.50 add can solve many problems,” Dr. Houston says.

Dr. Fischer advises, “In reality, you don’t necessarily need anything special in your office to test patients with higher adds. You can use your trial lens set. Just don’t be afraid to test higher adds—that may be all some patients need. Don’t forget to calculate the

Criteria for Low Vision Referral

Refer to a low vision specialist if:

- The patient complains about visual function and it is not likely to improve in the next three months.
- The patient’s best-corrected acuity is worse than 20/30 in the better eye and is not likely to improve in the next three months.
- The patient’s visual field in the better eye is 50° or less.
- The patient has any visual impairment and asks you to fill out a driver’s form, or asks you if driving is OK.

“I have traditionally asked my referring docs to refer using these criteria, while giving special attention to referring patients with mildly reduced best-corrected acuity (20/30 to 20/50),” Dr. Houston says. “These patients have the most urgent need because they are typically in the early stages of vision loss and are in danger of losing function and independence.”

He prefers that the primary care optometrist continues to do the routine exam with dilation but sends the patient to him annually for a low vision device update. “In this way, we’re working as a team, each delivering the service we are best equipped to provide,” he says.

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Low vision patients don't necessarily need sophisticated, high-tech equipment. For example, a simple telescope can be a big help to see street signs, says optometrist Michael Fischer (on right) with a low vision resident and patient.

proper working distance for the add you are testing and make sure the patient holds the print at that distance.”

- **Handheld magnifiers.** “If your patient has very reduced acuity and needs a lot of magnification, you might feel more comfortable referring to a low vision practitioner. But for patients with acuities better than 20/200, you can test hand magnifiers that are not that difficult for the patient to use and help them quite a bit,” Dr. Fischer says. “Have some magnifiers with lights—they can be a big help in restaurants and other places where the lighting is not the best.”

- **Telescopes.** “Again, you don't

have to do anything sophisticated to help some of your patients with low vision,” Dr. Fischer says. “A simple telescope to see signs in the supermarket or on the street can be a big help. Shoot for an acuity in the 20/40 to 20/50 range through the telescope. It won't always be the answer, especially if the patient has poor contrast, but it might help.”

- **Filters/tints.** “Sunglasses in a contrast-enhancing tint, such as brown or amber, are useful as long as the patient's green color perception is not desaturated beyond what is needed for driving,” Dr. Houston says.

Cutting-edge Technologies and Gadgets

Recent advances in technology can enhance the lives of low vision patients.

- **E-readers (Kindle) and Tablets (iPad).** “The Kindle and the iPad have been great tools for my patients because of their relatively large screen size and reading- and contrast-enhancing functions,” Dr. Houston says.

The iPad (as well as the latest iPhone and other Apple products) incorporates a number of accessibility features for visually impaired users, including Zoom and VoiceOver, Dr. Fischer adds.

Smartphones, on the other hand, are visually demanding and require a visual acuity of 20/20 to 20/40 to be fully operational, even with magnification tools, Dr. Houston says. “So, ask your visually-impaired patients if their vision prevents them from using or purchasing a smartphone. Handheld magnifiers are cumbersome to use with a phone or tablet and something spec-mounted, such as a loupe or high add, is typically needed,” he says.

- **GPS with voice commands.** “For driving and mobility, the voice-guided GPS systems have helped to improve patients' safety. My bioptic drivers who have GPS are less dependent on their telescope for reading signs, which frees them up to concentrate more on vehicle control and braking,” Dr. Houston says.

Also, O.D.s should “become familiar with some of the accessibility features incorporated into the operating systems of PCs and Macs, as well as the options built into web browsers,” Dr. Fischer says. “Most browsers allow the user to magnify a web page simply by holding down the control key and hitting the plus key repeatedly until the page is large enough to see.”

Seek the assistance and expertise of a true low vision specialist early on, say Drs. Protosow, Fischer and Houston. Low vision specialists are often very willing to comanage with the referring optometrist, they say. (See “Find a Low Vision Specialist,” at left.)

In short, providing low vision doesn't have to be complicated, tedious or time consuming. Do what is best for these patients to provide them with optimal visual function in their everyday lives, and the sooner the better. ■

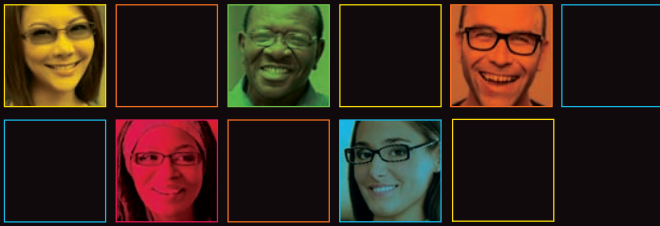
Find a Low Vision Specialist

To find a low vision specialist in your area, contact:

- American Academy of Optometry Fellows and Diplomates in Low Vision, www.aaopt.org/section/lv/diplomates.

- American Optometric Association Vision Rehabilitation Section. Or use the AOA's Dr. Locator search, www.aoa.org/x5428.xml, and choose “low vision rehabilitation” under the “Practice Emphasis” pull-down option.

Also, a retina specialist in your area is often able to give a recommendation for low vision rehabilitation.



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Adult Vitelliform Maculopathy

Management: From Anti-VEGF to Low Vision

An 80-year-old patient has a gradual and asymmetrical decrease in vision. He was referred from the retina clinic to the low vision specialist.

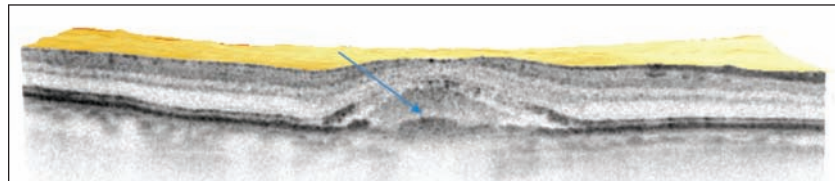
By Virginia Monteith Hodges, O.D., and Wendy Marie McGonigal, O.D.

Adult-onset vitelliform maculopathy initially presents in adulthood as bilateral subtle and often asymptomatic macular lesions, which are often misdiagnosed as early age-related macular degeneration (AMD). Although the prognosis is generally good with this condition, in some cases, retinal findings may progress to visually debilitating and blinding retinal pigment epithelium (RPE) disruption. So, in advanced cases, standard of care management should include a referral to a low vision specialist, who can improve visual function and increase quality of life for affected patients.

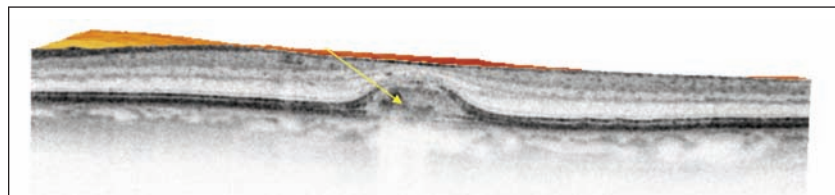
This case report offers a comprehensive overview of the medical intervention and low vision management of an elderly white male with complaints of a gradual and asymmetrical decrease in vision who was subsequently diagnosed with adult-onset vitelliform maculopathy.

History

An 80-year-old white male presented to our eye clinic describing a gradual decrease in vision in both eyes. He had been followed for 11 years for primary open-angle



A large, well-demarcated vitelliform lesion with underlying cuticular drusen was visible, along with sub- and intra-retinal pigment epithelium fluid accumulation, in the right eye.



The vitelliform lesion in the left eye is smaller than that in the right and is without fluid accumulation. Cuticular drusen is visible within the area of RPE disruption.

glaucoma in his right eye only, as well as AMD, pseudophakia, dry eye syndrome and chronic allergic conjunctivitis in both eyes. Previous examinations were negative for diabetic or hypertensive retinopathy.

Current ocular medications included dorzolamide hydrochloride b.i.d., timolol maleate q.d. and brimonidine tartrate b.i.d., all O.D. only for treatment of his glaucoma, as well as preservative-free artificial tears q.i.d. O.U. and Zaditor (ketotifen fumarate, Alcon) b.i.d. O.U. for treatment of dry eyes and aller-

gic conjunctivitis.

His systemic history was positive for insulin-dependent diabetes mellitus, hypertension and hypercholesterolemia with carotid artery disease.

Systemic medications included low-dose aspirin (81mg), clonidine hydrochloride, simvastatin, Lantus (insulin glargine, Sanofi-Aventis) and Humalog (insulin lispro, Eli Lilly) injections, lisinopril, metoprolol succinate, and nitroglycerin. The patient had no known drug allergies.

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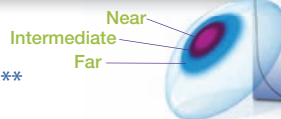
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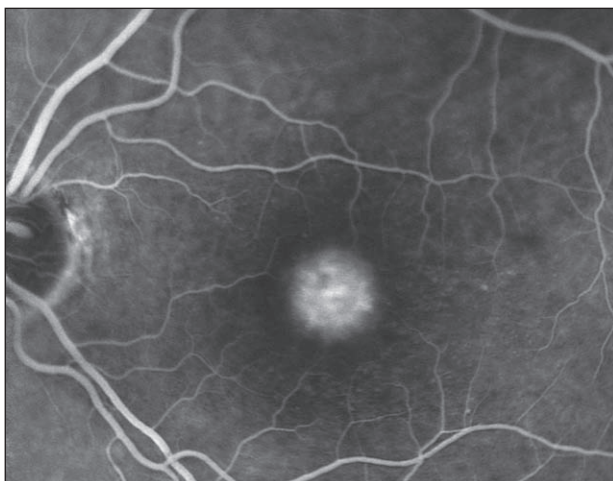
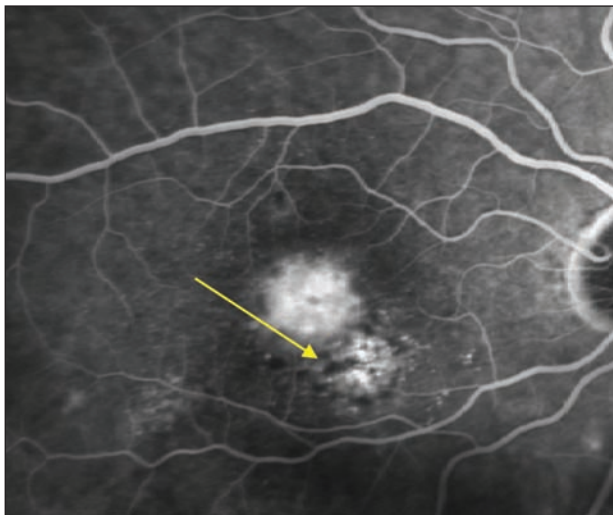
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Fluorescein angiography revealed central staining O.U., with an occult choroidal neovascular membrane inferior to the macula in the right eye and no leakage or membrane noted in the left.

Diagnostic Data

The patient initially presented in May 2000 because he had lost his habitual spectacles. Uncorrected visual acuity was 20/60 O.D. and 20/50 O.S. Best-corrected visual acuity improved to 20/20⁻² O.D. and O.S. with a thorough refraction. Dilated fundus evaluation was unremarkable except for the presence of increased macular pigmentation and fine drusen, which were diagnosed as early dry AMD.

Subsequent evaluations revealed progressive RPE dropout, clumping and drusen. The patient experienced a corresponding vision loss, O.D. greater than O.S.

Nine years later, the patient's best-corrected visual acuity had progressed to 20/50⁻¹ O.D. and O.S. External examination, including extraocular motilities and

confrontation fields, was normal with no afferent pupillary defect. Biomicroscopy found normal and healthy anterior segment structures. Goldmann appplanation pressures measured 20mm Hg O.D. and 18mm Hg O.S. Dilated fundus evaluation revealed large, bilateral choroidal lesions with overlying pigment epithelial detachments, O.D. greater than O.S.

Optical coherence tomography (OCT) scans revealed bilateral, dense choroidal elevations with questionable fluid accumulation in the right eye, irregular reflectivity in the left eye, and possible choroidal neovascular membranes in both eyes.

We referred the patient to a retinal specialist for further evaluation. At that visit, Spectralis OCT (Heidelberg Engineering) scans of the macula revealed vitelliform lesions in both eyes, with subretinal fluid only in the right eye.

Diagnosis

After a thorough review of past macular OCT scans and fluorescein angiography studies, it appeared that bilateral and well-demarcated vitelliform lesions had been present as far back as December 2010.

The patient was formally diagnosed with adult-onset vitelliform maculopathy (AOVM) in both eyes, with an active choroidal neovascular membrane in the right eye only.

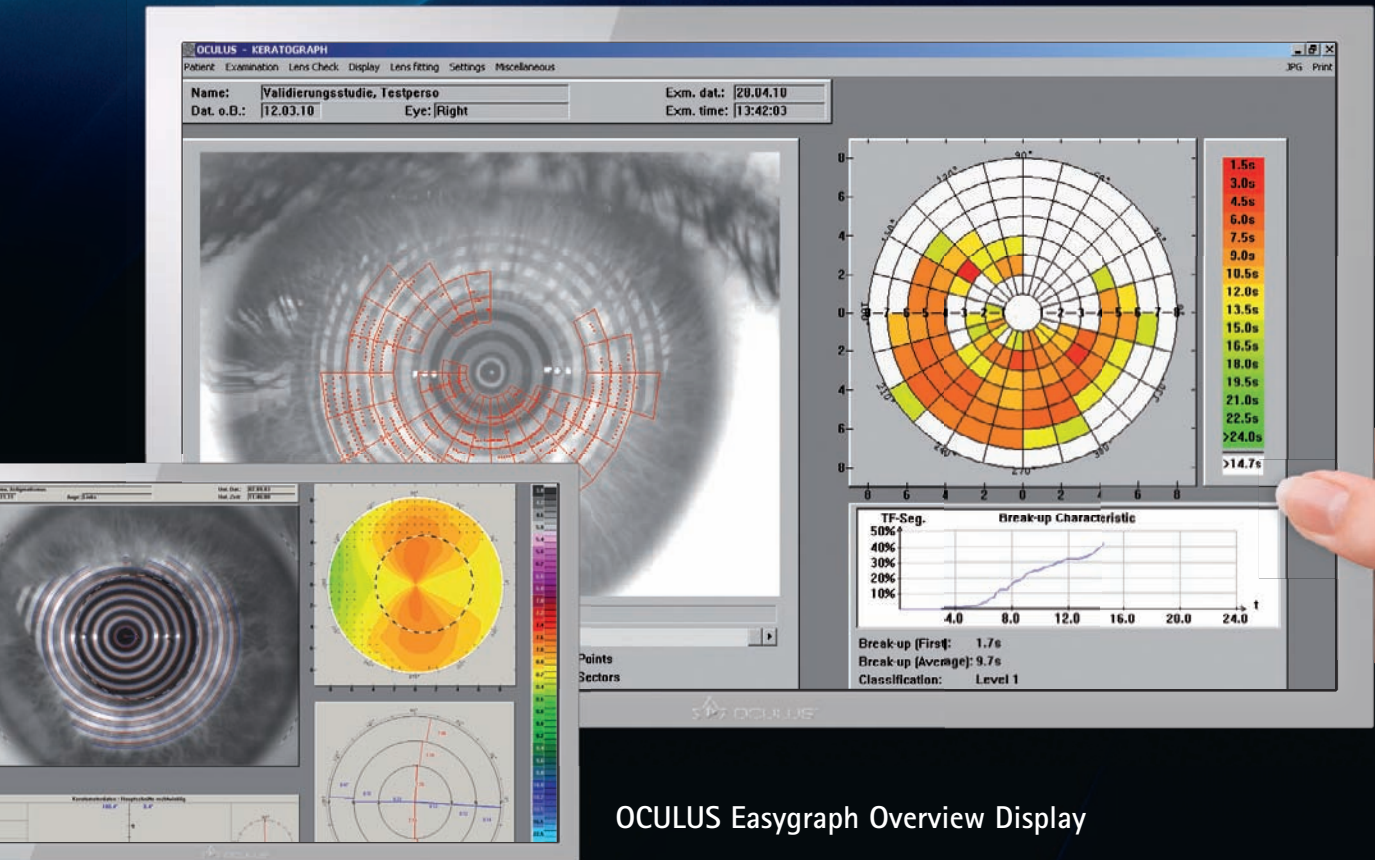
Treatment and Follow-up

Due to the choroidal neovascular membrane in the right eye, the patient received approximately 20 monthly intravitreal injections of either Avastin (bevacizumab, Genentech/Roche) or Lucentis (ranibizumab, Genentech/Roche) over the course of two years. Residual RPE disruption resulted in decreased visual acuity, central scotomas and metamorphopsias in both eyes, which affected the patient's quality of life. At this point, the patient's visual acuity was 20/100⁻² O.D. and 20/50⁻² O.S. with no improvement on pinhole. This prompted a referral from the retina clinic to the low vision specialist.

Low Vision Evaluation

The patient's entering complaint for the low vision examination was decreased vision at near, with the right eye affected more than the left. Goals for the patient were to increase his ability to read newspaper print and to be able to read words on his 32-inch television screen.

At this visit, the patient's best-corrected distance visual acuity through trial frame refraction was 20/100



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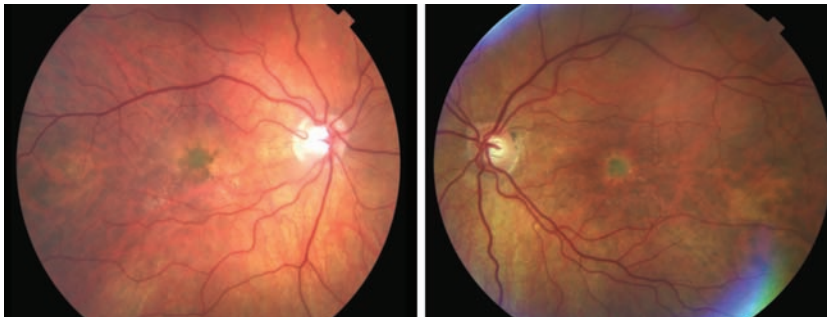
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Fundus evaluation reveals subtle and irregular vitelliform lesions after three years of intravitreal injections in the right eye (on left) and observation in the left (on right).

O.D. with eccentric viewing at 3 o'clock, and 20/30 centrally O.S. without eccentric viewing. The patient was able to read 0.6m optotype at 26cm through a +3.00D add over the manifest without rivalry. But, binocular arc perimetry visual field testing revealed a paracentral 18-degree scotoma as well as decreased contrast sensitivity in both eyes.

After a detailed examination, the patient was prescribed near spectacles with a safety frame, polycarbonate lenses and anti-reflective coatings. Yellow slip-in filters were given to the patient for use when reading to increase contrast. The patient was also given a pocket typoscope to decrease lateral masking, and new spectacles were ordered. Also, amber-filtered lenses were ordered for outdoor use to lessen glare and photophobia. MaxTV telescopic glasses (Eschenbach Optik) enhanced the patient's distance acuity; these were recommended for use when the patient was stationary and for viewing distant objects such as the television. No further referral for orientation and mobility training was recommended.

The devices were mailed to the patient's home, and he was instructed to return to the low vision clinic as needed. He was also scheduled to continue care with the

retinal specialist for ongoing medical management.

Discussion

Adult-onset vitelliform maculopathy, also known as adult foveomacular vitelliform dystrophy or late-onset Best's disease, is a rare condition with onset between the fourth and fifth decades of life, and mostly affecting females.¹⁻³ The patient classically presents with bilateral, round and discrete yellow lesions that are in or near the macula, which result in disturbances in central vision.^{1,2} The disease rarely progresses to development of choroidal neovascular membranes or areas of geographic atrophy and concurrent cuticular drusen.¹

AOVM has been controversially theorized to be a variant of Best's disease and is often categorized as a pattern dystrophy.^{4,5} Several hypotheses exist for the origin of the characteristic yellow lesions, including the theory that they represent melanin and lipofuscin-laden macrophages or are the result of a proliferation of RPE cells or sub-retinal fibrin.⁶

In AOVM, fluorescein angiography (FA) results vary with disease progression. Early changes appear as central hyper-fluorescent staining due to RPE disruption.⁷ More developed disease shows a "corona sign" or central hypo-fluorescent

atrophic lesion with surrounding hyperfluorescent halos due to loss of photoreceptor outer segments.⁷ Vitelliform lesions tend to stain late.¹ Other diagnostic findings include an electro-oculogram (EOG) and electroretinogram (ERG) that are relatively normal and mild tritan color vision defects.^{1,3}

The primary differential diagnoses for AOVM include Best's disease and AMD.⁸ Best's disease is genetically similar to AOVM, but Best's disease has a characteristically larger lesion size than seen in AOVM and presents with an earlier age of onset, abnormal EOG and a poorer visual prognosis.^{6,8,9}

AOVM is often misdiagnosed as AMD because macular degeneration occurs much more commonly than AOVM.⁸ Differentiation is possible due to the later onset of macular degeneration as well as with macular OCT analysis.⁸ Exudative macular degeneration shows a characteristic and irregular intra- and subretinal serous shadowing with fibrovascular changes.³ Alternatively, AOVM lesions have discrete and well-demarcated "hills" of subfoveal disruption with no optical shadowing effects.³

Current medical management of AOVM has been disappointing. Verteporfin photodynamic therapy (PDT) has been attempted unsuccessfully.³ Monthly intravitreal injections of the anti-vascular endothelial growth factor (anti-VEGF) medications Lucentis and Avastin have been shown to produce short-term visual improvement.³ The FDA recently approved the use of another anti-VEGF intravitreal injection known as Eylea (aflibercept, Regeneron).¹⁰ The pivotal aspect of this medication is that it is used only once per month for three months, then once every two

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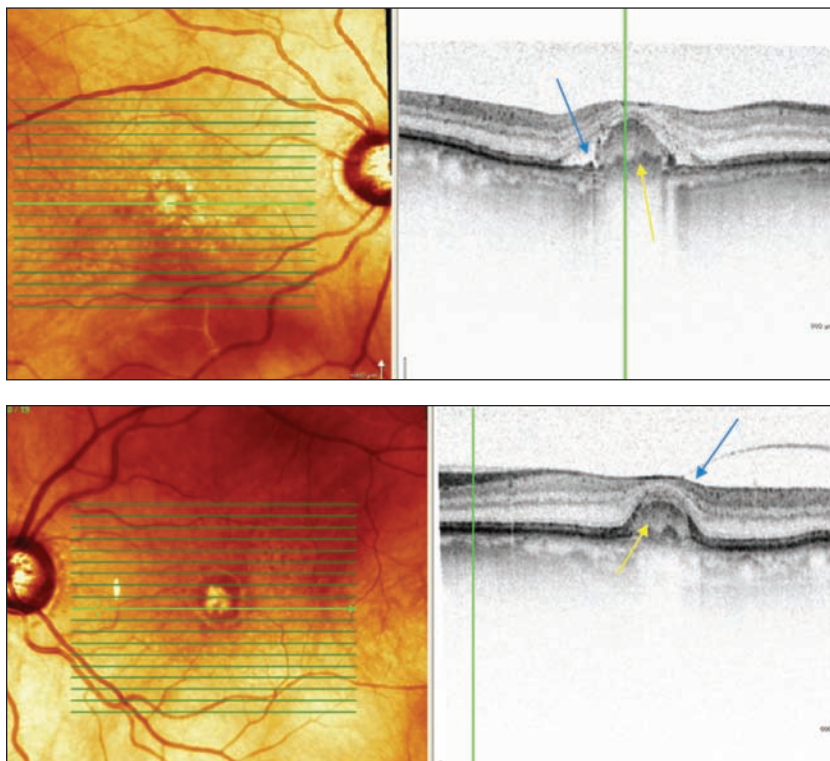
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Case Report



Spectralis OCT scans of the vitelliform lesions: Persistent subretinal fluid can be noted in the right eye (top), with a partial posterior hyaloid detachment appreciated in the left (bottom), and cuticular drusen confirmed in both eyes.

months thereafter.¹⁰ This requires fewer injections per year than its monthly-injection predecessors, Lucentis and Avastin.¹⁰

In milder presentations, visual prognosis for AOVN is typically good because the lesion lies in the RPE and does not involve the sensory retina.^{1,2} Prognosis worsens as subretinal fluid accumulates from choroidal disruption with progression to exudative detachments.¹ Visual prognosis is directly correlated with the magnitude of disconnect between the photoreceptor's inner and outer segments, as well as that between the RPE and choriocapillaris layers.^{6,11} Atrophy and thinning of affected retina are concerning because they increase the likelihood for development of a full-thickness macular hole, which can be visually debilitating.⁴

Low vision consultation is warranted for advanced cases of AOVN when the patient experiences difficulty performing daily tasks and is motivated for intervention. Laying the groundwork for the evaluation requires asking the patient questions to determine his or her functional status:

- “What brings you in to the examination?”
- “Do you live alone or with a spouse?”
- “Do you have a physical impairment or notice any environmental issues that hinder your mobility?”
- “How well do you get around in both familiar and unfamiliar settings?”

In order to determine in which direction the evaluation should go, the examiner should determine the

previous devices the patient has used, as well as the level of effectiveness experienced. The patient's chief complaint often guides the goal-selection portion of the examination.

Visual acuity should be measured with and without correction. Central acuity is required for determining whether legal blindness exists, but eccentric viewing acuities offer a valuable method for gauging the functional capacity of the patient. Be aware that reading speed drastically decreases as the distance of the preferred retinal locus (or eccentric viewing area) increases from central fixation.¹²

Notations such as “hand motion” and “counting fingers” to describe visual acuity should be avoided due to limited legal justification and variable reproducibility.¹³

For patients with visual impairment, the examination is limited by the use of the Snellen distance acuity chart.¹³ The Feinbloom chart has been found to be more beneficial than the Snellen chart due to varying optotypes and letter graduations, as well as its ability to measure up to 20/2800 distance visual acuity.^{13,14} The MNRead chart has been favored at near due to its logarithmic scale, which results in the ability to determine an estimated add power.¹³

Contrast sensitivity measurement is essential for patients with central visual impairment. Amplifying print contrast has been found to surpass enlarging font size as a technique for increasing reading efficiency in patients with visual impairment.¹⁵ The Pelli-Robson chart is favored for calculating impaired contrast due to its logarithmic contrast progression.¹³

Visual field testing is necessary for determining functional visual

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status. Central scotomas and metamorphopsias may be evaluated with use of the Amsler grid.¹⁶ Home Amsler grid testing also provides a proactive way for patients to monitor their own monocular visual changes.¹⁶

The optical foundation of the examination is laid by obtaining an accurate manifest refraction. Due to the patient's decreased visibility through the phoropter, individuals with central visual field loss should always receive a trial frame refraction.¹⁴ Retinoscopy and keratometry data are also helpful tools for guiding challenging refractions.¹³

When prescribing spectacles for patients with visual impairment, a safety frame with polycarbonate lenses provides additional impact resistance, while anti-reflective coatings provide added comfort from glare. For reading glasses, as the power of the lenses increases, base-in prism should be added to the prescription to increase binocularity.¹⁷ High-powered lenses may be prescribed up to +10.00D for visually impaired patients; beyond this amount, consider electronic aids.¹⁷

Handheld and stand magnifiers as well as visors are viable options for patients at near. Note that increasing a device's magnification decreases the visual span (or field) for reading. So, increasing magnification at near may not be the most effective way of increasing reading efficiency.¹⁸ Reading efficiency has been found to improve with use of a typoscope, which aids in decreasing the lateral masking effect of surrounding letters and words.¹⁸ This lateral masking effect refers to the negative effect adjacent words or letters may have on reading efficiency.¹⁸

Mounted and handheld telescopes are ideal for spotting

objects at a distance.^{19,20} Although dynamic visual acuity may improve with adaptation, these devices are typically recommended for use only when the patient is stationary, due to an increased risk of falling due to disorientation.^{19,20}

A few electronic alternatives for patients with advanced central vision loss include closed-circuit televisions, tablets and portable electronic magnifiers.²¹ Electronic talking or large-face wristwatches are also routinely considered for patients with central vision loss.²¹

AOVM is a condition that is often misdiagnosed as AMD. It is important to note the appearance on macular OCT and FA of the well-demarcated vitelliform lesions. These findings will guide the practitioner to the diagnosis of AOVM.

Once diagnosed, management involves the collaboration of care for visually impaired patients by low vision specialists and retinal specialists. Due to the increased life expectancy and concerns for improving quality of life for affected patients, demand for low vision rehabilitation is growing.²²

Currently, our patient continues to be followed by a retinal specialist and is undergoing treatment with Eylea for persistent subretinal fluid in the right eye. His vision has stabilized at 20/150 O.D. and 20/50-O.S., and he is scheduled to return to the low vision clinic as needed. ■

Dr. Hodges is currently completing her residency at the Lake City VA Medical Center in Florida. She will be joining a private practice in Winter Haven, Fla., following the completion of her residency in June 2012. Dr. McGonigal is the Associate Chief of Optometry and Optometry Residency Coordinator at the Lake City VA Medical Center.

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Monthly Multifocal Pearl



Managing Presbyopia

By Nathan Bonilla-Warford, OD, FAO

Like it or not, many of our patients are becoming presbyopic. In North America, it is estimated that 89.9 million people will have presbyopia by the year 2020.¹ To thrive, practices need to embrace the latest technologies in managing this common condition. Doing so will result not only in increased practice revenue from the services and sales of premium products, but also in the strong word-of-mouth referrals that come from satisfied patients. The old tried-and-true options for managing presbyopia may not be effective or desirable. Patients are more educated now than ever before and know about the newer options that are available. Therefore, we as practitioners must aggressively educate ourselves about the newest options and implement office systems that convey this knowledge to our patients.

EXPLAINING PRESBYOPIA

The typical patient does not understand presbyopia. Not only do they not know the term, but they are also not typically aware that in general, eyes must accommodate and that this accommodation decreases with age. Patients may be concerned that reduced clarity and comfort at near may be the beginning of serious vision loss and even eye disease. For this reason (although presbyopia is straightforward and omnipresent to eye-care professionals), we must explain it clearly and compassionately to the patient who is experiencing it.

Patients need to understand that although presbyopia is a perfectly normal part of life, it need not prevent them from leading a full life with clear and comfortable vision during work, recreation and other day-to-day activities. Importantly, they need to understand that proper management of presbyopia does not require treatment for eye disease, using eye drops or taking pills or supplements. Proper refractive care, however, is essential.

THE BENEFITS OF MULTIFOCAL CONTACT LENSES

Today's presbyopic patients have difficulty with near vision, but they are very active and wish to be free of their glasses when involved in certain activities. They may have the misconception that contact lenses are not an option for them or simply have never been offered the opportunity to experience clear distance and near vision without glasses.

Some current contact lens wearers may fear that if they complain about near vision, their doctor will tell them they are no longer a candidate for contact lenses. These patients should be reassured that options such as AIR OPTIX® AQUA Multifocal contact lenses exist and can provide additional power at near. These lenses are designed for superior breathability² and stability for continued comfort.

A LENS THAT WORKS FOR PRESBYOPE OF ALL AGES

With all of the near vision demands that modern life requires, it is necessary to have a lens that can be prescribed to meet a patient's needs. Fortunately, AIR OPTIX® AQUA Multifocal contact lenses come in low, medium and high add powers. These lenses can be refit directly from the fitting set to find the correct parameters that patients can wear throughout their presbyopic years with only minor adjustments and

without refitting a new material and design.

Even patients who have previously been successful in a monovision lens will benefit from a multifocal lens. AIR OPTIX® AQUA Multifocal contact lenses will not only restore binocular vision to these patients, but will also provide them with other benefits, such as superior breathability and stability.

PRACTICE PEARLS

Because many presbyopic patients don't know that multifocal contact lenses are an option, it's important to help make them aware of the possibility.

- Include a question on your intake form inquiring about glasses-free near-vision clarity and comfort.
- Include some educational materials—either digital screens, posters, or flyers—in your office.
- If a patient is already wearing contact lenses, discuss their near vision acuity. Even if they don't feel the need this year, they will remember the discussion and be more motivated next year.
- Don't forget the pre-presbyopes. Many of them spend long hours at work and may benefit from a mild addition of near power without any reduction in distance acuity.
- In all cases, understand how the patient feels about the situation and highlight what is uniquely beneficial or challenging about their case. Patients like to feel special and that helps get them excited about the process.

BE PROACTIVE!

As with many things in life, it's easier to adapt and make changes when we're younger. That said, emerging presbyopes are generally both more motivated and easier to fit in multifocal lenses than patients 10 or 20 years older. And once a patient is accustomed to the multifocal modality, it's easy to modify their prescription over time. Simply follow the fitting guide that comes with your AIR OPTIX® AQUA Multifocal contact lens fitting set and let the patient guide you to success.

Dr. Bonilla-Warford blogs to eyecare professionals about social media and practice management at Review of Optometric Business and is a member of the Social Media Committee of the College of Optometrists in Vision Development.

1. Holden BA, Fricke TR, Ho SM, et al. Global vision impairment due to uncorrected presbyopia. Arch Ophthalmol. 2008;126(12):1731-1739.

2. Based on the ratio of lens oxygen transmissibilities; Alcon data on file, 2009.

Important information for AIR OPTIX® AQUA Multifocal (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness and/or presbyopia. Risk of serious eye problems (i.e., corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

See product instructions for complete wear, care, and safety information.



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3rd Annual Retina Report

An Overview of Intravitreal Injection

Here, we examine the history and process of intravitreal injection as well as review your role in managing potential complications. **By Jay M. Haynie, O.D.**

In my practice setting, I have been fortunate to observe and manage countless patients with various sight-threatening retinal conditions. Many years ago, I was given the opportunity to participate in a retinal fellowship in a surgical practice located in Tacoma, Wash. This position allowed me to work with several retinal surgeons as well as share patients, experience surgical trends, observe outcomes, and witness the evolution of retina care firsthand.

Currently, I serve as the clinical director of the practice where I completed my fellowship. In this setting, I have found it worthwhile to consider comanagement as an evolutionary process. From a historical perspective, it is clear that the current approach to optometric comanagement has taken significant

time to emerge and differs greatly across various practice settings, states and local communities (*for further reading, see "What is 'Integrated Eye Care?'" March 2012*).

So, it seems logical to expect that the comanagement of intravitreal injections for retinal disease also will evolve over time and in different ways. This form of medical care likely will continue between retinal specialists and optometrists for several years to come. Therefore, optometrists must be educated thoroughly about the process of intravitreal injection as well as how to properly manage potential secondary complications.

The History of Intravitreal Injection

The administration of intravitreal injections has revolutionized the

treatment of many visually devastating retinal diseases, including age-related macular degeneration (AMD), diabetic retinopathy and retinal occlusive disease as well as vitreoretinal surgery. There is no question that the number of intravitreal injections performed by retina specialists has increased dramatically. During the past decade, there has been a veritable explosion of new drugs, techniques and indications for intraocular injection.

At one time, intravitreal injection was limited to antibiotic agents that were intended to treat a discrete subset of patients with endophthalmitis. Today, however, many subtypes of AMD and retinal vascular disorders are treated with intravitreal injections of anti-vascular endothelial growth factor (VEGF) agents. Because of the high frequen-

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Goal Statement: Because of the increased use of anti-VEGF therapy for wet AMD during the last several years, optometrists can expect to comanage these patients more than ever before. Therefore, we must be educated thoroughly about the process of intravitreal injection as well as how to properly manage potential secondary complications.

Faculty/Editorial Board: Jay M. Haynie, O.D.

Credit Statement: COPE approval for 2 hours of CE credit is pending for this course. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

Joint-Sponsorship Statement: This continuing education course is joint-sponsored by the Pennsylvania College of Optometry.

Disclosure Statement: Dr. Haynie has no relationships to disclose.

cy of reinjection associated with anti-VEGF therapy, it is inevitable that optometrists also will interface increasingly with these patients.

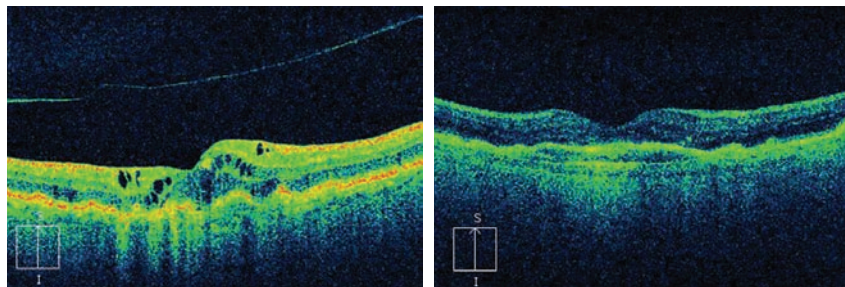
In the past, injections associated with the eye primarily were administered periocularly (e.g., frequent sub-Tenon's injections of steroids and antibiotics delivered following intraoperative surgery). Periocular injections of steroidal medications became a standard method for the management of uveitis patients. Additionally, several retina specialists determined that periocular steroid injections were extremely useful in the management of wet AMD (steroids helped limit the extent of fibrotic and inflammatory disease associated with neovascularization).¹

Subsequently, intravitreal steroid injection in an outpatient setting was attempted for a host of inflammatory conditions, including wet AMD and diabetic macular edema. Further, intravitreal ganciclovir injection also emerged as a technique for treating cytomegaloviral retinitis in patients with HIV. Over time, it became apparent that injection—both in and around the eye—was safe, efficient, practical and useful in helping patients with a variety of ocular conditions.

Understanding Pharmacotherapy Options

Anti-VEGF therapy has dramatically changed how eye care providers treat and manage sight-threatening retinal disease. During the last 15 years, we have witnessed a relative explosion in viable treatment options for our patients. Here is a look at several established and cutting-edge treatment options for advanced retinal disease:

- **Laser therapy.** Some of the earliest management strategies for significant retinal disease predominantly included the use of laser



1, 2. Note the multiple pockets of cystoid intraretinal edema seen in this patient with wet age-related macular degeneration (left). Following an injection of Avastin (bevacizumab, Roche/Genentech), the intraretinal edema resolved.

photocoagulation and photodynamic therapy. Although many clinical trials proved laser therapy to be beneficial for AMD, patients often experienced disease recurrence secondary to the continuous release of VEGF.²

VEGF is a protein produced by cells that stimulates angiogenesis. When unregulated by hypoxia, VEGF is released into the vitreous cavity, causing vascular permeability and associated retinal edema. Laser photocoagulation does not address the release of VEGF, and therefore underlying retinal disease continues to smolder in the background. Nonetheless, the ability to slow or suppress the release of VEGF has proven to be essential for long-term visual stability.²

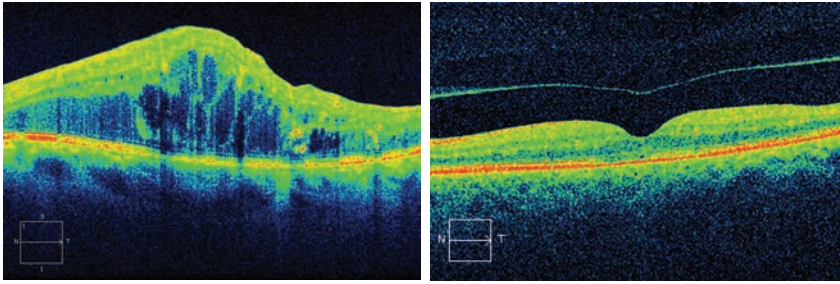
- **Macugen.** In 2004, Macugen (pegaptanib sodium, Pfizer and OSI/Eyetech Pharmaceuticals, Inc.) was the first anti-VEGF agent to receive FDA approval for the treatment of neovascular AMD. Its development marked a clear breakthrough in modern AMD management.

Macugen is a selective anti-VEGF compound that is designed to inhibit one strain of VEGF. It should be administered via intravitreal injection every six weeks. Although the use of Macugen has declined with the release of newer anti-VEGF agents, such as Lucentis (ranibizumab, Roche/Genentech) and Avastin

(bevacizumab, Roche/Genentech), it appears to be making a comeback because of its more favorable dosing frequency (e.g., every six weeks vs. every four weeks). Additionally, Macugen is associated with a lower risk of stroke than either Lucentis or Avastin.³

- **Lucentis.** In June 2006, the FDA approved Lucentis for the treatment of neovascular AMD and macular edema secondary to retinal vein occlusion (RVO). Unlike Macugen, Lucentis is thought to be effective against several strains of VEGF. It was the first compound shown to improve visual acuity in patients with wet AMD.⁴ The recommended dosing schedule for Lucentis is one injection per month until the patient stabilizes. Thereafter, Lucentis may be dosed on a p.r.n. basis.

- **Avastin.** Avastin is FDA-approved for the treatment of colorectal cancer. However, because the agent costs substantially less per dose than Lucentis, it has been widely used off-label since 2004 to treat several retinal diseases, including neovascular AMD. Recently, a major study supported by the FDA—the Comparison of Age-related Macular Degeneration Treatment Trials (CATT)—compared the safety and therapeutic efficacy of both Lucentis and Avastin for the treatment of neovascular AMD.⁵ CATT results confirmed



3, 4. At left, marked cystoid edema seen in a patient with an underlying central retinal vein occlusion prior to treatment with Ozurdex (dexamethasone intravitreal implant, Allergan). Following Ozurdex implantation, the cystoid edema rapidly cleared.

that both compounds exhibit statistically equivalent safety and therapeutic benefit in the preservation of visual acuity. Most managing clinicians opt to administer one intraocular injection of Avastin per month until the patient's vision stabilizes, and then on a p.r.n. basis thereafter (*figures 1 and 2*), much like Lucentis.

- **Eylea.** Also known as VEGF Trap-Eye, Eylea (aflibercept, Regeneron Pharmaceuticals) is the latest anti-VEGF agent to receive FDA approval for the treatment of neovascular AMD. Unlike Macugen, Lucentis and Avastin, Eylea is administered every other month (following a loading phase consisting of three monthly injections).

FDA approval for Eylea was secured following publication of the VIEW 1 and VIEW 2 clinical trials, which compared the agent's safety and efficacy profile to that of Lucentis.⁶ The primary endpoints of VIEW 1 and VIEW 2 were maintenance of visual acuity (defined as losing fewer than 15 ETDRS letters) for one year. Most importantly, the VIEW researchers concluded that subjects who received 2mg of Eylea every eight weeks exhibited comparable visual improvement to subjects who received 2mg of Lucentis every four weeks.⁶ These results suggest that, when compared to Lucentis, the reduced dosing frequency of Eylea likely will save

patients not only time and money, but also significant discomfort.

- **Kenalog.** For years, intravitreal Kenalog (triamcinolone acetonide, Bristol-Myers Squibb) has been used either alone or as an adjunct to anti-VEGF therapy in patients with a variety of retinal conditions. Intravitreal Kenalog is especially effective at treating macular edema secondary to uveitis, diabetic retinopathy and RVO.^{7,8}

To date, the therapeutic impact of Kenalog on patients with neovascular AMD is not well understood. However, clinical experience suggests that Kenalog may potentially slow the rate of visual acuity loss, which could reduce the number of total anti-VEGF or laser treatments required to maintain serviceable vision throughout the patient's lifetime. The primary concern associated with intravitreal Kenalog is an increased risk of posterior subcapsular cataract development and/or steroid-induced glaucoma.^{7,8}

- **Ozurdex.** Ozurdex (dexamethasone intravitreal implant, Allergan), a biodegradable steroid implant, is FDA approved to treat edema associated with RVO and non-infectious uveitis. Clinical studies have shown a rapid reduction in retinal edema secondary to RVO (*figures 3 and 4*) as well as elimination of vitreous haze associated with uveitis within one to two months after Ozurdex implanta-

tion.⁹ The implant may remain in place up to four months; however, the exact duration varies depending on the presentation and/or therapeutic response. Much like Kenalog, Ozurdex may increase a patient's risk for subcapsular cataract development and/or steroid-induced glaucoma.^{7,8}

The Process of Intravitreal Injection

Although slight variations exist between eye care providers, the practice of intravitreal injection is fairly uniform. Notable procedural differences include: use of gloves by the clinician, use of a sterile drape, a preoperative/postoperative measurement of intraocular pressure and the use of postoperative topical antibiotics. Here is the standard, step-by-step process for the administration of intravitreal injection:¹⁰

- Instill topical tetracaine and topical antibiotics.
- Administer a subconjunctival injection of lidocaine for anesthesia.
- Apply a povidone iodine scrub to lid margin and lashes.
- Insert the lid speculum.
- Administer a povidone iodine drop (or apply another swab) to the conjunctiva in the area of injection site.
- Inject the pharmacologic agent into the superior temporal quadrant.
- Instill a topical antibiotic.
- Remove the lid speculum.
- Confirm hand motion vision and/or measure the patient's intraocular pressure.
- Rinse the eye and lid margin with sterile saline.
- Prescribe topical antibiotics for at-home patient use.

It is essential to mention that the retinal surgeon's primary concern is avoidance of infectious endophthalmitis following intravitreal injection.¹¹⁻¹⁵ Fortunately, strict adherence to the aforementioned

injection administration protocol is believed to reduce this risk.¹¹⁻¹⁵

Adverse Reactions

The most common adverse reaction to intravitreal injection is superficial keratitis associated with the povidone iodine solution. Without question, this reaction is the sole reason for triage calls within the first 24 hours following injection. Patients are encouraged to use artificial tears regularly for symptoms of discomfort and irritation immediately following the injection.

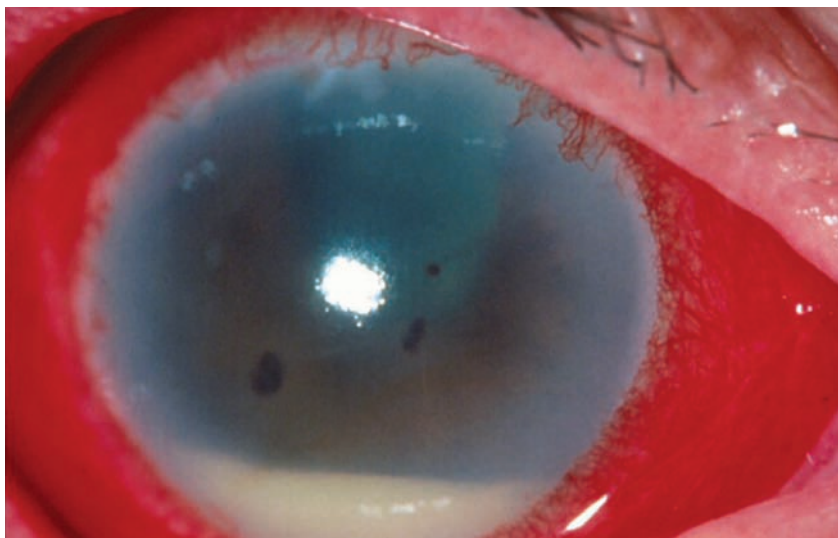
More serious complications include both infectious and pseudo-endophthalmitis, intraocular hemorrhage, retinal tear, retinal detachment, cataract formation and increased intraocular pressure.^{11,12,16,17}

Secondary Complications

- *Infectious endophthalmitis* is the single complication that is most feared by all ophthalmic surgeons. Although the risk of endophthalmitis following intravitreal injection remains low, we must familiarize ourselves with the clinical signs and symptoms.

The symptoms of endophthalmitis following intravitreal injection may include: increased ocular discomfort, light sensitivity, pain, floaters and a marked decrease in visual acuity.¹⁸ The onset of symptoms may vary; however, patients generally experience such secondary complications within three to five days following injection.

Clinical signs of endophthalmitis include redness; cellular reaction of the anterior chamber and vitreous; the abundance or complete absence of fibrin in the anterior chamber and/or the vitreous; corneal edema and keratic precipitates; marked vitreous haze; and a possible hypopyon (*figure 5*). It is important to note that a patient does not have



5. Anterior segment of a patient with infectious endophthalmitis. Note the global injection and steamy cornea in conjunction with the hypopyon.

to exhibit all of these complications; just one or more clinical signs should raise immediate concern in any individual who recently received an intravitreal injection.

- *Pseudo-endophthalmitis* also may occur following intravitreal injection, and it is believed to be a reaction to a preservative in the compound or to the compound itself. The signs of pseudo-endophthalmitis typically present within the first 24 hours following injection and will always be seen with an associated hypopyon (however, the vision may be intact and the level of discomfort usually is lower).

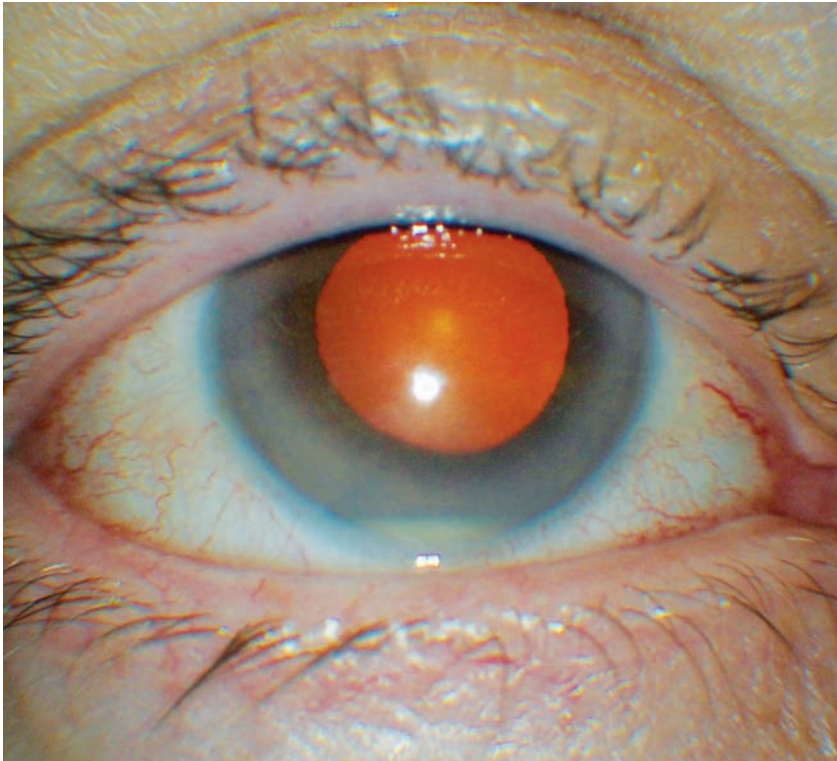
Compared to infectious endophthalmitis, the clinical appearance of eyes with pseudo-endophthalmitis generally is less hyperemic (*figure 6*). The differential between infectious and pseudo-endophthalmitis can be challenging, and may require increased clinical experience to confirm. As a safety measure, a vitreous tap with an injection of antibiotics often is performed if there is any concern that the presentation has an infectious etiology. To ensure patient safety, immediate

referral back to the retinal surgeon is strongly advised should any symptoms of either infectious or pseudo-endophthalmitis manifest.

- *Increased intraocular pressure* following intravitreal injection has been reported in up to 9.4% of cases; yet, just 5.5% developed sustained pressure elevation that required topical or surgical management. Keep in mind, however, that steroid-associated intraocular pressure elevation caused by inhibited aqueous outflow is far more common than pressure increases secondary to anti-VEGF therapy.¹⁹⁻²¹

Managing Complications

The management of infectious endophthalmitis has evolved during recent years, and currently includes a vitreous biopsy (with or without vitrectomy) as well as an intravitreal antibiotic injection (i.e., vancomycin hydrochloride and/or ceftazidime). Some retinal specialists advocate the use of 400mg gatifloxacin p.o. q.d. and 1gt topical gatifloxacin q.i.d. As a general guideline, consider the use of topical gatifloxacin for any patient who



6. Anterior segment photograph of a patient with pseudo-endophthalmitis. Note how quiet the eye looks, despite the presence of the hypopyon.

presents with a suspected case of endophthalmitis.

Patients who present with pseudo-endophthalmitis can be safely and effectively managed in a similar fashion to those individuals suspected of infectious endophthalmitis. In the absence of intraocular fibrin, however, I will initiate topical fluoroquinolones and topical steroids q.i.d., and schedule the patient for 24-hour follow-up. If the patient experiences a decrease in visual acuity or an increase in intraocular inflammation, you should make a referral for vitreous biopsy and intravitreal antibiotics.

When managing increased intraocular pressure secondary to intravitreal steroid injection, we must familiarize ourselves with the duration/efficacy of the compound in the vitreous cavity. One study indicated that at least 0.1cc of triamcinolone per 4mg dose was mea-

surable in patients' vitreous cavities for up to three months following injection.²² In such cases, patients required the use of topical intraocular pressure medications for an average of eight months following injection.²³

When deciding how to manage any elevation in intraocular pressure, it really depends on the status of the optic nerve, the visual field and the retinal nerve fiber layer. A patient with a healthy optic nerve, for example, can withstand a transient pressure increase better than a patient with known compromise or damage.

Nonetheless, patients with glaucoma and coexisting retinal disease should undergo optic nerve head photography, visual fields testing and retinal nerve fiber layer analysis at baseline prior to intravitreal injection. These tests can help predict which patients are at the great-

est risk of further damage in the event of even a modest increase in intraocular pressure.

Given that the etiology of elevated intraocular pressure is a compromise in aqueous outflow facility, treatment should include the use of topical agents that increase aqueous outflow. These agents include Alphagan P (brimonidine tartrate 0.15%, Allergan), Timoptic (timolol 0.5%, Aton Pharma), Trusopt (dorzolamide 2.0%, Merck) and Azopt (brinzolamide 1.0%, Alcon). Keep in mind, however, that prostaglandin analogues should not be used in patients with known macular edema, because they may exacerbate the underlying condition.^{24,25}

The concept of measuring intraocular pressure one week following intravitreal injection is somewhat controversial, and can be tiring for the patient given the frequency of examinations at the retina specialist's office. However, patients with advanced glaucoma should be monitored closely for pressure increases following injection. You should inform the comanaging retina specialist of any documented intraocular pressure spikes.

The O.D.'s Role

So, where do we fit into all of this? Ultimately, our role in the comanagement of intravitreal injection still remains undefined; however, we will continue to provide primary eye care for these patients. Similar to cataract surgery, we have the responsibility of discussing potential treatment options with our patients prior to considering a referral.

Additionally, providing the retina specialist with pertinent ocular history as well as any documented contraindications to intravitreal injection, such as ocular hypertension or glaucoma, is extremely important.

Further, completing a risk assessment of the patient is becoming more common, and it should include the following questions:

- Is there active blepharitis in either eye?
- Can the patient cooperate during intravitreal injection and not interfere with the sterile technique?
- Will the patient recognize symptoms of endophthalmitis and report them to you immediately?
- Can the patient instill topical antibiotics into the eye following the procedure?

• Is the patient going to return for scheduled follow-up appointments?

These are just a few baseline questions that can be asked of every patient being considered for intravitreal injection.

In addition to preparing the patient for a commitment to intravitreal injection, it is equally important to recognize signs of existing recurrent disease. Fortunately, we now have access to cutting-edge diagnostic technology, such as spectral-domain optical coherence tomography (SD-OCT) and fundus autofluorescence, that more easily enables us to identify subtle characteristics of retinal disease that may be difficult to see clinically or even with conventional retinal photography. For example, SD-OCT is vital in the postoperative management of patients with AMD, because we can document intraretinal edema (figure 7) or subretinal fluid (figure 8), which may help identify a recurrent neovascular membrane in the absence of new symptoms.

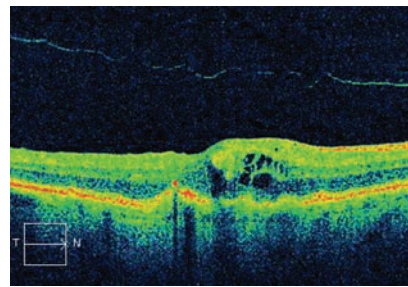
Unlike cataract surgery, intravitreal injection often requires a series of re-treatments over the course of the patient's lifetime. For some individuals, this translates into several years of care with a retina specialist and comanaging O.D.

Although it is not our job to

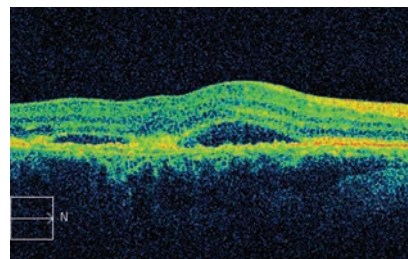
decide upon the actual treatment plan, we certainly can prepare our patients for what to expect. In this, we play an essential role in educating the patient about his or her condition as well as the most effective treatment options that could help maintain or even restore their vision. ■

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7. Intraretinal cystoid edema in a patient with wet AMD (top).



8. Subretinal fluid seen in a patient who presented with wet AMD.

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OSC QUIZ

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

1. What was the first sight-threatening ocular condition to be treated via intravitreal injection?
 - a. Age-related macular degeneration (AMD).
 - b. Retinal occlusive disease.
 - c. Endophthalmitis.
 - d. Diabetic retinopathy.
2. Vascular endothelial growth factor (VEGF) is a cellular protein that:
 - a. Upregulates hypoxia.
 - b. Stimulates angiogenesis.
 - c. Lowers intraocular pressure (IOP).
 - d. Decreases vascular permeability.
3. What was the first anti-VEGF agent to receive FDA approval for the treatment of wet AMD?
 - a. Avastin (bevacizumab, Roche/Genentech).
 - b. Eylea (aflibercept, Regeneron Pharmaceuticals).

- c. Lucentis (ranibizumab, Roche/Genentech).
- d. Macugen (pegaptanib sodium, Pfizer and OSI/Eyetech Pharmaceuticals, Inc.).

4. Which statement about Macugen is FALSE?
 - a. It is a non-selective anti-VEGF compound.
 - b. It is a selective anti-VEGF compound.
 - c. It is associated with a lower risk of stroke than Lucentis and Avastin.
 - d. None of the above.

5. What is the recommended dosing schedule for Macugen?
 - a. Every four weeks.
 - b. Every five weeks.
 - c. Every six weeks.
 - d. Every eight weeks.

6. Lucentis is FDA approved for the treatment of:
 - a. Wet AMD.
 - b. Macular edema secondary to retinal vein occlusion.
 - c. Central serous retinopathy.
 - d. Both a and b.

7. What is the recommended dosing schedule for Lucentis?
 - a. Every four weeks.
 - b. Every five weeks.
 - c. Every six weeks.
 - d. Every eight weeks.

8. Which statement about Avastin is FALSE?
 - a. It is FDA approved for the treatment of wet AMD.
 - b. It was shown to treat wet AMD as effectively as Lucentis.
 - c. It is less expensive than Lucentis.
 - d. None of the above.

9. Which major clinical trial recently confirmed that both Lucentis and Avastin exhibit similar safety and efficacy profiles for the treatment of wet AMD?
 - a. ANCHOR.
 - b. CATT.
 - c. LAST.
 - d. VIEW I.

10. Which anti-VEGF agent most recently received FDA approval for the treatment of wet AMD?
 - a. Avastin.
 - b. Eylea.
 - c. Lucentis.
 - d. Macugen.

11. Following a three-month loading phase, what is the recommended dosing schedule for Eylea?
 - a. Every four weeks.
 - b. Every five weeks.
 - c. Every six weeks.
 - d. Every eight weeks.

12. What is a potential side effect of intravitreal Kenalog (triamcinolone acetate, Bristol-Myers Squibb)?
 - a. Nuclear cataracts.
 - b. Posterior subcapsular cataracts.
 - c. Vitreous hemorrhage.
 - d. Retinal detachment.

13. Ozurdex (dexamethasone intravitreal implant, Allergan) is FDA approved for the treatment of?
 - a. Wet AMD.
 - b. Macular edema secondary to retinal vein occlusion.
 - c. Non-infectious uveitis.
 - d. Both b and c.

14. Ozurdex's average duration of efficacy is estimated to be:
 - a. Two months.
 - b. Three months.
 - c. Four months.
 - d. Six months.

15. Which step is NOT part of the standard intravitreal injection process?
 - a. Use of oral antibiotics.
 - b. Povidone lid scrubs.
 - c. Use of lid speculum.
 - d. Topical and subconjunctival anesthesia.

16. What is the most common adverse reaction to intravitreal injection?
 - a. Vitreous hemorrhage.
 - b. Pseudo-endophthalmitis.
 - c. Superficial keratitis.
 - d. Infectious endophthalmitis.



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4:00pm – 6:15pm CE Courses
6:15pm – 7:15pm Welcome Reception

Friday, July 20, 2012

6:30am – 7:00am Breakfast with Sponsors
7:00am – 9:00am CE Courses
9:00am – 9:30am Break with Sponsors
9:30am – 12:00pm CE Courses

Saturday, July 21, 2012

6:30am – 7:00am Breakfast with Sponsors
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9:00am – 9:30am Break with Sponsors
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Eye Tremor Points to Parkinson's

Tremor is a hallmark of Parkinson's. A new study shows tremor manifests earliest in the eye. This could mean earlier diagnosis and treatment. **Edited by Paul C. Ajamian, O.D.**

Q I have been reading about ocular tremors as a sign for Parkinson's disease.

Can you tell me more?

A "In addition to the more well-known characteristics of resting tremor, rigidity and bradykinesia, Parkinson's disease can also affect the ocular motor system," says Denise Goodwin, O.D., Associate Professor of Optometry and Coordinator of the Neuro-ophthalmic Disease Clinic at Pacific University College of Optometry, in Oregon.

"Because Parkinson's disease is a common disorder that can cause impaired visual function, we are likely to see these patients in our offices," she says. "So, it's important to be familiar with this condition and the ocular consequences."

Several studies have looked at ocular motor movement in people with Parkinson's disease. One recent study—the largest of its kind—found fixation instability in all 112 Parkinson's patients.¹ Of these, 63% had an amplitude significant enough to affect their vision. In comparison, just two of the 60 control subjects demonstrated a similar eye movement pattern. Interestingly, after being followed for two years, one of these two controls developed additional non-ocular parkinsonian features.

"The fact that the ocular tremor was found in every subject with Parkinson's disease, as well as one control subject who later developed parkinsonian features, suggests that



A subject with Parkinson's undergoes video eye tracking to measure ocular tremor.

convergence insufficiency," Dr. Goodwin says. "This can result in diplopia at near and reading difficulties. It can be treated with single-vision reading glasses with prism, but may be difficult to treat because of fluctuation."

Other ocular motor abnormalities include hypometric saccades, saccadic pursuit movements, square-wave jerks and limitation of up-gaze.

"Eyelid disorders commonly associated with Parkinson's disease include blepharospasm, apraxia of eyelid opening, and decreased blink rate resulting in dry eye symptoms, which is the most common ocular symptom among these patients," Dr. Goodwin says. In addition, be aware that up to one-fourth of these patients experience visual hallucinations.²

"Comanagement with a neurologist will help give these patients the best treatment," Dr. Goodwin adds. "Currently, double vision or irregular eye movements in conjunction with classic signs of Parkinson's disease, such as tremor or 'pill-rolling' movements, should prompt a consultation with the neurologist. Once diagnosed, management of the double vision with prism or vision therapy, as well as dry eye treatment, will help alleviate patient symptoms." ■

this testing may become useful in diagnosing Parkinson's disease," Dr. Goodwin says.

The eye tremors discussed in this study are too minute to be detected at the slit lamp. Special eye tracking equipment is needed. The testing instrument used in this study is a "research-grade" device (Eyelink II, SR Research), and is not likely to be found in most eye doctors' offices. So, whether such testing will soon become part of the comprehensive ophthalmic exam remains to be seen.

"If this study is validated, eye movement recording devices may become more common in optometric practices and allow earlier diagnosis of Parkinson's," Dr. Goodwin says. "Because patients are more likely to see an optometrist on a regular basis than a neurologist, optometry would be in an ideal position to identify these patients."

Q What other motility and eye disorders should I look for?

A "Another common ocular motor disorder associated with Parkinson's disease is

Photo: Mark Baron, M.D., Hunter Holmes McGuire VAMC

1. Gitchev GT, Wetzel PA, Baron MS. Pervasive ocular tremor in patients with Parkinson disease. *Arch Neurol*. 2012 Apr 9 [epub ahead of print].

2. Biouesse V, Skibell BC, Watts RL, et al. Ophthalmologic features of Parkinson's disease. *Neurology*. 2004 Jan 27;62(2):177-80.



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Softer Approach to Keratoconus?

Newer soft lens designs for keratoconus can deliver both comfort and excellent visual outcomes to many patients. **Edited by Joseph P. Shovlin, O.D.**

Q I noticed that there are several new soft lens options for thinning disorders like keratoconus. Are practitioners using these as first-line options or last-ditch efforts when rigid gas-permeable lenses or hybrids fail before grafting? How do the designs differ?

A While there are a couple of new soft lens options for managing keratoconus and other irregular corneal astigmatism, the idea itself is not a new one, says Mark André, a contact lens specialist and associate professor of optometry at Pacific University, in Forest Grove, Ore. He has been using soft lenses to manage irregular astigmatism for more than three decades—starting with the Flexlens for Keratoconus (Walman Optical) in the late 1970s. “With the advent of more sophisticated manufacturing techniques and higher Dk materials, there has not been a better time to incorporate custom soft lenses for keratoconus into your practice,” Mr. André says.

While rigid corneal contact lenses have long been the go-to treatment for keratoconus, many patients are looking for a more comfortable lens that can be worn all day. New soft lens designs have made it possible to address the complex optical issues that keratoconus creates, while also providing more comfortable wear for many patients.¹

The latest soft lenses for keratoconus on the market are NovaKone (Alden Optical) and KeraSoft IC (Bausch + Lomb). The biggest difference between the two

lenses is the material—NovaKone is comprised of Benz G4X 54% water hydrogel material with a Dk value of 21, and KeraSoft IC is a lathe-cut silicone hydrogel material that is 74% water with a

Dk value of 60. The NovaKone can be ordered in five different thicknesses in order to mask varying degrees of corneal distortion. Mr. André has found great success with both lens designs and recommends having both fitting sets if you have a large specialty contact lens practice.

“Historically, custom soft lenses that mask irregular corneal astigmatism have met resistance from practitioners concerned with their increased lens thickness,” he says. “I rarely observe hypoxia-related complications with these specialty lenses, and the best explanation that I have is that they move up to 1mm with a blink. The increased tear exchange under the lens must make up for the relative decrease in oxygen transmissibility.”

He always considers soft lenses as one of his contact lens options for keratoconus before resorting to surgical intervention. “Generally, I will consider the rigid lens designs first, but if the patient has exhausted the rigid lens options or dislikes the comfort of rigid lenses, I would not hesitate to recommend a custom soft lens,” he says. “The visual



Patient wearing a soft custom lens for keratoconus.

Photo: Patrick Caroline, C.O.T.

outcomes we achieve with these lenses are often equal to, and in some cases better than, the results we get with rigid lenses.”

The patient’s corneal topography plays an important role in how successful the soft lens approach will be. Due to corneal asymmetry, patients with large-diameter sagging may be poor candidates because the steepening of the inferior cornea can cause the lens to lift inferiorly.¹ However, corneas with nipple- and globus-type cones, with relatively concentric 360° peripheral topography, seem to respond well to soft lenses.¹

“Take advantage of the manufacturer’s consultants to help you through the learning curve,” Mr. André says. “Once you build your confidence with fitting these lenses, you will wonder how you managed your challenging contact lens patients without them.” ■

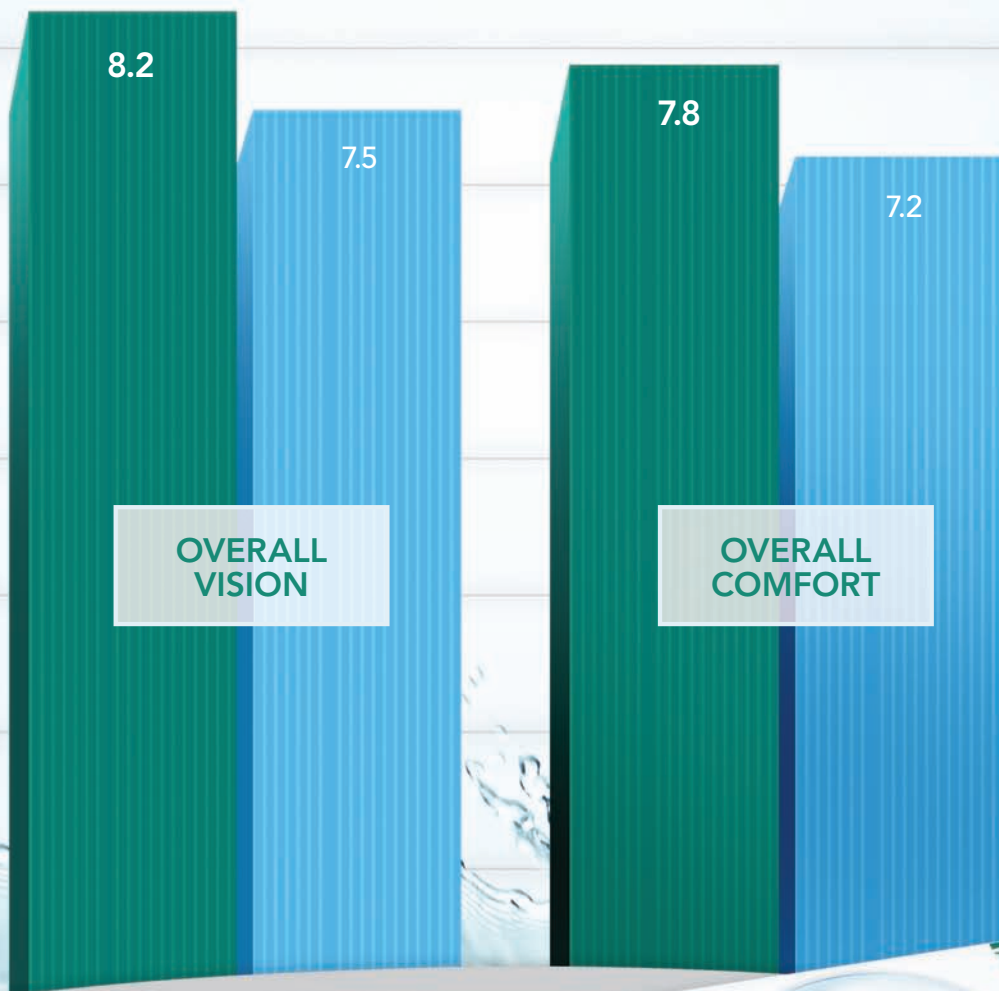
1. Caroline P, Andre M, Kinsohita B, Choo J. Etiology, diagnosis, and management of keratoconus: new thoughts and new understandings. Pacific University College of Optometry. Available at: www.pacificu.edu/optometry/ce/courses/15167/etiologypg4.cfm (accessed May 30, 2012).

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Alcon



Over-the-Hill and Overtreated

A 70-year-old patient presented with relatively normal findings, but he was on multiple glaucoma medications. Was he being overtreated? **By James L. Fanelli, O.D.**

In late December 2011, a 70-year-old white male presented to the office as a new patient to establish care. He had moved to the area approximately six months earlier and needed to have his glaucoma medications refilled. These included Lumigan (bimatoprost, Allergan) h.s. O.U., Azopt (brinzolamide, Alcon) b.i.d. O.U. and Alphagan P (brimonidine, Allergan) 0.1% b.i.d. O.U. He began glaucoma therapy approximately eight years earlier.

His systemic medications included lisinopril, Zetia (ezetimibe, Merck/Schering-Plough), atenolol, Plavix (clopidogrel, Bristol-Myers Squibb/Sanofi), low-dose aspirin (81mg), Cardizem (diltiazem, Valeant Pharmaceuticals/Abbott Laboratories), Nexium (esomeprazole, AstraZeneca), amiodarone and Arthrotec (diclofenac/misoprostol, Pfizer) b.i.d. He reported an allergy to penicillins.

Significant in his medical history was coronary artery bypass graft (CABG) surgery (x3) approximately 10 years earlier, and a left carotid endarterectomy shortly thereafter.

Diagnostic Data

At his initial visit, best-corrected visual acuity was 20/25- O.D. and 20/30- O.S. through myopic astigmatic correction. Pupils were equal, round and reactive to light and accommodation, with no afferent defect. Extraocular motilities were full in all positions of gaze. Confrontation fields were full O.U.

Slit lamp examination of the anterior segment was only remarkable for corneal arcus in both eyes. The anterior chamber was deep and quiet, and angles measured by Van Herick estimation were grade 3- open in both eyes. Intraocular pressure measured 8mm Hg O.D. and 10mm Hg O.S. at 9:45 a.m. Pachymetry readings were 571 μ m O.D. and 563 μ m O.S.

rule in either eye. Retinal vasculature was characterized by moderate arteriolar sclerosis in both eyes with scattered arteriovenous crossing changes. The retinal venules were mildly dilated, but no overt evidence of occlusive disease was present—including retinal hemorrhages, micro-infarcts or areas of retinal ischemia or hypoxia. Both maculae were characterized by fine



Heidelberg Structure and Function printout of the patient's left eye shows a normal optic nerve Moorfields Regression Analysis (middle left and inner green circle, left image) and a normal HEP field (middle right and outer green circle, right image). So, why was he on three glaucoma drops?

Dilated fundus exam showed crystalline lenses with moderate nuclear sclerosis in both eyes, with cortical cataracts encroaching on the visual axis (O.S. > O.D.), consistent with his best-corrected visual acuity. Posterior vitreous detachments were present bilaterally.

Stereoscopic evaluation of his optic nerves demonstrated cup-to-disc ratios of 0.65 x 0.65 O.D. and 0.50 x 0.50 O.S. The neuroretinal rims did not respect the ISNT (inferior-superior-nasal-temporal)

retinal pigment epithelial granulation consistent with his age. The peripheral retinal evaluations in both eyes were unremarkable. I obtained stereo-optic nerve photos at this visit.

We did not have the time to do a complete glaucoma evaluation at this initial visit, so I refilled his three glaucoma medications and asked him to return in a month for further glaucoma testing. In the interim, I sought to obtain his previous records to ascertain the chain

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of events that led to him being medicated for IOP of 10mm Hg and below.

In January 2012, the patient returned as directed. At this visit, we administered Heidelberg Edge Perimeter (HEP, Heidelberg Engineering) visual field testing in both eyes, which were normal with good reliability. IOP at this visit measured 9mm Hg O.D. and O.S. at 11:00 a.m.

Gonioscopy revealed open angles in both eyes with minimal trabecular pigmentation and no ciliary body visible. Angle anatomy was normal with no plateau iris configuration, peripheral anterior synechiae or other abnormalities. Ultrasonic B-scan of the anterior chamber angles confirmed normal iridocorneal angle anatomy and normal posterior chambers with normal iris configuration.

Heidelberg Retina Tomograph-3 (HRT-3, Heidelberg Engineering) imaging of both optic nerves confirmed my earlier description: relatively plush, average-sized neuroretinal rims that did not adhere to the ISNT rule. Moorfields Regression Analysis did not identify any sector of either optic nerve with statistically aberrant parameters.

Discussion

By this point, I had seen the patient twice; in both instances, I did not pick up on one piece of evidence that he had frank glaucoma (except that he has been medicated with three different drugs). His optic nerves looked healthy with moderate cupping. Selective perimetry was normal in both eyes, and medicated IOPs were in the single digits. I could come up with only a few rationales for his therapy with three glaucoma drops:

- Perhaps when he was first

placed on medications years ago, he was an ocular hypertensive patient with IOPs elevated enough to prompt the treating physician to drive the pressures down very low.

- Or, his history of cardiovascular disease, retinal arteriolar sclerosis and carotid artery disease may have given the previous provider the sense that ocular perfusion pressure to the eye(s) was low, which prompted prophylactic IOP lowering.

In reality, it may have been a combination of both factors that ultimately led him to be prescribed three glaucoma medications.

Whether he would be best served with a laser procedure to reduce the number of medications he is currently taking is not of concern here. The primary concern lies with the unanswered question as to why he is even on medication.

Fortunately, his past records arrived prior to his second visit. In reviewing these records, it appeared that initial IOP readings were in the low to mid-20s, as measured by non-contact tonometry on some occasions and by Tono-Pen (Reichert) on other occasions. IOP never exceeded 24mm Hg in either eye.

Furthermore, his records noted “multiple CV issues” on a few of the visits. Reading between the lines, my guess is that the previous provider may have been concerned with blood flow issues to the eyes; his initial visits to the previous eye care provider occurred shortly after his CABG and endarterectomy surgeries.

Now What?

The question now remains: How should we proceed? While there may be some benefit to ameliorating the risks of retinal vascular occlusion (in this case, reducing

the risk of retinal vein occlusions) by lowering IOP, I do not think he needs to have his IOP lowered to prevent glaucomatous damage, as he apparently is an ocular hypertensive patient.

After discussing my findings and thoughts with the patient, I relayed to him my strong feelings about keeping patients on the fewest number of medications that “get the job done.” Accordingly, I asked him to discontinue the Alphagan. He did so, and on follow-up in late March 2012, IOP measured 14mm Hg O.D. and 13mm Hg O.S. Optic nerves remained stable, as did the retinal vasculature. Continuing on the same thought process, I asked him at that visit to discontinue Azopt, and see me again in four to six weeks.

When last seen in early May 2012, his IOP measured 18mm Hg O.D. and O.S. at 9:45 a.m., with stable optic nerves, fields and retinal vasculature. While IOP is at an acceptable range for an ocular hypertensive with slightly thick corneas and normal optic nerves, it remains to be seen whether all will remain stable over time.

Underdiagnosis and Overdiagnosis

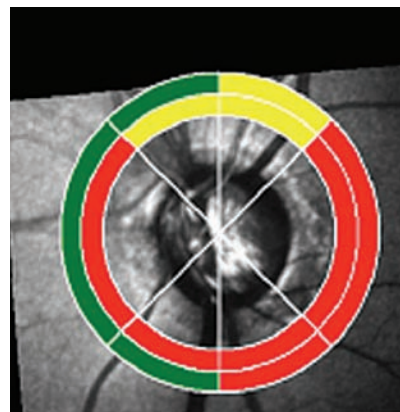
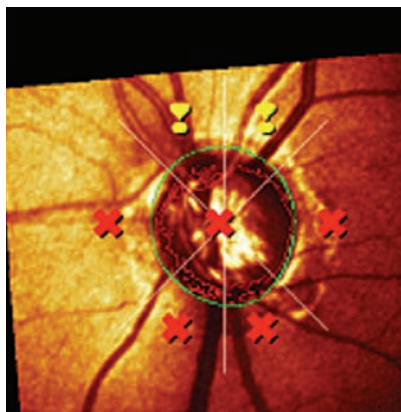
Given that we live in a very mobile society and that Americans move to different locations of the country more now than generations ago, we will be seeing more and more patients who need continuing care. And, when we see new patients, we will sometimes see individuals who are undiagnosed or overdiagnosed.

Interestingly, a recent study looked at Medicare claims in various regions of the country among patients who carried a diagnosis of glaucoma or glaucoma suspect.¹ The study found that, in

certain areas of the country (the Northeast corridor and Mid-Atlantic states), there is a higher likelihood of being diagnosed with either of those, whereas in other parts of the country (such as the South and Southeast), the likelihood of being diagnosed with glaucoma or as a glaucoma suspect is much lower.¹

It begs the question: In those areas where diagnosis rates are higher, are they higher because the providers in those areas do a better job of making the diagnoses, or is it because providers in those areas tend to overdiagnose their patients? And, what role does the variability in frequency of patient visits play in these differing diagnoses rates? These questions require further investigation.

In the interim, each of us is



A different patient showing structural and coincident functional deficits on one printout, as measured by the HRT-3 and HEP.

charged with rendering an opinion of our patient's status. Of course, we should take an objective look at what the previous provider (if there was one) was seeing and thinking when managing the patient. But, our primary goal is to

offer the patient the care that we think is best for them—whether that corresponds with earlier treatment, or contradicts it. ■

1. Cassard SD, Quigley HA, Gower EW, et al. Regional variations and trends in the prevalence of diagnosed glaucoma in the Medicare population. *Ophthalmology*. 2012 Apr 3. [Epub ahead of print]

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Not Just Trichiasis?

This patient presented with dryness and irritation in both eyes, as well as a history of ingrown eyelashes. But, is there more to the story? **By Mark T. Dunbar, O.D.**

An 84-year-old Hispanic male presented with complaints of dryness and irritation in both eyes. The patient stated that, in the past, he had “some issues with eyelashes poking him in the eyes,” which caused irritation and required removal. At the time of this visit, however, he reported that he saw well and that his vision was stable.

His ocular history was significant for successful cataract surgery in both eyes approximately seven years earlier. His medical history was remarkable for type 2 diabetes and hypertension. The patient reported taking a number of medications for both systemic conditions, but couldn’t remember their names.

His best-corrected visual acuity was 20/25 O.D. and 20/30 O.S.

Confrontation fields were full to careful finger counting O.U. The pupils were equally round and reactive, with no evidence of afferent defect. Amsler grid testing was normal.

During the anterior segment evaluation, we observed several misdirected eyelashes involving the upper lids in both eyes that were rubbing on the superior corneas. This low-grade mechanical trauma caused the development of mild punctate epithelial erosions O.U.

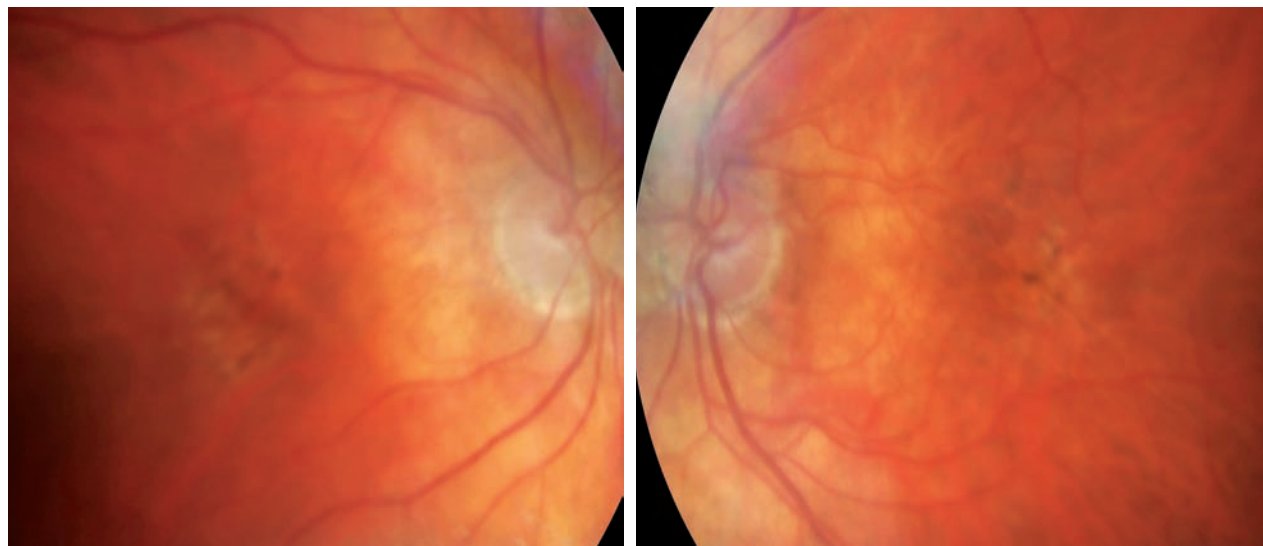
In addition, we noted that the patient’s bilateral posterior chamber intraocular lenses were well centered, with clear visual axes.

The dilated fundus examination showed significant asteroid hyalosis in both eyes, which partially obscured our view of the retina. He appeared to have healthy optic

nerves, with small cups and good rim coloration and perfusion O.U. We noted obvious changes in both maculae on fundus photography (*figures 1 and 2*). Additionally, we obtained a spectral-domain optical coherence tomography (SD-OCT) scan (*figures 3 and 4*) and performed fundus autofluorescence (FAF) imaging.

Take the Retina Quiz

1. What is the correct diagnosis in this case?
 - a. Pattern dystrophy of the retinal pigment epithelium (RPE).
 - b. Dry age-related macular degeneration (AMD).
 - c. Wet AMD.
 - d. Central serous chorioretinopathy.
2. What finding does the



1, 2. We noted peculiar macular changes in our patient (O.D. left, O.S. right). What do they represent?



SD-OCT scan show in the patient's left eye?

- a. Choroidal neovascularization (CNV).
- b. Geographic atrophy.
- c. Serous detachment.
- d. Pigment migration into the sensory retina.

3. What is the underlying etiology?

- a. Age-related.
- b. Degenerative disorder.
- c. Hereditary.
- d. Response to stress.

4. How should this patient be managed?

- a. Observation.
- b. Laser.
- c. Intravitreal injection of an anti-VEGF agent.
- d. Pars plana vitrectomy.

For answers, go to page 146.

Discussion

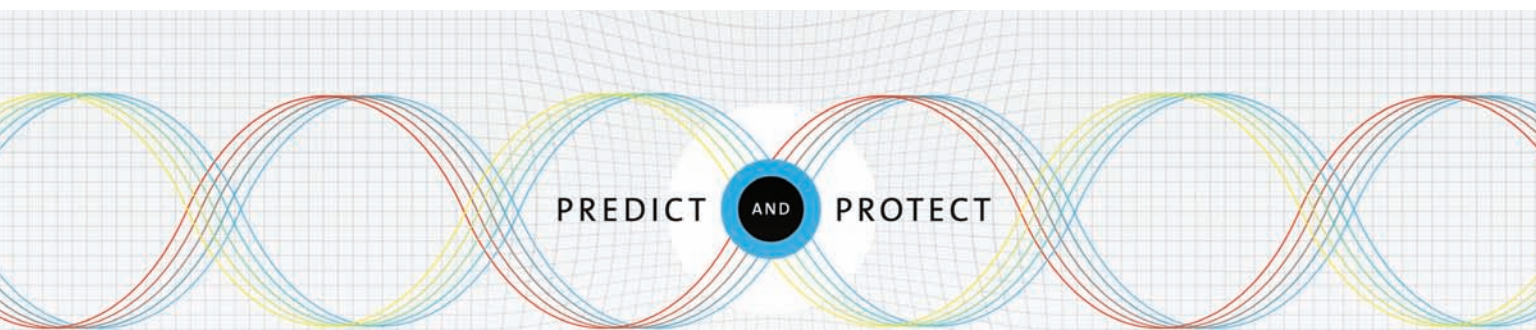
The view into our patient's retina was made difficult secondary to asteroid hyalosis O.U. However, on careful evaluation, unusual RPE changes could be seen in both maculae. There appeared to be RPE pigment clumping and mottling in an almost linear, radiating pattern (O.D. > O.S.), as well as lighter areas of surrounding RPE depigmentation.

In the left eye, there was a plaque of pigment that appeared superficial—unlike what was seen in the right eye (or even elsewhere in the left eye). What was most unusual is that the plaque of pigment appeared to be within the sensory retina. Drusen also appeared to be present; however, this was difficult

to determine because of the asteroid hyalosis. No fluid was detected in either eye.

The SD-OCT scan of the right eye showed thickening of both the RPE and Bruch's membrane, with an intact interior segment/outer segment junction. Further, the SD-OCT confirmed the absence of any subretinal fluid or CNV. The SD-OCT scan of the left eye also showed diffuse thickening of the RPE/Bruch's membrane complex. However, we also documented a highly reflective "spot" within the sensory retina that was located temporal to the fovea, which resulted in posterior shadowing. On careful inspection of the retina and fundus photographs, the spot likely represents an area of intraretinal pigment—not a CNV.

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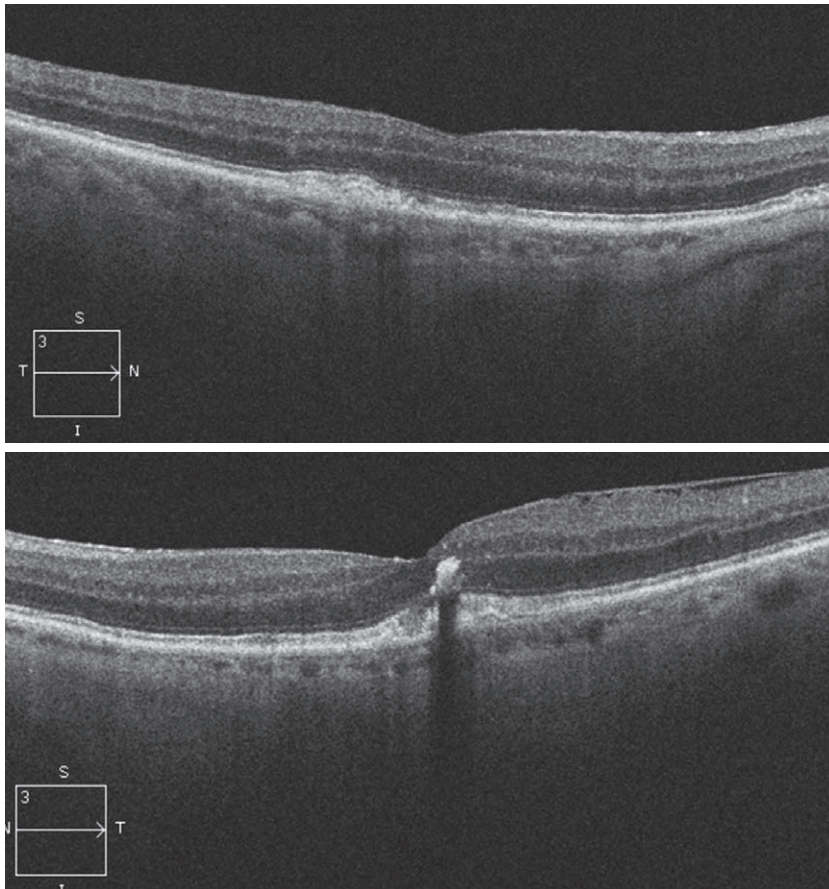
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3, 4. The SD-OCT scans of both maculae (O.D. top, O.S. bottom). What do you notice?

patient? He likely has a pattern dystrophy of the RPE. Pattern dystrophies represent an autosomal group of disorders that present in midlife, with mild visual disturbances in one or both eyes.

Because many patients with this condition may present later in life, they are often referred out or misdiagnosed with AMD. This is incorrect, however, because the pattern dystrophies are not a form of AMD—but rather a separate and distinct disease process.

It is important to note, however, that because most patients who develop pattern dystrophies are of advanced age, drusen may be present (as was the case in our patient). But again, drusen formation is indicative of a separate disease

process that must be followed and managed accordingly.

Patients with pattern dystrophies can present with a variety of macular changes that appear like deposits of yellow, orange or gray pigment mottling in the macular area. Based upon the pattern of pigment distribution within the macula, this disease has been subdivided into at least five principle groups:¹

- *Adult-onset foveomacular vitelliform dystrophy.* Patients in this group may have lesions that can simulate those commonly seen in Best disease.

- *Butterfly-shaped pigment dystrophy.* This form is characterized by yellowish flecks that concentrate in the macula, and may spread throughout the posterior pole. As

the name suggests, the lesions can simulate the appearance of a butterfly. This pattern dystrophy has been linked to a mutation in the RDS/peripherin gene.²

- *Reticular dystrophy of the RPE.* This form of pattern dystrophy can present with a reticulated “fishnet” appearance, in which the RPE changes are more contiguous with one another.

- *Multifocal pattern dystrophy simulating fundus flavimaculatus.* These individuals exhibit pisciform changes that are commonly seen in patients with Stargardt’s macular dystrophy. Unlike patients with Stargardt’s, however, there is no angiographic evidence of a dark choroid.¹

- *Fundus pulverulentus.* Patients with this form show prominent, coarse “punctiform” mottling of the RPE in the macular area.¹

So, which type of pattern dystrophy does our patient have? It is somewhat evident that our patient exhibits a butterfly-shaped pattern, which can be better appreciated in the right eye (with a little imagination). Although infrequently documented, choroidal neovascularization may occur in any of the subgroups. Still, the patient’s prognosis for maintaining good vision is excellent.

We explained the findings to our patient. We removed the misdirected eyelashes, and recommended the use of artificial tears. Additionally, we suggested vitamin supplements that contained lutein and zeaxanthan.

We asked the patient to return in one year for follow-up. ■

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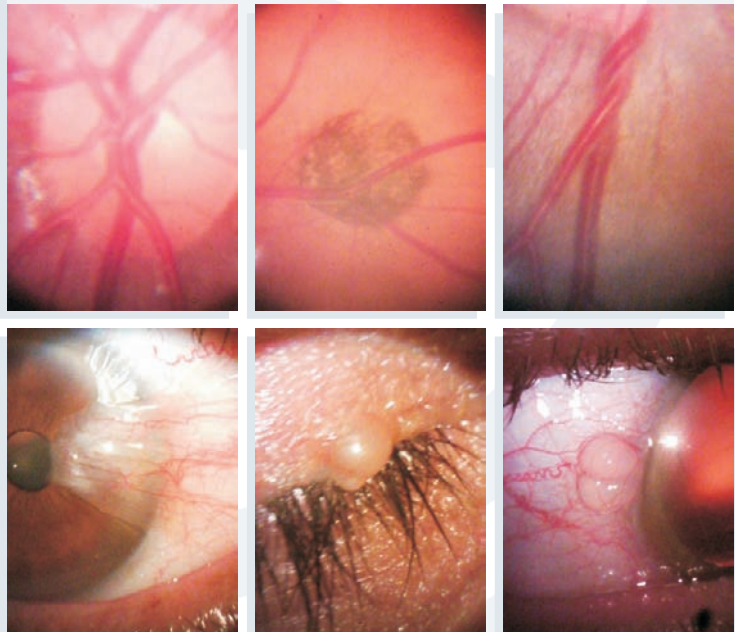
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Don't Hold Your Breath!

Valsalva retinopathy is a potential complication of exertional activity and a compromised vascular system. Here's how to recognize and address this unusual condition.

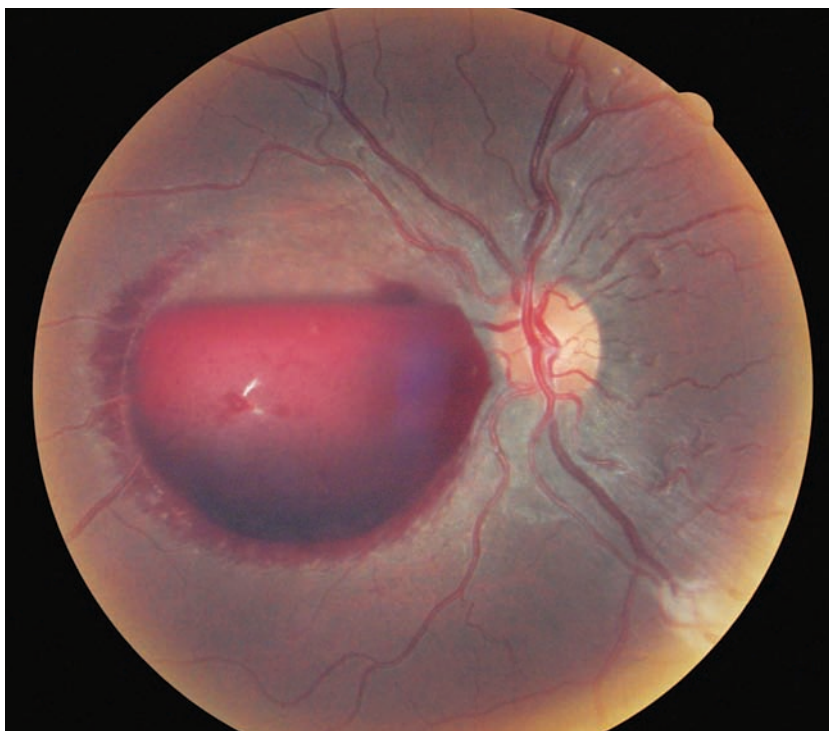
By Alan G. Kabat, O.D., and Joseph W. Sowka, O.D.

A 28-year-old white male presented with complaints of acute vision alteration. He reported that “something just wasn't right” in his left eye, and that there appeared to be a blurry spot in his peripheral vision. His ocular history was unremarkable for prior disease, surgery or trauma. He reported being very healthy, eating well and remaining physically fit. In fact, he mentioned that he first noticed these symptoms two days earlier, after returning home from the gym.

Best-corrected visual acuity measured 20/20 O.D. and O.S. His pupils were equally round and reactive, and his motilities were normal. Upon confrontation fields and Amsler grid testing, the patient consistently displayed a large temporal scotoma in the left eye. Dilation revealed a well-circumscribed pre-retinal hemorrhage located nasally to the left optic nerve (*figure 1*). His history, clinical appearance and lack of associated retinal findings, such as exudates, cotton-wool spots or retinal tears, suggested a diagnosis of Valsalva retinopathy. So, how should we manage this patient?

The Valsalva Maneuver

The term “Valsalva maneuver” is well known in medicine. It is defined as “expiratory effort against a closed glottis, which increases pressure within the



1. Appearance of our patient's left fundus is indicative of Valsalva retinopathy.

thoracic cavity and thereby impedes venous return of blood to the heart.”¹ Valsalva maneuvers can be attained through a variety of circumstances, such as vigorous coughing, sneezing, vomiting, straining through constipation, trying to lift a heavy object, or any such effort that involves straining and holding one's breath.

In our patient, his trip to the gym entailed running on the treadmill and lifting weights—both of which could induce a

Valsalva event.

In a clinical setting, the Valsalva maneuver might be employed to arrest episodes of supraventricular tachycardia, or diagnostically to help identify certain cardiac abnormalities.^{2,3} Complications may be seen when the maneuver is performed either too forcefully or for too long. This is a consequence of the exaggerated physiological changes that occur—particularly elevation of intravascular, intrathoracic (pulmonary) and intra-abdominal

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INDICATIONS AND USAGE:

TRAVATAN Z[®] Solution is a prostaglandin analog indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration:

One drop in the affected eye(s) once daily in the evening.

IMPORTANT SAFETY INFORMATION

Warnings and Precautions:

Pigmentation: Pigmentation of the iris, periorbital tissue (eyelid) and eyelashes can occur. Iris pigmentation likely to be permanent.

Eyelash Changes: Gradual change to eyelashes including increased length, thickness and number of lashes. Usually reversible.

Adverse Reactions:

Most common adverse reaction (30% to 50%) is conjunctival hyperemia.

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 please refer to the accompanying brief summary of prescribing information on adjacent page.

Reference:

1. Dubiner HB, Noecker R. Sustained intraocular pressure reduction throughout the day with travoprost ophthalmic solution 0.004%. *Clin Ophthalmol.* 2012;6:525-531.

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TRAVATAN Z[®]
(travoprost ophthalmic solution) 0.004%

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 sternbrae, 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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pressure and associated vascular reflexes.⁴ Specific systemic complications that have been associated with random or deliberate Valsalva events include syncope, alveolar rupture, inner- or middle-ear damage, migraine headache, cerebral hemorrhage and infarct, and even priapism.⁴⁻⁸

Ocular complications typically result from the rupture of small blood vessels in various tissues. Most commonly, this is seen as a non-traumatic subconjunctival hemorrhage. Valsalva retinopathy is another distinct and well-documented ocular manifestation of this phenomenon. All of these complications may be seen with increased frequency in patients who use systemic blood thinners (i.e., warfarin).⁹

The Clinical Appearance

The ophthalmoscopic picture of Valsalva retinopathy can be quite striking (*figures 2 and 3*). Often, the hemorrhage is fairly large, extending across several disc diameters. It is well demarcated and typically round in appearance. Most commonly, the presentation is unilateral, but bilateral cases have been documented.¹⁰

The pre-retinal blood typically is bright red—in stark contrast to deeper retinal hemorrhages encountered in diabetic retinopathy or neovascular membranes associated with macular degeneration—and obscures underlying retinal and choroidal detail. In our experience, most cases seem to involve the macula, explaining why patients often present with decreased acuity.

Fortunately, our patient's hemorrhage was situated in the nasal retina and therefore did not directly interfere with his acuity.

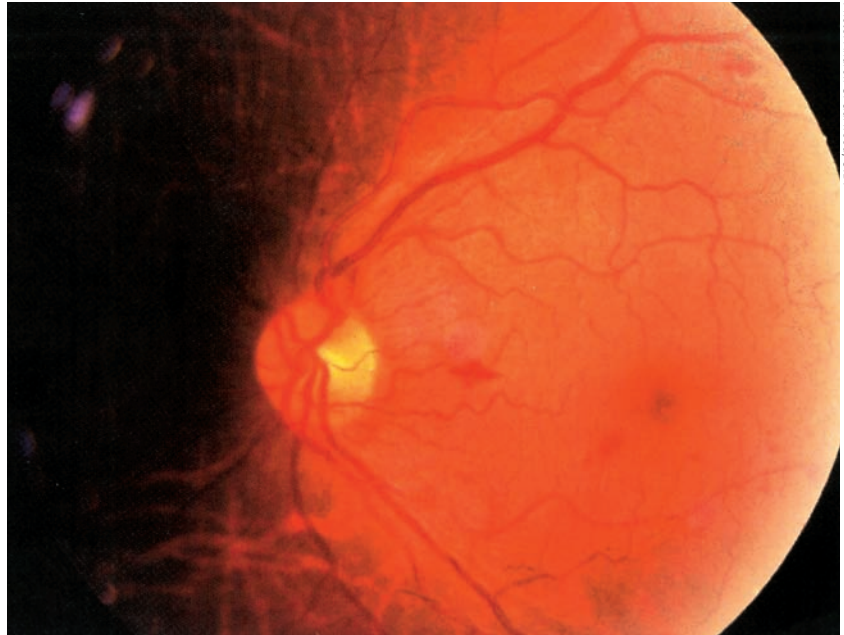


Photo: Andrew S. Gurwood, O.D.

2. This patient exhibited small, superficial hemorrhages following a Valsalva maneuver.

Management Strategies

Management of Valsalva retinopathy begins with a proper diagnosis. The patient history typically is the deciding factor in this regard. However, in cases with confounding elements, associated systemic disease or trauma, you must first rule out such conditions as proliferative diabetic retinopathy, hypertensive retinopathy, Purtscher's retinopathy, Terson's syndrome, retinal vein occlusion, retinal macroaneurysm and hemorrhagic vitreous detachment.¹¹

Once a diagnosis is confirmed, the management approach for Valsalva retinopathy generally is conservative. Advise patients to avoid anticoagulant medications and strenuous activities that could potentially induce a re-bleed. Additionally, instruct them to sleep in a seated or elevated position to promote blood settling, which could potentially improve visual acuity in cases that involve the macula. If appropriate,

antiemetics or stool softeners may be employed.¹²

The typical recovery time for Valsalva retinopathy is six weeks to six months, although larger hemorrhages may take longer to reabsorb.¹³ In more severe or protracted cases, there is always concern that prolonged retinal exposure to hemoglobin and iron may induce cellular toxicity, leading to irreversible visual impairment. In these instances, surgical intervention via Nd:YAG laser is most appropriate. Several case series have been published that describe the utility of laser hyaloidotomy (e.g., puncture of the posterior hyaloid face) to allow drainage of preretinal hemorrhages into the vitreous cavity, thereby expediting resolution.^{11,13-16}

Because our patient's macula was not involved and his vision was only mildly impaired, we didn't initiate therapeutic intervention. We advised the patient

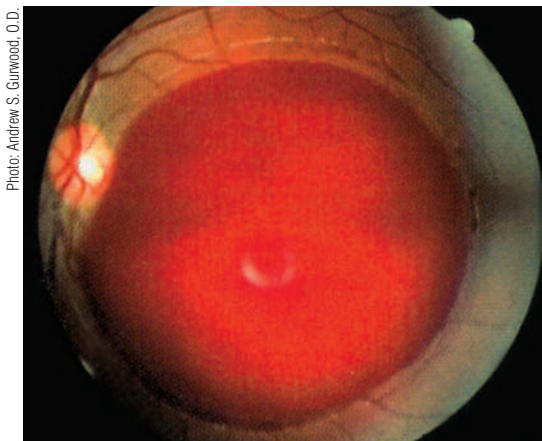


Photo: Andrew S. Gunwood, O.D.

3. Another patient developed a massive preretinal hemorrhage secondary to a Valsalva maneuver.

to restrict his physical activity for several weeks and avoid use of any blood-thinning agents. We also suggested a comprehensive health examination (which revealed no hematological or sero-

logical abnormalities).

“How soon will this annoying spot in my vision go away, Doc?” our patient asked. “Well... don’t hold your breath!” we responded. Fortunately, the hemorrhage, and his vision, resolved fully over the next four months. ■

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Time to Replace the Phoropter?

With the increasing accuracy and reliability of wavefront and point spread function aberrometers, can we finally bid adieu to our oldest standby?

By Paul M. Karpecki, O.D., and Diana L. Shechtman, O.D.

If you look at the modern-day optometric practice, you'll see countless examples of incredible technological devices. From spectral-domain optical coherence tomography to electronic medical records, our offices rapidly are becoming fully automated and computerized. Even our patients are now regularly using smartphones, iPads and all kinds of other electronic gadgets.

It is to the point where the term "manual" is an outdated concept. Yet, one of the most fundamental tools in optometry during the last 80 years has been the completely manual, absolutely non-digital phoropter. But, now that we are beginning to see sweeping technological advances in refraction, is it finally time to replace your "old-school" phoropter?

Out With the Old...

The late Dr. Borish once suggested there were three tests that bother patients the most:

- *Tonometry*, because of drops and the infamous air burst.
- *Visual fields*, because they are tedious and difficult for patients.
- *Looking through the phoropter*, because patients simply don't like it and can't easily differentiate the endpoints.

Advanced diagnostic technologies—including rebound tonometers and ultrafast, automated perimeters—have widely addressed patients' concerns associated with

the first two tests. Only the manual phoropter, as we know it, has yet to be reborn into the 21st century. In order to replace a mainstay tool like the manual phoropter, however, new instruments, such as wavefront aberrometers, must provide at least equal accuracy and efficiency as well as offer room for potential improvement.

Objective Measurements of Visual Distortion

Measurements of wavefront, point spread function and root mean square (RMS) error may all be used to determine the amount of objective visual distortion (e.g., myopia, hyperopia, astigmatism, halo, glare, etc.) associated with an individual's eyes.

- *Wavefront* is a measurement of precisely how light rays pass through a patient's cornea, ocular media and crystalline lens. Wavefront technology analyzes distortions in the patient's vision and may be used to determine his or her overall refractive error.

- *Point spread function* is the smallest achievable point of resolution. This measurement can help illustrate true distortions in a patient's vision.

- *RMS error* is a mathematical calculation that predicts the overall impact of the patient's visual distortions. An RMS error greater than 0.43 is considered significant, and could limit a patient's visual potential with current spectacle lens tech-

nologies for myopia, hyperopia and astigmatism in 0.25D steps.¹

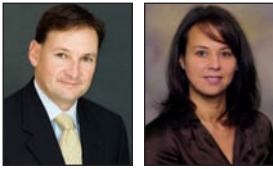
Wavefront Aberration

One study of 200 patients indicated that an individual's best subjective focus occurred when the central, aberration-free region of the pupil was maximized.² Further research showed that objective methods of refraction based on wavefront aberration maps can accurately predict the results of subjective refraction, and actually may be more precise than a manual phoropter.³

Other studies show that eliminating lower-order aberrations, such as myopia, hyperopia and astigmatism, does not necessarily optimize best focus or visual performance.⁴ So, if correcting higher-order aberrations, defocus and astigmatism will yield the best possible refraction, why haven't wavefront aberrometers largely replaced manual phoropters?

One reason is that, without a universally accepted metric of image quality, it can be challenging to convert an aberration map into a prescription.³ Additionally, wavefront devices have difficulty properly measuring highly aberrated eyes, such as those found in patients with a history of keratoconus or laser vision correction.⁵

Then, there is the ultimate subjective variable—we actually see with our brains, not our eyes. So, only the individual patient can truly con-



firm whether the refractive correction is “better or worse.”

But perhaps the main reason that wavefront aberrometers have not overtaken the phoropter is the risk of over-minusing the prescription.⁶ One study in particular showed that wavefront analyzer refractions resulted in 0.30D more myopic accommodation (instrument-induced myopia) than required.⁶

The Technologies

Some of the most widely available devices that can be used to determine objective refraction with great accuracy include: the OPD Scan III (Marco), the KR-1W Wavefront Analyzer (Topcon), iTrace (Tracey Technologies), the Z-View Aberrometer (Ophthonix), the i.Profiler (Carl Zeiss Vision) and the PSF Refractor (VMax Vision). But, can we fully utilize these systems in

A Look at the PSF Refractor

The PSF Refractor from VMax Vision utilizes point spread function to optimize a patient's visual acuity. Point spread function testing requires a subjective refraction, as opposed to objective measurements. By presenting point-spread function images above and below each other, patients generally find it easier to determine the best refractive choice when compared to Snellen-based manual refraction.¹¹

The PSF Refractor measures 0.05D changes, and because it uses point spread images, it automatically corrects for aberrations in the final prescription. Additionally, because a point-spread function image will blur when the patient looks beyond the endpoint—rather than simply appear smaller and darker—the technology prevents over-minusing. Further, researchers have determined that 95% of patients achieved identical or better refraction with the device compared to a manual phoropter.¹¹

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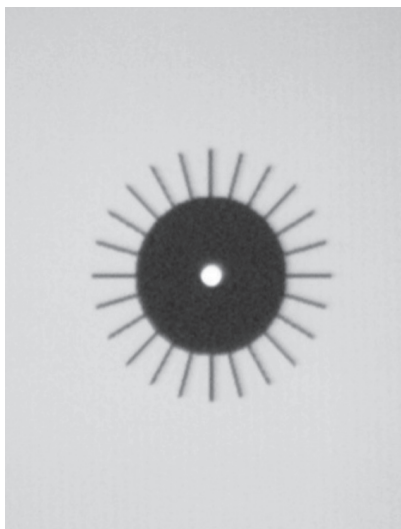
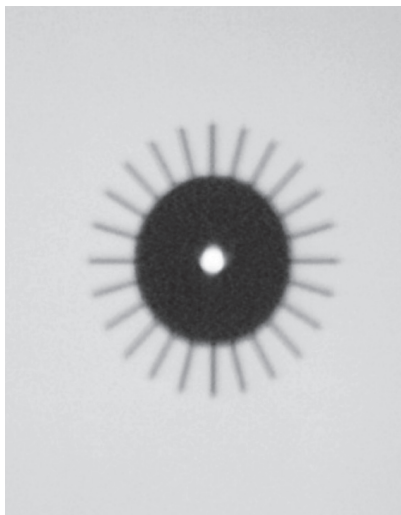
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Examples of the targets used during point spread function refraction.

our practices, given the potential limitations associated with highly aberrated eyes, neural pathways and over-minusing? Or, are these technologies now advanced enough to overcome these shortcomings?

Research has indicated that, while higher-order aberrations influence the amount of sphere and cylinder required to correct vision, subjective refraction can be predicted from the eye's optics alone by optimizing computed retinal image quality.⁷ Unfortunately, the data also showed that the range of

predictability decreased with greater amounts of higher-order aberrations (e.g., higher RMS numbers).

That said, newer devices, such as the KR-1W Wavefront Analyzer, have been shown to be highly predictable for patients with a history of significant aberrations secondary to dry eye or intraocular lens implantation.^{8,9}

Without question, however, these wavefront refraction devices can be used to increase efficiency in your practice. In my experience, for example, the OPD Scan III allows me to perform a 30-second refraction for nearly 75% of my patients. The refraction involves taking the cylinder and axis data as is, and then adding plus to the sphere to ensure that the patient is not over-corrected.

The remaining 25% of patients typically have RMS numbers greater than 0.43 due to irregular cornea shape or previous refractive surgery, and likely would best benefit from manual refraction. (However, it is worth noting that, for highly aberrated eyes, the iTrace has been shown to obtain accurate measurements with high repeatability.¹⁰) Regardless, being able to accurately refract 75% of your patients in 30 seconds can offer substantial advantages over solely using a manual phoropter.

Current research suggests that we are now at a point when new technological advances in both wavefront imaging and point spread function testing finally will make it possible for us to replace our phoropters.

These automated devices provide equal or greater accuracy compared to a manual phoropter. Additionally, they will help guide patient decisions by comparing the new refraction directly to the previous

one with a single touch of a button (which is difficult to do with a manual phoropter); reduce the rate of repetitive stress injury that many doctors experience; and ultimately make the process of a refraction less stressful on a patient.

It is important to note, however, that eye care providers still will need to assess the final prescription—just as is the case with manual phoropters.

Nonetheless, improved efficiency, more accurate refractive correction orders from patients and the natural patient response to cutting-edge technology should enhance our practices dramatically and finally allow us to replace the oldest piece of equipment in our office. ■

Dr. Karpecki has served as a paid consultant to Topcon, Marco and VMax Vision, and is on the Speakers Bureau at Carl Zeiss Vision. Neither he nor Dr. Shechtman have any direct financial interest in any of the technologies mentioned.

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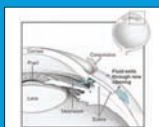
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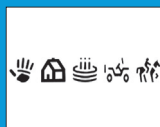
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Product Review

Contact Lenses

Proclear 1-Day Multifocal Daily Disposables

New Proclear 1-Day Multifocal Daily Disposable contact lenses, from CooperVision Inc., are designed to provide presbyopic patients with excellent vision at all distances and address age-related dryness. They offer the convenience of a daily disposable lens for the full-time or occasional wearer, along with all-day comfort and high performance for patients who want an overall, more natural visual experience. The lenses can ease the adaptation that presbyopes encounter and can help prevent contact lens dropouts, the company says.

The center-near aspheric design and simplified fitting approach is designed to make it easier to select the right lens for patients in all stages of presbyopia—whether they are emerging or existing presbyopes. Proclear 1-Day Multifocal lenses feature sphere powers from +6.00 to -10.00 (0.50D steps after -6.00); a base curve of 8.7mm; and a diameter of 14.2mm. They are also designed with a single power profile that can accommodate patients up to +2.50D ADD power. For more info, visit www.coopervision.com/multifocal.

Web Resources

DocBookMD App

Created by Tim Gueramy, M.D., and Tracey Haas, D.O., DocBookMD is a new app that allows doctors to send and receive secure HIPAA-compliant patient information right from an iPhone or iPad. The doctor couple came up with the idea for the app after having many special occasions interrupted by non-urgent calls or questions from the hospital. “The app allows us to more effectively communicate, which means we can more effectively treat our patients,” Dr. Haas says. “We can send and receive things like X-rays, lab results or EKGs and get a response back from another physician that uses DocBookMD in minutes.”

Medical liability companies fund the app for doctors who belong to a medical society in their county or state. All activity is run through a secure server with multiple encryption levels, and the data is stored on the server instead of the physician’s mobile device. Currently, doctors in more than 20 states use the DocBookMD app. It runs on iPhone, iPad and Android. For more information, visit www.docbookmd.com.



Redesigned VSP Website

VSP Vision Care has launched a completely redesigned website at www.vsp.com, making it easier and faster for visitors to find the information most important to them. After listening to more than 100 hours of customer service calls to determine how members and first-time visitors use the website, the newly launched pages now features more intuitive navigation and a fresh look and feel.

VSP expects the new site to reduce customer service calls by at least 10%. Site enhancements include mobile optimization, which makes it easier for members to access their account, promotions and Member ID card while on-the-go, as well as cleaner navigation in both the Benefits Manager and Broker portals, which allows access to the most popular information more quickly.

Diagnostic Equipment

RPS AdenoPlus

The FDA granted waived status under CLIA to the AdenoPlus, an improved version of Rapid Pathogen Screening’s first point-of-care diagnostic test for conjunctivitis. Being granted this waiver classifies AdenoPlus as a low-complexity device, which allows medical office personnel—not just a physician—to administer the test.

Obtaining the CLIA waiver, in addition to the FDA 510(k) clearance it previously received, enables AdenoPlus to be used throughout the United States, an expansion from its current use in many international countries.

AdenoPlus allows for the rapid detection of adenoviral antigens directly from human tears on the inside of the lower eyelid. A nurse or technician can

(Continued on page 133)



Frames

LaCoste Spring/Summer Eyewear Collection

The Spring/Summer 2012 LaCoste Eyewear collection features iconic Petit piqué print forms to the silhouette of uplifting cat eyes. Polished metal details are inlaid onto brows, strengthening a classic profile. The collection ranges from skilled craftsmanship displayed through layered materials and dual colorations



to pure simplicity with striking shapes saturated in rich colors.

- L2142. The LaCoste telescopic temples design of this ophthalmic style features metal end-piece tips that clasp together to allow versatile activity.

Polished metal brows and metal wrapped temples are available in gun-metal, brown, khaki, blue and bronze.

LaCoste Magnetic Collection Video

LaCoste also recently released “Form Meets Function,” a two-minute video that highlights LaCoste Eyewear style L640S.

To demonstrate the Magnetic Collection’s technology, LaCoste will release a magnetic display with the integrated video broadcasting in a continuous loop.

To the left of the inlaid screen, eye care professionals can display four of the 14 sun or optical styles within the collection.



The Magnetic Collection features seven sun and seven optical styles to create a 14-piece collection with innovative technology incorporated into the temples, the company says. Sleek, telescopic temples extend from around the head and clasp with a magnetic closure at end pieces for durability. Available for both men and women, styles range from classic aviators and soft rounds to fill-rimmed metal and translucent zyl optical. All styles are available in vibrant, classic LaCoste colors. For more information, visit www.marchon.com.

Costa Rimless Sunglasses

Galveston, Rockport and Seadrift—three new styles of Costa rimless sunglasses—are designed to provide full eye coverage and a lightweight fit, without compromising durability. Galveston features large, square-shaped lenses for optimal coverage on the water, while Rockport offers thicker, wider temples to block light from entering on the sides.

The smaller, super lightweight Seadrift is ideal for high-impact, velocity sports where speed is key, the company says.

All three styles feature adjustable no-slip Hydro-lite nose pads and Costa’s signature nylon co-injected temple tips. Each is available in Costa’s 580P 100% polarized lens technology, which blocks yellow light from entering the eye in order to



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Color combinations include tortoise frames with copper or silver mirror lenses, or black or silver frames with gray or blue mirror lenses.

They retail from \$169 to \$189, depending on lens selection. All three styles are now available online at www.costadelmar.com and also at authorized Costa retail outlets.

Harley-Davidson Eyewear Collection

The Harley-Davidson Eyewear collection for Spring 2012 from Viva International Group features 10 new styles for men and women, including sun and optical frames. The men's styles evoke the masculine, yet trend-driven quality of the Harley-Davidson man, and deliver a unique mix of fashion-forward styles.

The ladies' collection offers a fashionable blend of design elements, including subtle flame accents and stone embellishments.

The new prescription-ready sunglass models from Harley-Davidson deliver full UV/UB protection, along with sporty, yet fashionable design elements. Visit www.vivagroup.com for more information.



HDX 833



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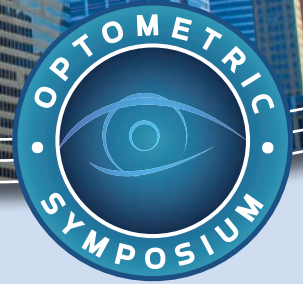
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Frames

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Maui Jim Limited Edition Spiderman Sunglasses

Maui Jim will debut Kekoa (SKU# H427-04W), a new, limited-edition sunglass that will be available exclusively at Sunglass Hut in the United States and at select retailers worldwide when *The Amazing Spider-Man* movie debuts in July 2012.

Emulating Spider-Man's webbed eyewear, the sunglasses feature a lightweight Red Web Grilamid frame and lenses with webbed mirroring. The lenses are lightweight polycarbonate in HCL Bronze.

Someone looking at the glasses can see the webbing on the lenses, but the person wearing them cannot. The lenses feature Maui Jim's proprietary PolarizedPlus2 lens technology, which blocks 100% of UV rays, 99.9% of glare, and enhances colors, the company says.

Just 7,200 sunglasses will be produced; and each sunglass is individually numbered. The glasses come with a custom-branded case, storage pouch and packaging, and are priced at \$219. The eye size is 68mm; bridge size is 13mm; and temple length is 127mm. For more information, visit www.mauijim.com.

Nicole Miller Eyewear

L'Amey America launched the Nicole Miller eyewear collection for Spring/Summer 2012, offering a range of rich colors and on-trend shapes, with 19 ophthalmic styles and 15 sunglasses.

The collection features five themes with elements from each—Celtic, bicycles and bicycle parts, nstar, grafik and material effects. New York City street names are used for each style to reflect Miller's New York City lifestyle.

The Nicole Miller Greene optical and Stanton sun are feminine and delicate with a modern shape and touch of gothic in rich colors. The Astor sun and Varick ophthalmic styles translate the urban chic of NYC in two distinct bicycle-inspired temple details. The collection comes with a corresponding clutch case featuring Nicole Miller's logo and signature nstar pattern in chartreuse on the inside.

Visit www.lamyamerica.com for more information.



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(Continued from page 126)

perform the test when a patient presents with a red eye or other symptoms of conjunctivitis. The patient can then be isolated while they wait just 10 minutes for the result. AdenoPlus demonstrates a clinical sensitivity of 90% and a specificity of 96% when compared against cell culture as the reference method, the company says. They anticipate that AdenoPlus will be available for sale in the U.S. by early August. To learn more, visit www.rpsdetectors.com.

Volk Digital Series Lenses

Volk Optical now offers its Digital Series lenses with new colored ring options for easy identification and organization. The Digital Wide Field, High Mag and Clear Field can now be ordered in standard Volk blue, or with a choice of black, red, silver, gold, purple or green finish.

Colored rings can help practitioners easily distinguish their lenses from those of their colleagues or to identify one style of lens from another with just a quick glance. It's also a convenient way to organize larger practices by color-coding the lenses in each exam room.

The Digital family's three lenses increase general diagnostic capabilities and shorten exam time, the company says. Volk also offers its Pan Retinal 2.2, 20D, Super66, Super Field, 28.00D, 78.00D and 90.00D lenses in a choice of gold, red, green, purple, blue or silver finish.

For more information, visit www.volk.com.



Endurance Tilt Chair and Stand

Reichert Technologies launched the Endurance tilt chair and stand, which has a 45° tilt angle and 360° rotation. It features a Swiss-made, industrial-strength



linear actuator that provides a precise, maintenance-free alternative to common hydraulic lifts, the company says. It has upward rotating armrests, a fully adjustable headrest and a generous seat width designed to provide improved comfort for the patient. Reichert includes a standard two-year warranty on the chair and five years on the actuator.

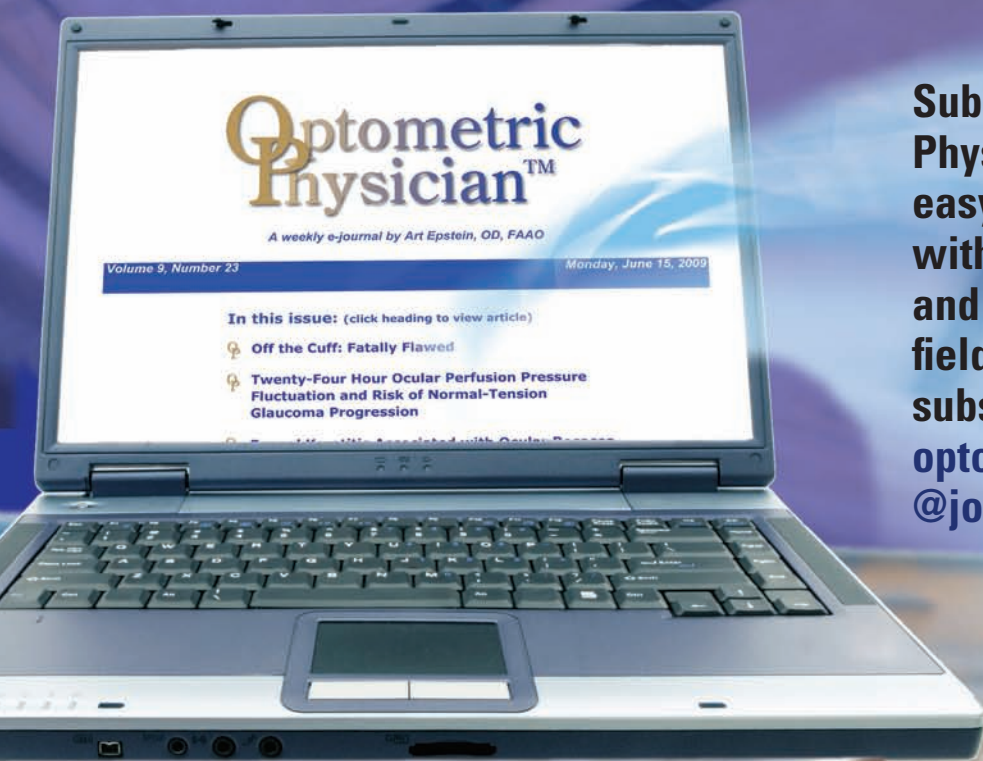
Endurance features three additional accessory outlets and four AC charging wells for the consolidation of power cables for auxiliary equipment, all in one location. The stand comes fully equipped with an overhead lamp, counterbalanced refractor arm and a counterbalanced lower instrument arm that allows for both standard and wheelchair access. An optional keratometer arm is also available. For more information, visit www.reichert.com.

Easyfield C Visual Field Perimeter

The Easyfield C is a real perimeter designed for visual field tests up to 30° eccentricity and is suitable for screening and threshold exams. This compact, lightweight perimeter is capable of performing the threshold tests in less than three minutes, the company

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saves valuable boot-up time. Maintenance is almost non-existent, as it does not contain any light bulbs or moving parts.

The 30cm (11.8") radius bowl of the Easyfield, its background luminance and the size and luminance of the target stimuli all comply with the original Goldmann standard for perimeters.

The Easyfield C comes with a mini laptop and the patient data management software, and the standard package includes the SPARK Light strategy, Threshold Noiseless Trend progression analysis and a Glaucoma Staging Program. The Oculus software is compatible with most EMR systems. For more information, visit www.oculususa.com.

Magnifiers

Task-Vision New Magnifiers

Task-Vision New magnifiers are optical-quality, magnification devices for low vision, the visually impaired, those with macular degeneration, dyslexia, diabetes, glaucoma, cataracts, other diseases of the eye, as well as back and neck pain, osteoporosis, arthritis and a number of neurological disorders.

These high-powered magnification products include: bar magnifiers, digital magnifiers, handheld illuminated and LED magnifiers, handheld and pocket non-illuminated magnifiers, linen testers, loupe magnifiers, mirrors, page magnifiers, pendant magnifiers, stand illuminated and LED magnifiers, stand non-illuminated magnifiers, TV and computer screen magnifiers and bulbs. For professional pricing and information, visit www.techopticsinternational.com. ■

Frames Display

G2 Locking Frame Support

The new Generation 2 (G2) Locking Frame Support from Fashion Optical Displays is designed for eye care professionals who want to deter theft of their inventory. Simply snap each Locking Frame Support onto the system's crystal clear display tubes, then place a frame on the G2 support and close the lock to keep frames in place and secure.

With this new snap-and-go lock support, it is easy to unlock and remove the eyewear for customers to try on or to easily rearrange the display. All that is needed is the unique key that will unlock all of your G2 Locking Frame Supports. The new reasonably priced G2 Supports are easily retrofitted into existing displays and casework. For more information, visit www.fashionoptical.com.



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Meetings + Conferences

June 2012

■ **27-July 1.** *Optometry's Meeting.* McCormick Place West, Chicago. Hosted by: The American Optometric Association and the American Optometric Student Association. Call (866) 229-3691 or visit www.optometrymeeting.org.

July 2012

■ **2-6.** *CE in Belize.* Sunbreeze Hotel, Ambergris Caye, Belize. Hosted by: The International Academy of Optometry. Contact Edward Paul, Jr., O.D., Ph.D., at (910) 256-6364 or epauljr@aol.com. Visit www.CEInBelize.com.

■ **11.** *Summer Seminar.* Ritz Charles Conference Center, Carmel, Ind. Hosted by: The Indiana Optometric Association. Call (317) 237-3560 or e-mail blsims@ioa.org. Visit www.ioa.org.

■ **11.** *2012 Summer Continuing Education Series.* Time: 9:00 a.m. to 4:30 p.m. The Breakers by the Ocean, Spring Lake, N.J. Hosted by: The New Jersey Society of Optometric Physicians. CE hours: 7. Visit www.njsop.org to register.

■ **12-15.** *Colorado Vision Summit.* The Steamboat Grand, Steamboat Springs, Colo. Hosted by: Colorado Optometric Association. Call (877) 691-2095 or e-mail CVSummit@visioncare.org. Visit www.visioncare.org.

■ **13-15.** *OEP/SCO Conference on Clinical Vision Care: Time, Rhythm and the Visual Process.* Southern College of Optometry, Memphis, Tenn. Sponsored by: SCO and The Optometric Extension Program Foundation. Call OEP at (949) 250-8070, or e-mail Howard Bacon, O.D., at hbacon@familyoptometry.net.

■ **18-22.** *44th Annual NOA Convention.* Hyatt Regency, Toronto. Hosted by: The National Optometric Association. Keynote Speaker: Joseph Pizzimenti, O.D. CE hours: 13. Visit <http://www.nationaloptometricassociation.com/convention.html>.

■ **19-21.** *Northern Rockies Optometric Conference.* Snow King Resort, Jackson Hole, Wyo. CE hours: 16. Contact Marian Schulz at (307) 632-8819 or wycschulz@yahoo.com. Visit www.nroc-meeting.com.

■ **19-22.** *Puerto Rico 2012.* Ritz Carlton, San Juan, Puerto Rico. Hosted by: *Review of Optometry*. Meeting chair: Paul Karpecki, O.D. CE hours: 14. Contact Lois DiDomenico at ReviewMeetings@Jobson.com or (866) 658-1772. Visit www.revoptom.com/conferences.

■ **26-28.** *2nd Annual OD Excellence National Conference.* Manchester Grand Hyatt Hotel, San Diego. CE hours: 13. Call (855) 201-1639 or e-mail johanna@odexcellence.com. Visit www.odexcellence.com.

■ **26-29.** *SECO Vancouver 2012.* The Westin Bayshore, Vancouver, British Columbia. CE hours: 14. E-mail info@secostaff.com or visit www.seco2012.com/vancouver.

August 2012

■ **3-4.** *Summer Education Event.* Blue Harbor Resort, Sheboygan, Wis. Hosted by: The Wisconsin Optometric

Association. Call (800) 678-5357 or e-mail joleenwoaoffice@tds.net. Visit www.woa-eyes.org.

■ **3-5.** *32nd Annual Educational Retreat 2012.* South Seas Island Resort, Sanibel, Fla. Hosted by: Southwest Florida Optometric Association Inc. CE hours: 12. Contact Brad Middaugh, O.D., at (239) 481-7799 or swfoa@att.net. Visit www.swfoa.com.

■ **10-11.** *Key West Educational Conference.* Key West, Fla. Hosted by: The Foundation for Ocular Health. Contact Gloria Ayan at gayan@araneye.com or call (305) 491-3747.

■ **19.** *Orlando Super Sunday #1.* Orlando Campus, NOVA Southeastern University, Orlando, Fla. CE hours: 8. Contact Vanessa McDonald, M.S., at (954) 262-4224 or oceaa@nova.edu. Visit <http://optometry.nova.edu/ce/supersunday>.

■ **23-25.** *Idaho Optometric Physicians Association Annual Congress.* The Grove Hotel, Boise, Idaho. Contact Randy Andregg, O.D., executive director, at randregg@frontiernet.net or (208) 461-0001. Visit <http://idaho.aoa.org>.

■ **23-26.** *105th SCOPA Annual Meeting.* Myrtle Beach Marriott Resort & Spa at Grande Dunes, Myrtle Beach, S.C. Hosted by: The South Carolina Optometric Physicians Association. CE hours: 20. Visit <http://southcarolina.aoa.org>.

September 2012

■ **5-8.** *International Vision Expo & Conference West 2012.* Sands Expo & Convention Center, Las Vegas. Call (800) 811-7151 or visit www.visionexpowest.com.

■ **6-9.** *72nd Annual Middle Atlantic Optometric Congress.* Doubletree Hotel and Convention Center, Pittsburgh/Monroeville, Pa. Under the auspices of: The Optometric Extension Program Foundation & the Western Pennsylvania Optometric Society. CE hours: 12. E-mail Barry Cohen, O.D., at barryc51@gmail.com.

■ **6-10.** *The Art and Science of Optometric Care: A Behavioral Perspective.* Grand Rapids, Mich. Held by: The Optometric Extension Program Foundation. CE hours: 35. E-mail Theresa Krejci at TheresaKrejciOEP@verizon.net or visit www.oepf.org.

■ **8-9.** *Fall Conference 2012.* Terry Auditorium, Nova Southeastern University, Fort Lauderdale, Fla. E-mail oceaa@nova.edu or visit <http://optometry.nova.edu/ce/index.html>.

■ **12-15.** *Envision Conference.* Hilton St. Louis at the Ballpark, St. Louis. E-mail info@envisionconference.org or call (316) 440-1530. Visit www.envisionconference.org.

■ **13-14.** *South Dakota Optometry Society Fall Conference.* Hilton Garden Inn, Sioux Falls, S.D. Call (605) 224-8199 or e-mail deb.mortenson@pie.midco.net. Visit www.sdeyes.org.

■ **14-16.** *SWCO 2012.* InterContinental Hotel, Dallas. Sponsored by: The Southwest Council of Optometry. Call Niki Bedell at (713) 743-1856 or e-mail nbedell2@uh.edu. Visit www.swco.org.

■ **14-16, 18-20.** *CE in Italy: Florence and/or Castiglion Fiorentino, Tuscany.* To register for one or both programs, contact James L. Fanelli, O.D., at (910) 452-7225 or jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

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- **21-23.** *New Technology and Treatments in Vision Care.* California. Hosted by: *Review of Optometry.* Meeting chair: Paul Karpecki, O.D. CE hours: 15. Contact Lois DiDomenico at ReviewMeetings@jobson.com or (866) 658-1772. Visit www.revoptom.com/conferences.
- **23.** *CPOS CE Forum XVI.* The Hotel Hershey, Hershey, Pa. Hosted by: The Central Pennsylvania Optometric Society. Featured speakers: Ron Melton, O.D., and Randall Thomas, O.D., M.P.H. CE hours: 6. E-mail Mary Good, O.D., at cposrsvp@gmail.com.
- **27-30.** *GWCO Congress 2012.* Oregon Convention Center, Portland. Hosted by: The Great Western Council of Optometry. CE hours: 59. Visit <http://www.gwco.org/Congress.html>.

October 2012

- **4-7.** *EastWest Eye Conference.* Cleveland Convention Center, Cleveland. Hosted by: The Ohio Optometric Association. Call (800) 999-4939 or e-mail info@ooa.org. Visit www.eastwesteye.org.
- **6-7.** *PSS 2012: 2nd Annual Forum on Ocular Disease.* The Castle Hotel & Resort, Orlando, Fla. Hosted by: PSS EyeCare. CE hours: 18. Call (203) 415-3087 or e-mail education@psseyecare.com. Visit www.psseyecare.com.
- **10-11.** *44th Annual Fall Seminar.* The Lansing Center, Lansing, Mich. Hosted by: The Michigan Optometric Association. Contact Amy Possavino at amy@themoa.org or (517) 482-0616. Visit www.themoa.org.
- **12.** *HVOS Fall Seminar.* The Grandview, Poughkeepsie, N.Y. Hosted by: The Hudson Valley Optometric Society. Contact Robert Greenbaum, O.D., at robertgreenbaum58@gmail.com or (845) 473-0220. Visit www.hvos.org.
- **12-13.** *Northwoods Education Events.* Black Bear Lodge, St. Germain, Wis. Hosted by: The Wisconsin Optometric Association. E-mail joleenwoaoffice@tds.net or (800) 678-5357. Visit www.woa-eyes.org.
- **13-14.** *Fall Conference.* Lansdowne Resort, Leesburg, Va. Hosted by: The Virginia Optometric Association. Call (804) 643-0309 or visit www.thevoa.org.
- **16-20.** *COVD 42nd Annual Meeting.* Omni Fort Worth Hotel, Fort Worth, Texas. Hosted by: The College of Optometrists in Vision Development. Contact info@covd.org or (330) 995-0718. Visit www.cvod.org.
- **24-27.** *Academy 2012 Phoenix.* Phoenix Convention Center. Hosted by: The American Academy of Optometry. Visit www.aaopt.org/meetings/academy2012. ■

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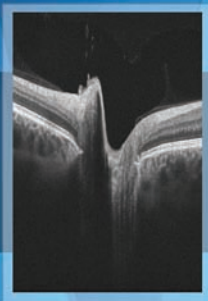
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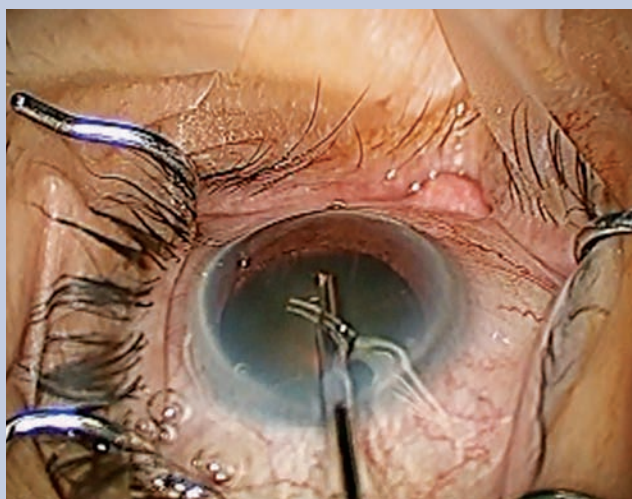




Trabectome

This procedure can be a great stand-alone option, or it can be combined with cataract surgery. Read on for what every O.D. needs to know about it, and visit *Review of Optometry* online for a video demonstration of the procedure.

By **Derek N. Cunningham, O.D.**, and **Walter O. Whitley, O.D., M.B.A.**



Go to www.revoptom.com to see video footage of this minimally invasive glaucoma procedure.

On The Web >> View a narrated video of this procedure on a patient with glaucoma.

The Trabectome (NeoMedix) procedure is becoming an increasingly popular surgical option for my glaucoma patients who concurrently suffer from visually significant cataracts; however, it can be performed as a stand-alone as well.

The Trabectome is an FDA-approved device for the minimally invasive surgical treatment of open-angle glaucoma. The procedure is designed to improve fluid drainage from the eye, with clinical results that show a 33% decrease in postoperative IOP. This ab interno trabeculotomy combines an electrosurgical device with irrigation, aspiration and a protective footplate to ablate and remove a 60° to 120° strip of trabecular meshwork and the inner wall of Schlemm's canal.

The surgeon starts with a clear corneal incision and then places a gonio lens onto the cornea to verify the angle anatomy. The Trabectome device is advanced across the anterior chamber and inserted into Schlemm's canal anterior to the scleral spur. Ablation of the trabecular meshwork is performed up to 120° of the angle with continuous irrigation and aspiration.

Once complete, the surgeon will irrigate and aspirate the remaining viscoelastic material from the anterior segment. If performed as a stand-alone procedure, a dissolving suture can be applied and the anterior chamber re-pressurized. Otherwise, the surgeon can continue on to the cataract procedure.

This procedure is a great option for patients who have early-to-moderate stages of glaucoma. Ideal candidates for this procedure include those who demonstrate progression despite maximal medical therapy, are already using one or two IOP-lowering medications, require target pressures in the mid-tens or exhibit cataract development.

This procedure has several advantages. First, it's non-penetrating; there is no disturbance of the conjunctiva. It also requires no bleb and is easily combined with cataract extraction. We are seeing lower complication rates with this procedure, which is a huge advantage with any surgery. And, the outcomes result in a reduction of glaucoma medications. This procedure also requires fewer follow-up appointments than other glaucoma procedures.

That said, there are some distinct disadvantages to the procedure as well. Notably, some 20% experience a postoperative IOP spike. Additionally, postoperative hyphema is typical. Also, it can result in synechia formation around the cleft and/or cause an injury to Descemet's membrane. And, the cost of equipment can be an obstacle for many providers.

Preoperatively, candidates require a full glaucoma work-up with ancillary testing (visual fields, pachymetry, optical coherence tomography, etc.). Postoperative care is similar to traditional cataract surgery, with the exception of topical miotics prescribed b.i.d. to q.i.d. for three to four weeks. ■

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Persistent Ocular Discomfort

By Andrew S. Gurwood, O.D.

History

A 48-year-old black male presented to the emergency department for follow-up care after being seen by an ophthalmologist.

His chief complaint was discomfort in his left eye, which was accompanied by both watering and light sensitivity.

His systemic history was unremarkable, and he reported no known allergies.

His current medications included 500mg Valtrex (valacyclovir, GlaxoSmithKline) t.i.d. and erythromycin ointment O.S. qhs, which presumably were prescribed by the

ophthalmologist for a herpetic simplex outbreak.

Diagnostic Data

His best-corrected visual acuity was 20/20 O.D. and 20/25 O.S. External examination was normal, and there was no evidence of afferent pupillary defect O.U. An anterior chamber reaction was observed O.S.

His intraocular pressure measured 16mm Hg O.U. The dilated fundus exam was normal, with quiet nerves, grounds and peripheries O.U. The pertinent anterior segment examination findings are

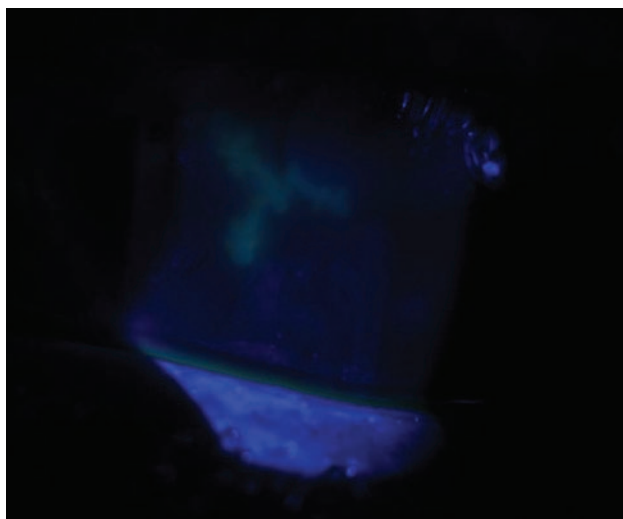
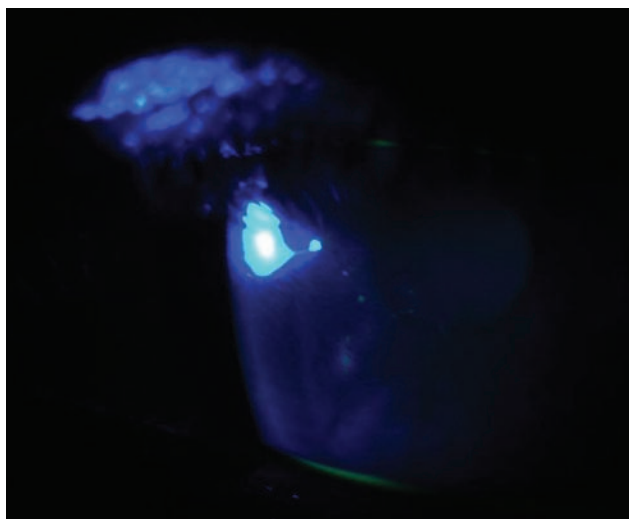
illustrated in the photographs.

Your Diagnosis

How would you approach this case? Does this patient require any additional tests? What is your diagnosis? How would you manage this patient? What's the likely prognosis?

To find out, visit www.revoptom.com. Click on the cover icon for this month's issue, and then click "Diagnostic Quiz" under the table of contents. ■

Thanks to Alissa Coyne, O.D., of Philadelphia for contributing this case.



Our patient's left eye at initial presentation (left) and three days after we began therapeutic intervention. What is your diagnosis?

Retina Quiz Answers (from page 112): 1) a; 2) d; 3) c; 4) a.

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¹ Luthe, R. Supplemental Information. *Ophthalmology Management*. November, 2010.

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