Case Report: How to Identify and Confirm Multifocal Pattern Dystrophy, **p. 66**

REVIEW® OF OPTOMETRY

December 15, 2015

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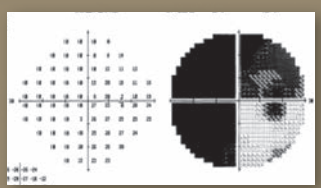

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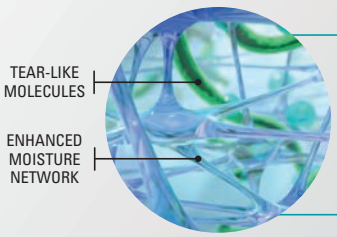

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VOL. 152 NO. 12 ■ DECEMBER 15, 2015

IN THE NEWS

Researchers have successfully brought **donor retinas** back to life for hours after death, suggesting a new method of **preserving human retinas** for research and transplant. The technology could reduce the use of animals for vision research and take a big step to one day **transplanting human retinas**.

Auven Therapeutics recently announced results from its Phase 2b/3 clinical trial evaluating the safety and efficacy of Seciera, a formulation of cyclosporine, for the treatment of **dry eye disease**. The study shows a **significant increase in tear production** and a **reduction in signs of ocular surface inflammation** compared to placebo-vehicle. Depending on upcoming studies, the company plans to submit a New Drug Application for **Seciera** in early 2017.

The results of a recent meta-analysis found the **risk of any-stage ROP appeared to drop by about 75%** for babies exclusively fed breast milk. **The risk of severe ROP** seemed to be reduced by 90%. The studies indicate that breastfeeding in any amount appeared to reduce the risk of ROP, and the more breast milk, the better.

The Accreditation Council on Optometric Education recenlty granted the **University of Pikeville-Kentucky College of Optometry** the pre-accreditation classification of **preliminary approval**. "With no other colleges of optometry in Kentucky, West Virginia, Virginia, North Carolina, South Carolina or Georgia, Kentucky College of Optometry will be the **most accessible college of optometry in the Southeastern portion of the country**," the university said in a press release.

Anti-VEGF as a Treatment for PDR

Research suggests ranibizumab can be just as effective as PRP for patients with proliferative diabetic retinopathy.

By Rebecca Hepp, Senior Associate Editor

A study recently published in *JAMA* and presented at the American Academy of Ophthalmology annual meeting showed that treating proliferative diabetic retinopathy (PDR) patients with injections of the anti-VEGF agent ranibizumab had a non-inferiority (not worse than) outcome of visual acuity change after two years compared with the PRP group. Understanding that PRP often damages the retina, researchers with the Writing Committee for the Diabetic Retinopathy Clinical Research Network conducted a study of 394 eyes with PDR to explore less damaging treatment options.

Individual eyes were randomly assigned PRP treatment, completed in one to three visits, or treatment with ranibizumab by intravitreous injection at study entry and as frequently as every four weeks. The mean visual acuity letter improvement at two years was +2.8 in the ranibizumab group vs. +0.2 in the PRP group, meeting the study's predetermined noninferiority outcome.

In addition to comparable visual acuity outcomes, they found more peripheral visual field loss occurred, more vitrectomies were performed and diabetic macular edema (DME) development was more frequent in the PRP group compared with the ranibizumab group, resulting in those participants also receiving ranibizumab to treat DME.

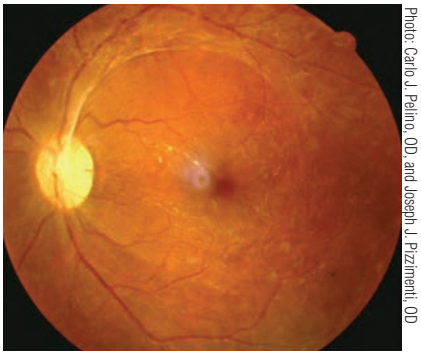

Photo: Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

This patient with proliferative diabetic retinopathy had a hemoglobin A1c of 9%.

"This article confirmed many of our clinical experiences," says Nate Lighthizer, OD, assistant professor and assistant dean of Clinical Care Services at Oklahoma College of Optometry. "With these results, perhaps treating ophthalmologists and referring optometrists have decisions to make on which procedure to perform and refer for."

"The follow-up was only for approximately two years, so longer follow-up studies are needed," he adds. "This study represents, however, a groundbreaking finding that may change the protocol for treating PDR."

Gross JG, Glassman AR, Jampol LM, et al. Writing Committee for the Diabetic Retinopathy Clinical Research Network. Panretinal photocoagulation vs intravitreous ranibizumab for proliferative diabetic retinopathy: A randomized clinical trial. JAMA. 2015 Nov 13:1-11. [Epub ahead of print].

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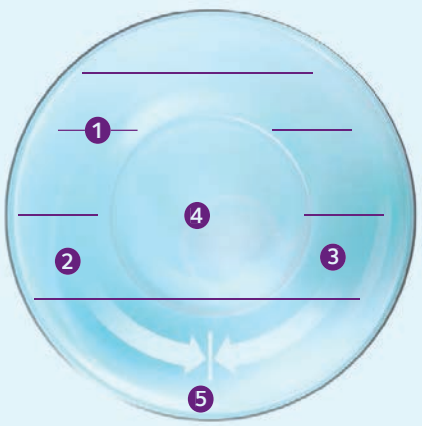

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Not-so Novel Dry AMD Treatment

Research now suggests treatments currently used for other conditions such as wet age-related macular degeneration (AMD) could also work for dry AMD. Using a mouse model, investigators from the Gifu Pharmaceutical University found that injecting placental growth factor (PlGF) into the retinas of mice before and after exposure to intense light—a procedure that mimics dry AMD-like conditions—aggravated the degeneration, contradicting previous study results.

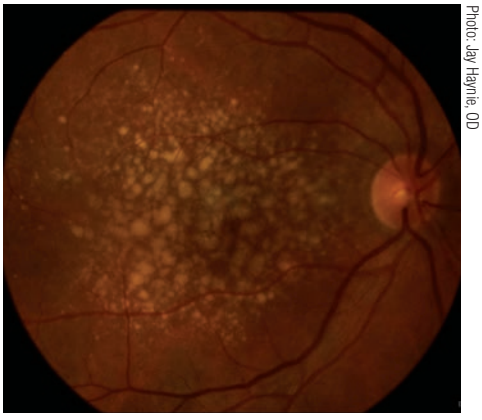

Photo: Jay Haynie, OD

Soft drusen seen in a patient with AMD.

In response, they tried injecting anti-PlGF, an antibody already found in the wet AMD treatment aflibercept, and found it protected against retinal degeneration induced by light exposure.

"There is a very great likelihood that aflibercept shows efficacy in dry AMD," study author Hideaki Hara, PhD, of Gifu Pharmaceutical University's Department of Biofunctional Evaluation, said in a press release.

"Currently, there is no effective treatment for dry AMD, and our treatment revolves around lifestyle modification, nutrition/supplementation and monitoring for conversion to wet AMD," says Steven Ferrucci, OD, of the US Department of Veterans Affairs in North Hills, Calif., and professor at the Southern California College of Optometry at Marshall B. Ketchum University in Los Angeles. "It is possible that this already-approved medication could have some positive effects in dry AMD patients based on this study." "Repurposing an existing drug in such a manner could save time and money, rather than developing a novel drug," he adds. "However, it must be understood that this is an animal model only, and it would be a long time until we know whether this is a viable treatment in humans."

Still, the new findings highlight the importance of research into treatment options for dry AMD, as well as staying abreast of breakthroughs that may benefit patients with this disease.

Izawa H, Inoue Y, Ohno Y, et al. Protective effects of antiplacental growth factor antibody against light-induced retinal damage in mice. Invest Opthalmol Vis Sci. 2015;56(11):6914.

Can Eye Drops Slow Myopia in Kids?

A five-year clinical trial, the results of which were presented at the American Academy of Ophthalmology annual meeting, suggests treating children with low doses of atropine could slow the progression of myopia by as much as 50%.

Four hundred children ages six to 12 were randomly assigned a nightly dose of 0.5%, 0.1%, or 0.01% atropine for two years. After stopping treatment for one year, investigators started another round of 0.01% atropine for two more years for children who became more myopic during the year off.

The results show that, after five years of treatment, children using the low-dose 0.01% atropine were the least myopic when compared with those treated with higher doses. They further mention an earlier study, which revealed 0.01% atropine eye drops slowed myopia progression by an estimated 50% compared with untreated children.

While low doses of 0.01% atropine appear safe enough to use in children ages six to 12 for up to five years, the researchers stress the need for more study. Higher concentrations of atropine have been known to cause light sensitivity and blurry vision when looking at objects up close, and can also cause allergic conjunctivitis and dermatitis. These risks explain why atropine use for myopia is fairly uncommon in the United States, but the investigators suspect this trend could change now that much lower doses appear to offer a similar benefit in reducing nearsightedness progression, without the side effects.

Chia A, Lu QS, Tan D. Five-year clinical trial on atropine for the treatment of myopia 2: myopia control with atropine 0.01% eyedrops. Ophthalmology. 2015 Aug 11. [Epub ahead of print].

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Amblyopia Treatment Goes High-tech

A new study, presented at the American Academy of Ophthalmology annual meeting in November, found that programmable electronic glasses help improve vision in children with amblyopia just as well as patching. Researchers at the Glick Eye Institute at Indiana University completed a randomized clinical study of 33 children with amblyopia between the ages of three and eight who wore spectacles to correct their vision.

One group was patched for two hours daily, while the other wore occlusion glasses for four hours daily. During the study, the lens over the eye with better vision switched from clear to opaque every 30 seconds. The study found both groups gained two lines on a reading chart after three months to treatment.

"When you talk to adults who had to do patch therapy as a child, they universally comment about how much they hated it, and with good reason," said Daniel Neely, MD, a pediatric ophthalmology professor at Indiana University who led the study. With the electronic occlusion glasses, "the upside is, because the good eye is covered for only 30 seconds at a time, the child may be less distressed and more compliant with the treatment. Not only does this make it more 'fun' or tolerable, but it may improve outcomes if the compliance is better."

Photo: XpanD

Researchers studied the effects of using electronic glasses rather than patching to treat amblyopia.

While the study only involved children with moderate amblyopia (20/40 to 20/100), the researchers hope to study effects of the new treatment option on patients with worse amblyopia or those whose treatment had failed in the past. They also hope to explore the outcomes after shortening the treatment intervals, possibly making "the treatment even more tolerable with almost seamless transitions and limited impact on the child's function at play and school," Dr. Neely said.

The glasses used in the study, Amblyz occlusion glasses (XpanD), have been approved by the FDA as medical devices.

Why Birth Order Affects Myopia

First-born children are more likely to be more nearsighted than their younger siblings, according to new research published in *JAMA Ophthalmology*. Investigators at Cardiff University's Eye Clinic in the United Kingdom used data from the British Biobank longitudinal survey to study nearly 90,000 adults between the ages of 40 and 69. Participants had a vision assessment, reported no history of eye disorders and shared potential confounders such as highest educational qualification through questionnaires. The researchers defined myopia and high myopia as autorefraction of –0.75D or less and –6.00D or less, respectively.

Using this information, they found that firstborns were 10% more likely to develop myopia and 20% more likely to show signs of severe myopia. "The risk for myopia became progressively lower for later birth orders, suggesting a dose response," the researchers wrote in the study.

The investigators further found that adjusting for education changed the results considerably: the dose response disappeared once they took each child's education into account.

While the researchers are leery of claiming a direct correlation, they say the results support "a role for reduced parental investment in education of children with later birth orders in their relative protection from myopia." ■

Guggenheim JA, Williams C, UK Biobank Eye and Vision Consortium. Role of educational exposure in the association between myopia and birth order. JAMA Ophthalmol. 2015 Oct 8:1-7. [Epub ahead of print].

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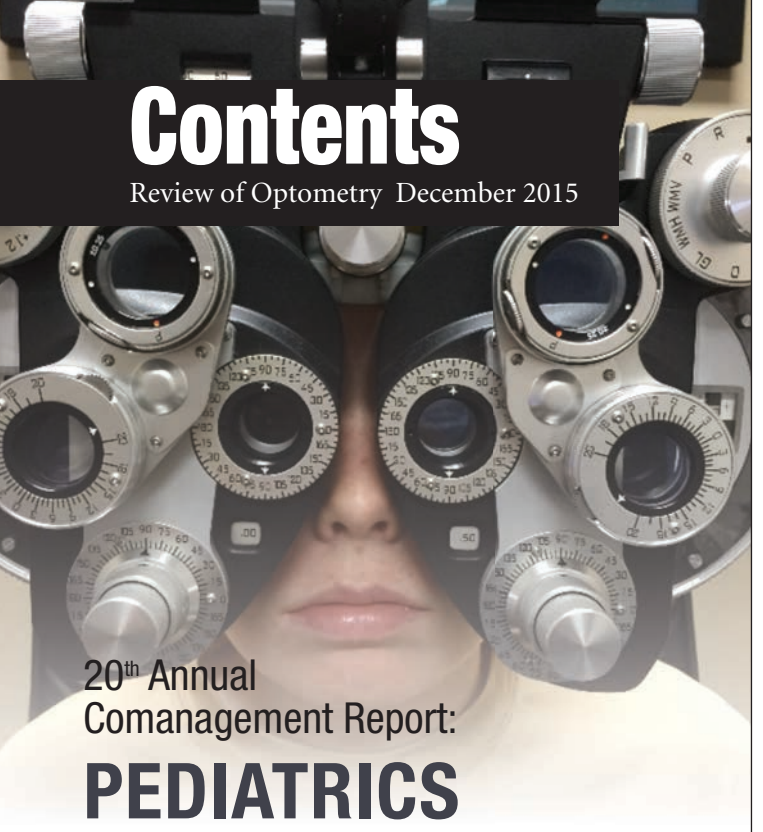

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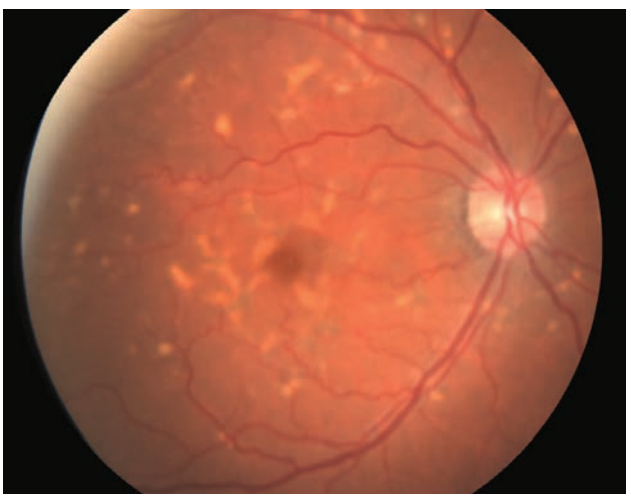
PEDIATRICS



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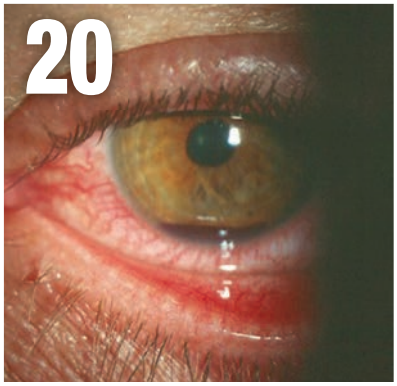

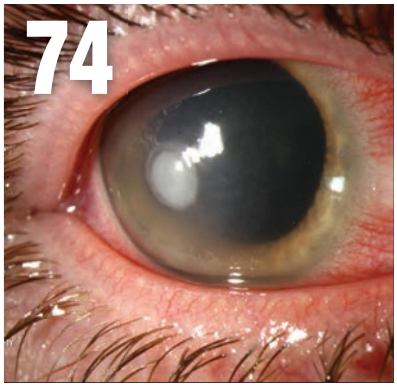

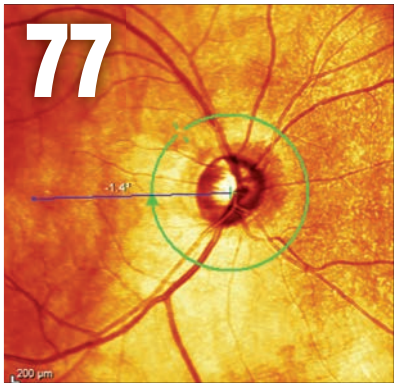

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Down, Boy.

Help Tame Postoperative Ocular Inflammation and Pain With LOTEMAX® GEL

Indication

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

Important Safety Information about LOTEMAX® GEL

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

Please see brief summary of Prescribing Information on adjacent page.

LOTEMAX® GEL
loteprednol etabonate
ophthalmic gel 0.5%

BAUSCH + LOMB

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

Lotemax (loteprednol etabonate ophthalmic gel) 0.5%

Rx only
Initial Rx Approval: 1998

INDICATIONS AND USAGE

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

DOSAGE AND ADMINISTRATION

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

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

CONTRAINDICATIONS

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

WARNINGS AND PRECAUTIONS

Intraocular Pressure (IOP) Increase

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

Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing

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

Bacterial Infections

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

Viral Infections

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

Fungal Infections

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

Contact Lens Wear

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

ADVERSE REACTIONS

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects: Pregnancy Category C.

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

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

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

Nursing Mothers

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

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment Of Fertility

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

PATIENT COUNSELING INFORMATION

Administration

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

Risk of Contamination

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

Contact Lens Wear

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

Risk of Secondary Infection

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

Bausch & Lomb Incorporated
Tampa, Florida 33637 USA

US Patent No. 5,800,807

US/LGX/15/0042
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The Kids are Alright

Or are they? Vision screenings are no substitute for a true eye exam. **By Jack Persico, Editor-in-Chief**

The phrase *quality-adjusted life year* (QALY) is something only a statistician could love. It may not be the most accessible concept, but it's a vital part of the equation in how health interventions are measured and policy is made. The goal is to work out the cost/benefit ratio of medical interventions by comparing their price tag against the impact on both the quality of life *and* duration of effect on the individual. Efforts that improve the lives of children usually do well in such an analysis because of the potential for lifelong improvement; the same isn't always true in older patients.

In one report, universal eye exams for kids come in at $18,930 QALY and (in a separate, older study) ambylopia treatment specifically is priced at just $2,281 QALY. By contrast, liver transplant has been pegged at $350,000 QALY, given its phenomenal cost and the low life expectancy of the patient.

Children's health should be taken care of as a moral value regardless of cost. But when the interventions are also a bargain—as most routine eye care is—it's a no-brainer. So, it's all the more distressing that this great opportunity is being missed for many kids in need.

"Our nation's historical misreliance on vision screening has plagued our education, mental health and juvenile justice systems for too long, serving as major contributive factors to children's inability to perform and conform to the demands of school and society," says the AOA in a recent statement.

Vision screening (vs. a full exam), "by falsely telling too many children that they have no vision problem, when they actually do, has long closed the door to vision health and education opportunities," says the AOA, noting that screenings have a sensitivity of just 27%.

The Affordable Care Act made preventive care a priority, and pediatric vision is a part of its essential health benefit. But one small study, presented at the recent Academy of Optometry meeting, found that only 56% of optometry practices have seen ACA patients. ODs in solo optometric practice had the lowest participation rate (45.5%) and those in group OD/MD practice had the greatest (61%).

We're focusing on pediatric eye care in this month's 20th annual comanagement report to help bring attention to these matters.

"Optometrists will have the chance to make history because of the profession's unique position to provide greatly needed services," says Kathleen Foster Elliott, OD, in her article on the ACA and pediatric eye care, which opens our series this month (*see page 32*). In her article and the ones to follow, experts give concrete advice on how to make routine pediatric eye care an essential part of your practice. It'll take outreach to community leaders, pediatricians and local ophthalmologists, but the need is too great to ignore. Only 14% of all children have had an eye exam by age six, and 10 million have undetected vision problems. A little extra effort could make a tremendous difference. ■

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Back to the Future, or the Past?

Who knew a direct ophthalmoscope could still be my most valuable tool in the age of high-tech doodads? **By Montgomery Vickers, OD**

It was recently *"Back to the Future* Day." I'm not sure what that means, but I seem up-to-date on the news by mentioning it, don't you think? But the phrase "back to the future" began to mean a lot more to me once I started working in two starkly different worlds this past month.

Back to the Past

On Mondays and Thursdays, I work with a traveling nursing home practice. This is immensely important work, and I salute doctors who do this once in a while, part-time or full-time. They are true healers, physically and spiritually.

But these days take me back to the past. It was humbling to realize my biggest challenge was the lowly—but revered—direct ophthalmoscope. I had barely touched one in 20 years. I felt like an idiot and, yes, thank you for asking, at least a dozen times have begun the procedure like any first-year OD student, blinding myself by shining the light into my eye. On the chart where you state the quality of the view, I've had to mark "poor due to miosis—my miosis." Geez. And have you ever tried to epilate lashes using a hand-held slit lamp? OK, Dr. Shaky-hands, go ahead and try.

Back to the Future

On Tuesdays and Fridays, I am back to the future in our high-tech private practice with all the bells and whistles. My practice was pretty darn modern back in West Virginia, but here we have these gizmos that show detail in the retinal layers that make my OCT look like connect-the-dots. When patients ask, "What's that a picture of?" I just say, "How about them Cowboys?" and they seem to forget their inquiry.

We work hard to get to a place where we have a handle on the testing, findings, diagnoses and treatments for everything—from patients who accidently put two contacts on one eye and complain they can't see right to patients who will only see right if they put two contacts on one eye, and by "see right" I mean their vision sucks less.

Then some no-account researcher figures out we can spend as much as a nice car costs on some doodad that drags us back to the future, when the definition of "future" is "a time when the doctor's career-long ignorance is revealed by this new doodad."

Lessons of the Past

But working in the nursing home, I see that a touch on the shoulder does way more than using secret rays of light to reveal the eye's mysteries. The world of optometry vacillates between knowing the patient and knowing the vision plan.

Are efficiency and reduced costs the most important things in the future? Maybe we should put the care back in eye care and see a single patient every six hours like I did when I first started. An eye exam would cost $10,000 to support my high-tech equipment and beer supply. And yes, I had days with only two patients: Mom and Dad.

As I traverse these two worlds of optometry, I am reminded that, at the end of the day, all the patient wants is someone who actually gives a crap about how they see. Whether it's back to the past or the future, either way you better make 'em see right. That's the deal.

So, quit pushing buttons and ask the patient exactly what they need to see. You can use your fancy doodads to figure stuff out, but at the end of the day, it comes down to whether they can read the hymnbook on Sunday. ■

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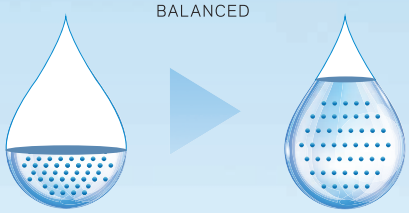

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Reference: 1. Research in dry eye report of the Research Subcommittee of the International Dry Eye WorkShop (2007). *Ocul Surf.* Apr 2007; 5(2): 179-193.

AKORN Consumer Health

War Paint

Sports can be all fun and games until someone winds up with ocular trauma.

By Richard Mangan, OD

The popularity of paintball—a strategic "war game" where the goal is to eliminate opponents by "tagging" them with gelatin-covered capsules containing a non-toxic, water-soluble dye—has grown immensely and is even used in military, law enforcement and security training. The paintballs themselves are propelled from a "gun" (referred to as a paintball marker), which uses a compressed air or CO_2 tank. The muzzle velocity from a compressed CO_2 powered paint gun can meet or exceed 300 ft/s. Eye doctors will naturally recognize the game's distinct risk: ocular injury. In fact, since the game's popularity has grown, the incidence of ocular injury has increased significantly. Optometrists aren't the only ones picking up on this. The American Society for Testing and Materials includes eye protective devices among its minimum safety requirements for paintball games due specifically to the high risk of ocular injury. Still, most paintball-related ocular injuries occur when players neglect or remove protective eyewear.[4]

This article reviews managing patients who present with urgent injuries sustained from these, and other sports-related, activities.

Casualty Presentation

An 18-year-old male with a right eye injury presented with entrance acuity at the level of hand motion (HM) and an intraocular pressure (IOP) of 17mm Hg. An anterior segment examination revealed right upper lid edema and bruising. A slit-lamp examination confirmed corneal edema with mild folds centrally, a subconjunctival hemorrhage with chemosis and 3+ cell and flare with a 2mm hyphema in an adequately formed anterior chamber. The cornea and conjunctiva were Seidel negative. The right pupil was 5mm with poor reactivity. The lens appeared centered, but it was difficult to assess with clarity. Funduscopic exam was limited due to severe vitreous hemorrhage. A B-scan ultrasound did not suggest retinal detachment.

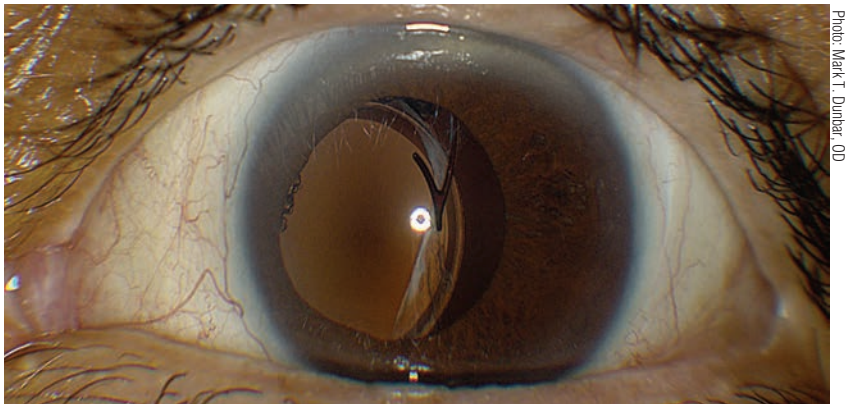

Photo: Mark T. Dunbar, OD

This slit lamp image shows traumatic IOL dislocation, a potential complication of ocular trauma, such as being struck with a projectile paintball.

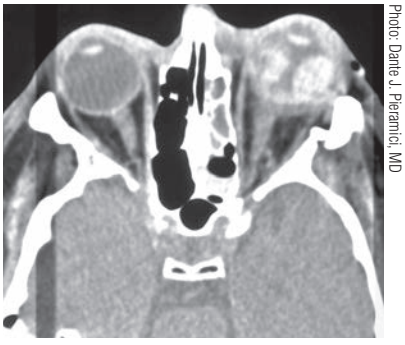

Photo: Dante J. Pieramici, MD

This CT scan reveals a ruptured globe following ocular trauma. It can be difficult to make such a diagnosis without a CT scan if profuse hemorrhaging obscures visibility.

Learn more about the prevelence of ocular paintball injury at www.reviewofoptometry.com or scan the QR code.

The patient was referred to our staff retinal specialist the next day. A biometry was repeated and, once again, did not suggest retinal detachment.

However, as the vitreous hemorrhage began to clear, a fundus examination did reveal a retinal detachment in the superior quadrant with an associated sub-macular choroidal rupture. A pars plana vitrectomy (PPV), lens extraction and IOL implantation were performed. In the patient's final examination, the retina was attached and his visual acuity was recorded as 20/200.

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Reference: 1. Patel S, Henderson R, Bradley L, Galloway B, Hunter L. Effect of visual display unit use on blink rate and tear stability. *Optom Vis Sci.* 1991;68(11):888-892.

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Types of Trauma

Paintball-related ocular trauma most frequently consists of coup and contrecoup trauma resulting in a significant increase in IOP and equatorial expansion. Blunt trauma from paintball projectiles can cause anterior segment injury resulting in hyphema, traumatic cataract, crystalline lens dislocation, angle recession and iridodialysis. Posterior segment injury may include vitreous hemorrhage, commotio retinae, choroidal rupture, traumatic macular hole and retinal tear or detachment. Scleral rupture related to a suspected centerline hit at relatively close range has also been reported.[5,6]

A Bascom Palmer data analysis found 28% of patients with paintball-related eye injuries suffered from a ruptured globe, 19% had a retinal detachment, 81% required surgery and, ultimately, 22% of eyes required enucleation.

When confronted with a patient who has suffered a sports-related traumatic ocular injury, be sure to determine the nature of the injury. The impact from larger objects, such as basketballs, soccer balls, boxing gloves, etc., are likely to be partially absorbed by the periorbital rim. These objects can still cause significant ocular injury, but smaller and fast-traveling objects like paintballs have greater potential to adversely affect the globe.

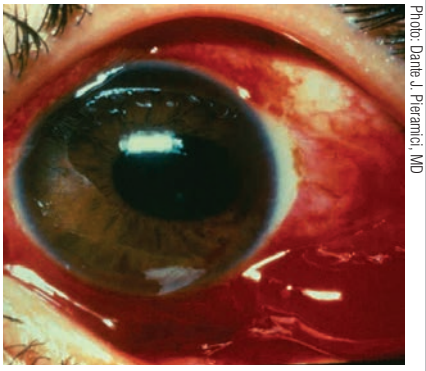

Photo: Dante J. Pieramici, MD

This ocular hemorrhage following trauma may be accompanied by a ruptured globe.

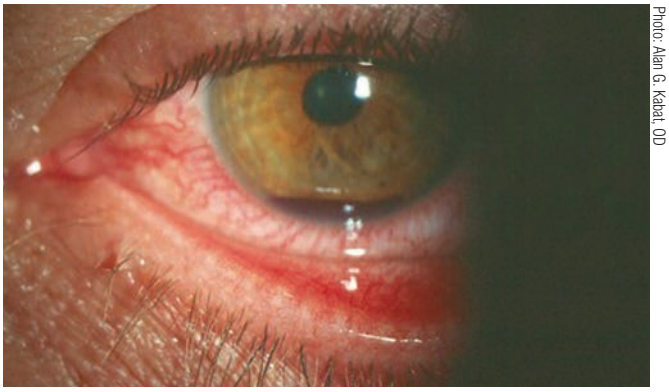

Photo: Alan G. Kabat, OD

Hyphema, like the kind seen in this slit lamp photograph, is often caused by blunt trauma, such as being struck with a projectile paintball.

Friendly Fire

In our case, the patient indicated that he was wearing an eye protective device during an organized paintball competition. When the signal sounded that the game was over, he immediately took his mask off and was struck by an errant round. He did not know the distance or direction from which the projectile was fired.

Assessment of an eye immediately following such an injury can be difficult due to significant edema, haze or blood obscuring the view. As this case shows, even B-scan ultrasound can be inconclusive when addressing posterior segment damage.

Ordering restricted activity, proper head positioning (minimum 45 degree upright angle) cycloplegics, IOP lowering agents, a Fox shield and close monitoring are sometimes warranted until the view of ocular structures improve.

The main goal at the initial visit is to rule out signs of a ruptured globe. Risk factors for an open globe include penetrating lid injury, hyphema, hypotony, peaked pupil, shallowed or absent anterior chamber, 360 degree bullous subconjunctival hemorrhage, iridodialysis, lens dislocation, relative afferent pupillary defect, positive Seidel test and significant reduction in vision.[7] If you suspect a ruptured globe, consult a retinal specialist immediately. The patient will likely be started on oral antibiotics and scheduled for exploratory or globe reconstruction surgery within 24 hours.

Not all ruptured globe cases will present with each and every finding listed above. If there is any question regarding the integrity of the globe, consider a CT scan of the eye and orbit, or refer.

Finally, make sure all patients participating in eye-threatening sports are educated on the importance of wearing eye protection and—take a lesson from our patient—remind patients not to remove their EPD until they have exited the field of play. ■

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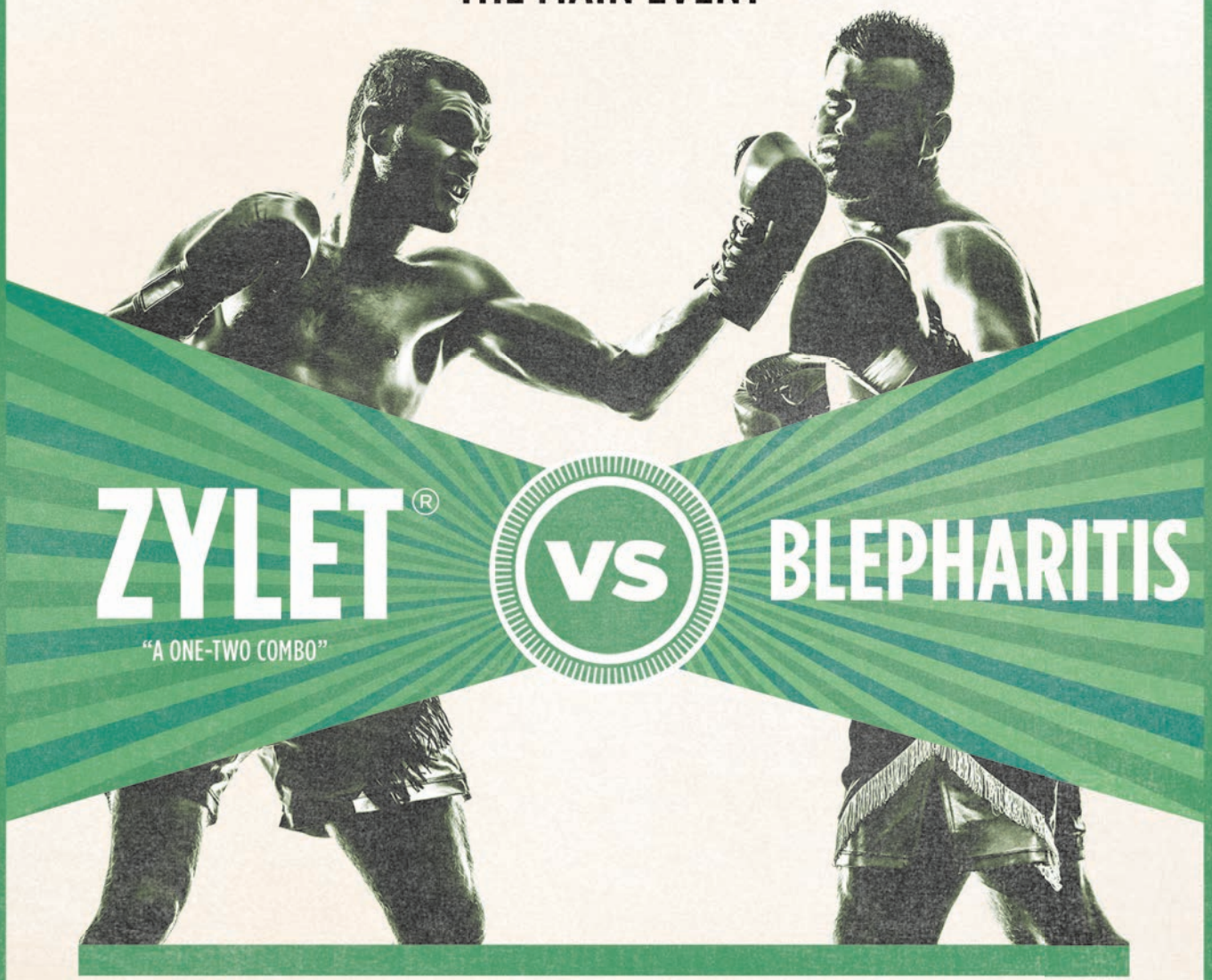

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INDICATIONS AND USAGE

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

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

INDICATIONS AND USAGE (continued)

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

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

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

IMPORTANT SAFETY INFORMATION

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

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

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

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

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

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

Initial U.S. Approval: 2004

DOSAGE AND ADMINISTRATION

2.1 Recommended Dosing

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

2.2 Prescription Guideline

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

CONTRAINDICATIONS

4.1 Nonbacterial Etiology

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

WARNINGS AND PRECAUTIONS

5.1 Intraocular Pressure (IOP) Increase

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

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

5.2 Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

5.3 Delayed Healing

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

5.4 Bacterial Infections

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

5.5 Viral Infections

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

5.6 Fungal Infections

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

5.7 Aminoglycoside Hypersensitivity

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

ADVERSE REACTIONS

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

Zylet:

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

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

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

Loteprednol etabonate ophthalmic suspension 0.2% - 0.5%:

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

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

Tobramycin ophthalmic solution 0.3%:

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

Secondary Infection:

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

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

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

USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

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

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

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

8.3 Nursing Mothers

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

8.4 Pediatric Use

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

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

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

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

8.5 Geriatric Use

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

NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

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

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

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

PATIENT COUNSELING INFORMATION

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

MANUFACTURER INFORMATION

BAUSCH & LOMB INCORPORATED

TAMPA, FLORIDA 33637 USA

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Nothing to Sneeze At

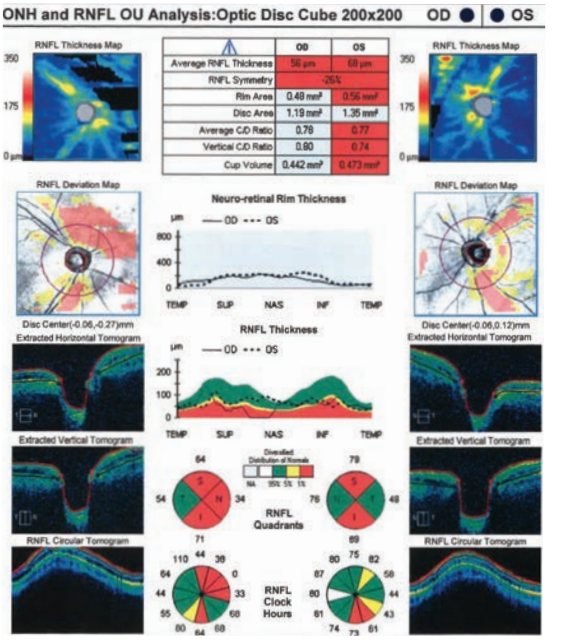
A patient with well-controlled glaucoma experiences a sudden spike in IOP—could his steroidal nose spray be the culprit? **Edited by Paul C. Ajamian, OD**

Q I have a patient who has had well-controlled COAG for years. The pressure went up dramatically and fairly suddenly after a month on a nasal spray. Could this be related? "Your

A patient's sudden and dramatic increase in pressure can most certainly be related to her-or-his use of nasal spray," says Kristen Thelen, OD, of Advanced Eye Center in Gainesville, GA. In 2013, Nasacort (triamcinolone acetonide, Sanofi-Aventis) became the first over-the-counter steroid nasal spray available in the United States.

"We have known for years that steroids of many forms can cause an increase in outflow resistance of the trabecular meshwork, thus leading to an increase in intraocular pressure (IOP)," says Dr. Thelen. One study showed that inhaled nasal glucocorticoids, when used at high doses and regularly for three or more months, put patients at a one-and-a-half times increased risk for ocular hypertension and glaucoma, she says. The study also suggested that certain populations are at higher than average risk for developing a steroid response, and glaucoma patients in particular.[1]

Dr. Thelen describes a recent case: A 59-year-old white male presented with increased intraocular pressure following two months of using Nasacort nasal spray. The patient suffered from fall allergies and started taking over-the-counter triamcinolone acetonide twice a day. Intraocular pressure increased from 19mm Hg and 14mm Hg to 40mm Hg and 39mm Hg in the patient's right and left eyes, respectively. The patient was using Combigan (brimonidine tartrate/timolol maleate ophthalmic solution 0.02%/0.05%, Allergan) OU BID and Xalatan (latanoprost ophthalmic solution 0.005%, Pfizer) OU QHS for his glaucoma, which had been well controlled for roughly 30 years.



Cirrus OCT analysis following IOP spike shows decline in retinal nerve fiber layer thickness.

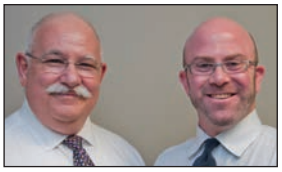
The patient was treated with 500mg of Diamox sequels (acetazolamide ER, Duramed Pharmaceuticals) PO BID and instructed to discontinue use of the triamcinolone acetonide indefinitely. Within two weeks, the patient's intraocular pressure dropped to 19mm Hg and 15mm Hg OD OS. Upon discontinuation of Diamox, the patient's intraocular pressure increased. A second round of Diamox was started and the patient's IOP decreased permanently to normal levels.

Dr. Thelen advises doctors to look into a patient's history for possible risk factors prior to administration of steroidal nasal sprays.

"Some things to focus on are your patient's stage of glaucoma, how many glaucmatous risk factors are present, including age, ethnicity and family history, and the duration and frequency at which your patient was using the nasal spray." All these factors can play a part when trying to determine the likelihood of a steroid nasal spray resulting in an IOP spike, says Dr. Thelen.

Dr. Thelen says that managing acute elevations in intraocular pressure is nothing to stress over. "Find reassurance knowing that IOP spikes are treated no differently than glaucoma—the spike will usually go away when discontinuing the steroid." Dr. Thelen recommends closely monitoring this patient until intraocular pressure returns to normal. ■

1. Garbe E, Lelorier J, Boivin JF, Suissa S. Inhaled and Nasal Glucocorticoids and the Risks of Ocular Hypertension or Open-angle Glaucoma. JAMA. 1997 Mar 5;277(9):722-7.



The Endpoint Endgame

The questions you ask to determine your refraction endpoints can cause differential results. What should you ask—and why? **By Marc B. Taub, OD, MS, and Paul Harris, OD**

Our offering this month is another back-to-basics kind of discussion, indended to give you pause and help you step back for a moment to think about how you come to the endpoints of your refractions. Over the years, we have found that the specific questions asked of patients often result in different endpoints of refraction.

The standard 20/20 line in two different sizes. Although here the size difference is exaggerated, this is the kind of change we look for patients to report when asked, "Which of these looks bigger?"

Definitions

We have found that many assumptions are made regarding the terms used to describe refraction, so it will be helpful to provide some definitional clarity of the relevant endpoint terms before moving on:

• ***Binocular balance.*** The most plus or least minus to the first good 20/20 as measured binocularly. For the purposes of this article, we will assume we are not dealing with unequal visual acuities as a result of amblyopia, strabismus or pathology.

• ***Manifest.*** The most plus or least minus that gives the patient their best binocular visual acuity. Routinely, once the binocular balance has been identified, we reduce plus or increase minus binocularly until some endpoint is reached, then take a visual acuity and record this as the manifest.

Many Questions

A number of years ago, while involved in an Optometric Extension Program (OEP) study group in the Baltimore area, I (Dr. Harris) participated in exercises that led to some interesting insights into how different questions contribute so significntly to a differential in endpoints.

The topic of endpoint questions and their impact on refraction endpoint measurements came up as a result of having attended a two-day seminar by Bruce Wolff, OD, known for developing the Wolff wand. A private practitioner from Cincinnati, he was a noted lecturer and researcher involved with the Skeffington/Alexander National Optometry & Education Learning Research Center in Lancaster, Ohio.

The endpoint discussion began by having each member present some of the questions they asked patients at this critical point in the refraction. A few examples included:

• Which of these looks blackest or clearest?

• Which of these looks the clearest?

• Which of these look the boldest or blackest?

• Which of these do you see better with?

• Which of these looks larger?

• Which of these feels the most comfortable to look at?

Empirics: Finding the Best Question

The sheer number of questions that emerged from just 12 study group participants was shocking. We realized that, at various times, we had each tried several of these approaches, yet had no real rationale for why we chose one over the other. So, we decided to do some "quasi-research." We all agreed to try one of the questions for a month and report back on our experiences.

Over several months we cycled through each of these questions. We then narrowed the list of questions down to those that seemed to yield the cleanest endpoints.

Nearly a year later we emerged with the most utilitarian question. Originally, this question didn't appear to be useful or easily explained:

"Which of these looks larger to you?"

Clinical Pearls

- In myopic patients, the bigger the difference between the two endpoints discussed, the greater the risk for the myopia to continue getting worse and worse over time.
- In hyperopic patients, the bigger the difference between the two endpoints discussed, the more easily you can cut plus at distance. In a future article we will look at how to help hyperopes reduce their dependence on plus.

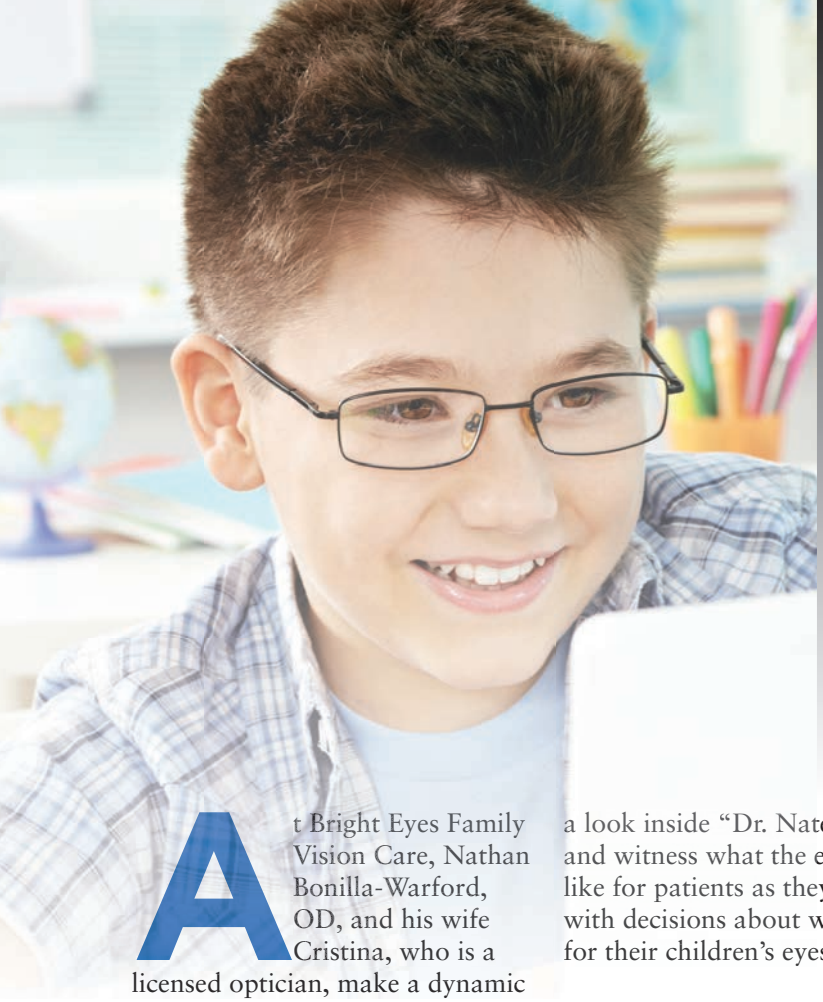
The Rationale

Specifically, we continued decreasing plus or increasing minus binocularly from the binocular balance until the person reported that the next new lens altered their perception of what they were seeing (e.g., making the letters appear to shrink in size). We recorded that as our second endpoint, making certain to record visual acuities with this lens as well.

So you might ask, how could less plus in the case of patients with hyperopia, or more minus for myopic patients, lead to the perception of increased letter size? And that is a wonderful question, which—if answered purely on the basis of optics—would lead to the opposite answer than what is observed in patients. Less plus or more minus should minimize the image, and there is no question about what the optics do; however, this is not what the majority of patients report seeing.

In general, the vast majority of patients indicate that the endpoint of 0.25 or 0.50 less plus or more minus looks the largest. About 10% or so see no size change or, at the first click of the lenses, report that the letters get smaller. About 15% show a 0.75 or more difference between the two endpoints

We hope this gets you thinking about how you investigate different endpoints of your refraction. We would love to hear from you about your experiences with this. Many in the profession believe that one answer—a single refraction—is the patient's prescription. The value of having two distinct endpoints, and how we use them to derive a patient's final prescription, is a topic that we are building to. We have found great use in having two endpoints for our patients: (1) The endpoint with the most plus or least minus to the first good 20/20, and (2) the endpoint through which we get the perceptual response of seeing the letters on the chart the largest. ■



What Children **Really Need** in a Lens Rx

Advice on how to communicate lens benefits to parents from all walks of life.

At Bright Eyes Family Vision Care, Nathan Bonilla-Warford, OD, and his wife Cristina, who is a licensed optician, make a dynamic team. Over the past nine years, the duo has built a successful practice with a strong focus on children's eyewear. Their model has worked so well that it led them to open a second location, called Bright Eyes Kids, which exclusively serves pediatric patients. Now in its second year of operation, Bright Eyes Kids is a huge hit in the community and draws patients from areas far beyond its Tampa, Fl. location.

What's most surprising about Bright Eyes is not its size, it's the approach used by the doctors and staff to continue driving its growth. Bright Eyes doesn't rely on a huge inventory, massive ad campaigns or other pie-in-the-sky methods that are out of reach for most private practitioners. Bright Eyes is a recognized leader in children's vision care because both the doctors and the optical staff rely on honest, patient-centered attitudes when it comes to lens education and dispensing.

In this up-close report, you'll get a look inside "Dr. Nate's" practice and witness what the experience is like for patients as they are faced with decisions about what's best for their children's eyes.

Q. What is your goal in terms of how parents should feel when buying a pair of glasses at Bright Eyes?

Dr. Nate: When parents bring their kids to Bright Eyes, they leave feeling empowered and proud, knowing they've made the best choices they could for their children's vision care needs.

Q. That's a tall order. How do you achieve this?

Dr. Nate: It's a two-pronged approach. My role as the doctor is to increase awareness and to educate. I don't get overly specific. I simply establish that certain features are important when choosing a lens for kids and then I briefly discuss why.

Cristina: When the patient gets to the dispensary, we build on the doctor's message. We focus more heavily on the specifics of certain lens attributes and describe individual lens benefits, explaining why we believe each one of them is so important.

Q. What features, specifically, do you believe every child's Rx should have?

Dr. Nate: Obviously, safety is king. But good optics, anti-reflective (AR), anti-smudge, anti-scratch, and UV protection make a big difference in how well kids can see and perform throughout the year. Parents understand that kids are hard on glasses. This is evidenced by their attraction to warranties. However, warranties aren't enough and they don't ensure good optics.

Cristina: It's also important to note that we don't reserve better lenses for certain patients. We treat every family the same, whether the patient is using insurance or paying out of pocket. Every parent is offered a lens that we believe in, along with an explanation of why we think it's the best. Of course, some may choose to go with a different option. But generally, once you explain the

benefits, most parents can see the value in features like AR and anti-smudge and they feel good about choosing a lens with these important qualities.

Q. How do you explain lens features so that parents recognize their value?

Dr. Nate: We never assume that parents are aware of differences in quality and performance. It's our job to inform them. The key is to find a strategy that allows you to connect in a way that resonates. I stumbled upon an online survey once about the need for AR coating on kids' lenses. A vast majority of parents agreed that it was essential. But what was most compelling was the commentary about why they chose AR. One parent after another said that, if you don't get AR, your kids won't look good in pictures. From a parent's perspective, this is tangible proof that AR and anti-smudge make a difference. If that's how the lens appears from the outside looking in, imagine the experience from the inside looking out, every hour of every day.

Q. We readily accept that adults can have high visual demands, but do children really require the same high quality that adults do?

Dr. Nate: While young children don't drive at night or work in environments where life and limb may depend on crisp vision, 80% of a child's learning occurs through their eyes[1] and almost all classroom learning comes to students via the visual pathways. That's a meaningful visual demand. Lenses like Crizal Kids UV™ have an important place in our practice because they help avoid distracting smudges and other visual obstacles and help children see without the glare that can cause visual strain and discomfort.

Q. Do you think it's important to recommend a lens that helps to filter out blue-violet light?

Dr. Nate: Protecting kids' eyes in our modern-day world requires more than polycarbonate alone can deliver. Our culture is vastly different than it was a few decades ago. Now, 83% of children use an electronic device for three or more hours a day.[2] Some kids are using them much more, especially in schools that issue student computer tablets. This affects my lens choice since there is growing concern that extended near work and blue light could be harmful over time and could have visual repercussions. Lenses like Crizal® Prevencia® Kids that selectively filter out harmful blue-violet light while letting good blue-turquoise light pass through have definitely earned a spot in practices that serve children.

Q. It sounds like the success of Bright Eyes is predicated largely on offering every patient the best in lens technology. But certainly some of your local competitors must offer options at a lower price point. How do you compete with this?

Dr. Nate: I make it clear that selecting a good lens for a child is the normal thing to do, and I don't recommend anything that I don't believe in. If I wouldn't choose it for my own family, I won't choose it for my patients. Crizal® for Kids has performed exceptionally well for us. It's a long-lasting, durable lens that offers outstanding clarity in addition to safety. Yes, parents may find something cheaper somewhere else—but what will it cost them in the long run, and how will it perform?

Dr. Nate's KIDS

K now—Do your research on the latest lens options for kids. Unless you're offering the cheapest lens in town, be prepared to answer parents' questions about why qualities like UV, AR, anti-smudge and blue-violet protection matter.

I nform—It is our responsibility as optometrists to inform families and to offer choices that we would feel good about making for our own kids. That's what we do at Bright Eyes, and our patients seem to love it.

D ifferentiate—Take time to explain how different lenses offer varying degrees of clarity and protection, and discuss how this can impact a child's visual experience.

S erve—We're in the business of preventing problems before they occur. High-quality solutions like Crizal Kids UV and Crizal Prevencia Kids help us do that in a way we feel good about.

1. School-aged Vision: 6 to 18 Years of Age Available at: http://www.aoa.org/patients-and-public/good-vision-throughout-life/childrens-vision/school-aged-vision-6-to-18-years-of-age?sso=y (last accessed November 9, 2015).

2. Lubell J. (2014 July/Aug) Solving For Sight. AOA Focus. p.22-31. Available at: http://aoa.uberflip.com/i/348635-aoa-focus-july-august-2014/27 (last accessed November 9, 2015).

ACA Children's Vision Health Benefit: BOOM OR BUST?

The Affordable Care Act's pediatric essential health benefit has opened the door to comprehensive eye care for millions of children—but will it make a difference in the office? **By Kathleen Foster Elliott, OD**

The pediatric population is underserved in optometric care. For years, federal agencies and educational systems have touted vision screenings as the gold standard in maintaining eye health. Optometrists know screenings are not sufficient to reveal undetected vision problems; in reality, they require a complete workup and, often, comanagement subsequent to their detection. With the integration of the pediatric cohort into eye care via the Patient Protection and Affordable Care Act (PPACA, or ACA for short), millions of children in the United States now have access to comprehensive ophthalmic examinations, that will eventually reveal undetected vision problems.

Optometrists will have the chance to make history because of the profession's unique position to provide greatly needed services. What is the our role within the new care model ushered in by the ACA's historic passage? Let's address six key questions.

Call To Action

Here are three things you can do immediately and expediently to improve patient access:

1. Work with the American Academy of Optometry's (AOA) "Think About Your Eyes" and "InfantSEE" programs to publicize the importance of eye health in your community.

2. Create a public health initiative to educate parents that a school screening or pediatrician eye screening does not take the place of a comprehensive examination.

3. Investigate efforts to strategically position optometrists in accountable care organizations (ACOs) and other health care inter-professional organizations that will be providing access to care.

Has the emphasis on prevention brought more pediatric patients into ODs' offices?

The premise for the inclusion of the pediatric essential health benefit (EHB) was that it would help reduce the high prevalence of amblyopia—3% to 5% in the United States in some studies—that the ACA highlighted to the nation and health care providers across specialties.[1]

When the ACA debuted, enrollment numbers were dismal, and those who did enroll were mainly young, single adults. The enrollment of pediatric patients fell short of expectations; however, two years into the program, enrollment numbers are on par with what were anticipated—almost 10 million people of all ages have either signed up for health insurance in state and federal marketplaces under the ACA or were re-enrolled in coverage for 2015.[2]

It is now more reasonable to expect an uptick in pediatric patients. The implementation of the law was not without its setbacks, so it is only after two full years that the impact of the law on patient coverage can be seen.

A poster presented at this year's Academy meeting showed preliminary data on ACA patient visits in optometric practices (see *Table 1*).[3]

Amblyopia patients, who are also taxpayers, may not be able to function in the workforce to the same degree as their binocular counterparts. They may have an aversion to reading and fall behind in school—80% of learning is derived from vision, according to the AOA—or have a higher failure rate in workforce entrance.[4]

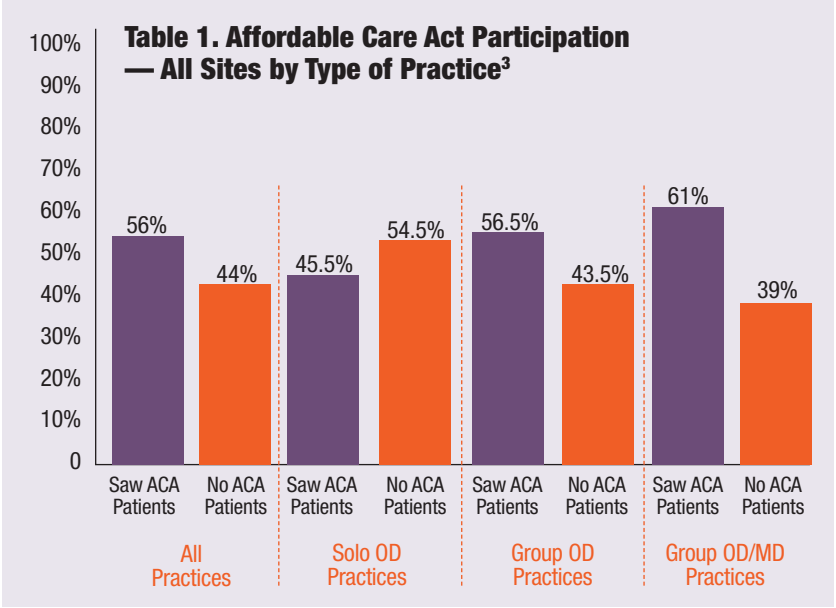
These exact points were made to ACA policymakers by the AOA, and it had a significant impact on their decision to include pediatric comprehensive eye exams as one of the 10 essential health benefits obtained in the plan. In general, amblyopia is very expensive for the government if it is not caught early.

Over time, with more families participating, we will see an uptick in the number of pediatric patients entering optometric offices.

How serious is the impact of access on pediatric eye health?

Alarming statistics have prompted a call for action in the optometric profession.[5] Ten million children in the United States have undetected vision problems, and only 14% of all children have had an eye examination by age six. Only 51% of school-age children ages six to 16 have had a comprehensive eye examination, and more children receive a vision screening than receive a comprehensive eye examination before kindergarten.[5,6,7] Of course, unless amblyopia is caught and treated before age 14, permanent visual impairment can occur.[8] Aggressive occlusive and vision therapy and accurate refractions, ascertained from a cycloplegic refraction, are needed to ensure that amblyopia is prevented. It is generally known that the earlier that you catch amblyopia, the better—preferably before the age of eight. Optometric and pediatric ophthalmology guidelines recommend first eye exams between the ages of six and 12 months, then again at both three and six years of age.[5] The ACA's inclusion of pediatric comprehensive eye exams thus aims to improve the eye health of the pediatric population with early detection; it works well—almost in concert with the recommended pediatric exam timeline/schedule.

The high prevalence of amblyopia was the major reason for the inclusion of pediatric eye examinations into the ACA.



A 2015 survey polled 125 externship sites in 23 states. Fifty-six percent of sites participated in ACA. Solo OD practices participated at a rate of 45.5%, group practices 56.5% and group OD/MD practices at 61%. Six out of 125 sites participated in an ACO. The survey showed that only a slight majority of surveyed ODs see enrolled patients.

What are some practical applications?

To succeed within the ACA health model, be intentional about opening the doors of your practice to pediatric patients. Here are some tips for better clinical treatment and patient workflow:

- Make your office kid-friendly; have a child-designated area in your reception room.
- Consider having a pediatric examination lane devoted specifically to pediatric patients.
- Always cyclolege pediatric patients in order to obtain the most accurate refraction, and become proficient using lens bar retinoscopy.
- Scheduling: Remember that pediatric patients require an initial five to 10 minute evaluation by the

doctor and technician. Then they are cyclopleged, which can take up to 45 minutes to obtain maximum dilation. The second part of the exam is the wet refraction and internal health examination. To save time on established patients with no strabismus or near complaints, cyclopentolate 1% can be prescribed for the parent to administer one hour before the yearly appointment or follow-up. The patient comes in already dilated once your initial measurements are obtained.

What can be done to improve care and access?

Three ways that optometry's access to pediatric patients can improve are as follows:

• ***Educate other health care providers about the value of pediatric optometric care.*** Pediatric access to optometry depends on grassroots efforts with other medical professions, institutions of higher education and professional schools. For instance, optometry can reach out to physician assistant schools, the schools of occupational therapy, physical therapy and nursing, as well as community health clinics, to educate these professions on the value of optometric pediatric care. Strategically positioning designated "education key people" in your state can help get the word out about how optometry plays a vital role in accomplishing the goals of the pediatric vision EHB.

Make your office kid-friendly with a large selection of pediatric frame designs.

• ***Partner with your state or national educational associations.*** This can be a great way to increase access to care. Teachers are on the front lines in detecting eye problems linked to students' learning. For instance, teachers in kindergarten through sixth grade are the ideal group for recognizing vision problems in children, such as convergence insufficiency. Promote the pediatric vision EHB to these organizations so they understand that pediatric comprehensive eye exams are now easily available through the coverage provided by the ACA.

• ***Increase awareness of how important and easy it is to access pediatric comprehensive eye examinations through state Medicaid programs.*** The Medicaid pediatric population in schools sometimes does not take advantage of its Medicaid benefits. Many states provide a yearly comprehensive eye examination for Medicaid enrollees. The challenge is that the enrollees may not realize their benefits cover eye examinations and in some cases their materials, depending on the state's benefit plan. Reach out to your local school systems to educate parents about the vision health benefits offered by Medicaid for their children.

The details of how to access care can also be found on the state health exchange programs. For information about your individual state, go to www.cms.gov and select "State Marketplace Resources," or contact your state association. Additionally, it's essential to work with key people in the legislature and have access to your state Department of Education.

There has never been a better time to hear from us as a profession. Optometrists must continue to be vigilant in our grassroots efforts to ensure the pediatric EHB stays in place and has the opportunity to show its merit.

Online ACA Resources

General ACA overview:
www.Healthcare.gov/glossary/essential-health-benefits

Individual state vision information:
http://obamacarefacts.com/vision-insurance

State health exchanges:
http://obamacarefacts.com/state-health-insurance-exchange

What are strategies for comanaging with pediatricians or pediatric ophthalmologists?

Better communication with pediatricians and pediatric ophthalmologists can easily improve pediatric access to optometrists.

The reality is that most pediatric ocular emergencies come through the pediatrician's office, and then get referred to pediatric ophthalmology. But there are far fewer pediatric ophthalmologists than optometrists in the nation and the demand for care has increased substantially There are fewer than 500 pediatric ophthalmologists nationwide, which has left the specialty unable to meet the needs of a growing pediatric population. As a result, there is typically a backlog of patients trying to obtain care through a referral to pediatric ophthalmology from their primary care physician. In contrast, 40,000 optometrists are currently in practice nationwide.[9] Therein lies the argument for increasing access to care for the pediatric population through optometric practices.

Reaching out to medical schools, pediatric residency programs and pediatric offices is the key to educating physicians on the expertise that optometry offers in the area of pediatrics. Most pediatricians are very uncomfortable even using an ophthalmoscope, and most pediatric residents get less than a month of rotation in pediatric ophthalmology. Pediatricians and ancillary staff are generally very grateful when optometrists reach out to their practices to offer training, correspondence, comanagement and educational tips

Additionally, working directly with the pediatric ophthalmologists in your area can help patients access optometric care. For instance, amblyopia usually requires follow-ups every two to three months in the initial phases of treatment. There is opportunity for shared care between optometry and pediatric ophthalmology in this particular disease process, as well as many other pediatric ocular conditions.

Intentionally reaching out, being available, educating, training and communicating help the pediatric population gain access to optometric care. There has never been a better time for optometrists to provide care, and the demand is only going to increase. Now is the time to be fiercely intentional about strategically placing yourself as a pediatric-friendly optometric practice, and in doing so, being better positioned for a greater influx of ACA patients.

What common pediatric conditions need to be caught by schools and pediatricians?

• ***Amblyopia.*** Affecting upwards of 3% to 5% of the population, it is the single most important reason why comprehensive pediatric eye exams were included as an EHB.

• Refractive error, especially latent ***hyperopia.***

The ACA provides the unique opportunity to integrate pediatrics into your practice.

• ***Retinoblastoma,*** typically presenting as ***leukocoria*** or ***strabismus.***

• Retained ***foreign bodies.***

• Differential diagnosis between ***optic nerve drusen*** and ***papilledema.***

• ***Convergence insufficiency, strabismus*** and other ***oculomotor disorders.***

• ***Preseptal cellulitis*** and ***lid abnormalities*** such as ***chalazion*** and ***nasolacrimal duct*** obstructions.

Optometry is equipped to diagnose and manage or comanage these conditions at a level of excellence that many children are not currently receiving, so it is critical that we engage pediatric patients, ultimately for the child's benefit.

Final Thoughts

Now is the time to position yourself as a pediatric-friendly practice by incorporating an environment for children in your office. Become intentional about marketing your pediatric skills to schools, hospitals, pediatricians, pediatric ophthalmologists and ancillary health professionals. Finally, take advantage of the many resources available to help improve your skills and your knowledge of the pediatric population. By engaging in pediatric eye care, you will not only help children's eyes, but their lives as well. ■

Dr. Elliott is the director of a high volume pediatric ophthalmology practice in Tulsa, Okla. She was 2012 legislative doctor of the year and 2014 optometrist of the year for Oklahoma. She is cofounder of www.take10vision.com and a continuing education speaker.

1. Rainey, AM. What's New and Important in Pediatric Ophthalmology and Strabismus for 2011. Paper presented at the 37th Annual American Association for Pediatric Ophthalmology Meeting, April 1, 2011; San Diego, CA.
2. Armour S. Affordable Care Act Enrollment Near 10 Million. www.wsj.com/articles/affordable-care-act-enrollment-near-10-million-1423070147. Updated 2/4/2014. Accessed October 2015.
3. Ruskiewicz J, Gurwood A. Vision care providers and the and the affordable care act; a snapshot of participation analyzed by one college of optometry's externship providers. Paper presented at American Academy of Optometry Meeting 2015, October 8; New Orleans, LA. 4. School-aged Vision: 6 to 18 Years of Age. www.aoa.org/patients-and-public/good-vision-throughout-life/childrens-vision/school-aged-vision-6-to-18-years-of-age.
5. Rein D, Wittenborn J, Zhang X, Song M, et al., Vision Cost-effectiveness Study Group. The potential cost-effectiveness of amblyopia screening programs. J Pediatr Opthalmol Strabismus. 2012; 49(3);146-155.
6. Holmes JM, Lazar EL, Melia BM, et al. Effect of age on response to amblyopia treatment in children. Arch Ophthalmol. Nov 2011;129(11):1451-7.
7. InfantSee website. www.infantsee.org. Accessed October 2015.
8. Amblyopia. www.aapos.org/terms/conditions/21. Updated 05/2015. Accessed 10/2015.
9. American Optometric Association official website. www.aoa.org. Updated November 2015. Accessed October 2015.

Pediatric Eye Care Resources for Optometrists

1. AOA Think About Your Eyes: http://thinkaboutyoureyes.com/about.

2. InfantSee: www.infantsee.org.

3. Your local college of optometry, and the College of Optometrists in Vision Development (COVD): www.covd.org.

4. The Pediatric Eye Disease Investigator Group (PEDIG): www.pedig.net.

5. Pediatric optometrist/ophthalmologists in your local area for knowledge and referrals.

6. Local, state and national associations.

SYSTEMIC MANAGEMENT of Pediatric Ocular Disease:

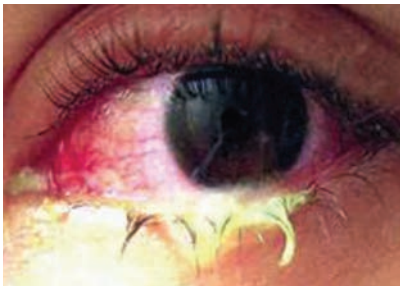
Small Patients, Big Decisions

A little pharmacology expertise can go a long way in treating kids using oral medications. **By Bruce Onofrey, OD**

When managing ocular disease with systemic medications, clinicians must select drugs and dosages that are both safe and effective—and the process becomes much more complicated when the patient is a child. We must give special consideration to this vulnerable patient group, as certain medications considered safe in the adult population may be contraindicated in children. Additionally, the pathogens most common in adult infectious disease may be different in this population, requiring the use of an antibiotic different from what is used in the adult population for similar-appearing infectious ocular disorders.[1]

Dosing Dilemmas

Choosing the correct dose of a systemic medication for the pediatric population is not a simple linear calculation based on the size of an individual; instead, one must consider differences in pharmacokinetics—the ability to absorb, distribute, metabolize and excrete a medication—to individualize the treatment to the patient. For example, a 13-year-old may be tall for his age, yet still have the liver maturity of a young teenager. Similar to other special populations such as the elderly, pregnant patients and those with diabetes, the clinician must first choose a drug that is safe for children and then carefully calculate a modified dosage. This modification is dependent on several factors, including age, weight, body surface area, liver and kidney function and pre-existing systemic disease.[1,2]



***H. Influenzae*, a gram-negative bacteria, is a common cause of conjunctivitis.**

There are three popular dosing calculations. Clark's rule was an attempt to adjust dosage strictly on weight and was used for individuals weighing less than 100lbs (40kg). Because it neither takes into account factors such as liver and kidney function nor is specific enough for many of the potent drugs used today, its use should be avoided. Its greatest flaw is seen in overweight children for whom the calculated dosage would be well above the appropriate amount.

Fried's rule, used in infants, is based on age only and does not take into account the variability in

children's sizes or the pharmacokinetics of the prescribed medication.

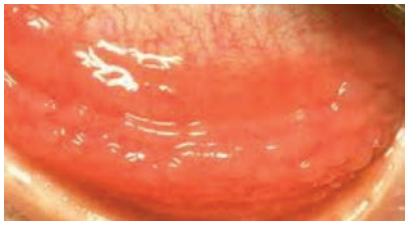
The third formula uses body surface area (BSA) in square meters to calculate IV dosages in infants. The calculation is a ratio of the child's BSA divided by an ideal adult's BSA. That ratio is multiplied by the average adult dose to produce a reduced dose for the child. It is a complicated calculation that is prone to error.[3]

The most precise calculation is based upon the recommendations developed by the drug companies themselves. These recommendations are listed in the package insert and are available via many drug databases. These calculations are always in metric measurements and usually in mg/kg of body weight. Most importantly, these dosages also list a maximum pediatric dose. Note that the recommended dose may be listed for each administration of drug, given a certain number of times per day, or as a 24-hour dose that you must divide by the recommended frequency of daily dosing. Pay strict attention to the exact language to avoid dosing errors.

Indications

Except for degenerative conditions, such as AMD, children present with many of the same conditions as adults. Ocular disorders include acute infectious disease, allergic (atopic) disease, acute injuries and inflammation. By far, the most common conditions in children involve treatment with antibiotics. Most acute ocular disease is treated with topical agents. The following conditions commonly need systemic therapy with or without topical agents.

• ***Conjunctivitis.*** Although not normally treated with systemic medications, the most common cause of acute infectious conjunctivitis in children is the gram-negative



Chlamydia is a common cause of ophthalmia neonatorum.

pathogen *Haemophilus influenzae*. This is different from adults, who most commonly present with adenoviral conjunctivitis. *Haemophilus* colonizes children in their upper respiratory tract, from which it travels via the eustachian tube to the middle ear (otitis media). It can travel via the nasolacrimal system to the eye to cause conjunctivitis, to the lung to produce pneumonia and to the CNS to cause meningitis.

The introduction of a *Haemophilus* vaccine has reduced the incidence from more than 40% of children's eye disease to approximately 32%. It still remains the major cause of acute infectious eye disease. Because there is a reservoir of bacteria, children colonized by *Haemophilus* who show evidence of systemic infection (e.g., otitis media, fever) should be treated both topically and systemically.[9]

• ***Ophthalmia neonatorum.*** Any ocular infection within the first 30 days of life is known as ophthalmia neonatorum. The patient must be tested to rule out *Neisseria* infections. Currently, the most common cause of ophthalmia neonatorum is chlamydia, which must be managed with systemic therapy. If chlamydia is present secondary to maternal transfer, the mother and her partner should be treated as well.

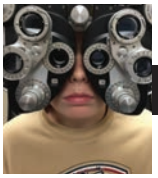
Closely related to chlamydia is trachoma, which causes inflammation and scarring of the conjunctiva and cornea. Trachoma represents a major cause of vision loss worldwide, though far less so in the United States and other developed countries. The infection is spread via flies, with children as the primary reservoir.

Another condition, preseptal cellulitis, is commonly caused by injury or insect bites, and must be differentiated from orbital cellulitis.[9] Orbital cellulitis, a deeper, more serious condition, is almost always caused by an adjacent severe sinusitis. The hallmarks of orbital cellulitis are decreased ocular motility and globe displacement, often proptosis. This can be life-threatening because of the orbit's proximity to the brain, and should be considered a true emergency requiring immediate IV antibiotics and hospitalization.[9]

Common pathogens that can produce both preseptal and orbital cellulitis include *Staphylococcus* species, both *aureus* and *epidermidis*, as well as *Streptococcal* species. *Strep*. is particularly dangerous in that it produces the enzymes streptokinase and hyaluronidase, allowing it to spread through tissues in a disease process known as erysipelas.[9]

• ***Bacterial dacryocystitis/dacryoadenitis.*** Characterized by acute or chronic infection or inflammation of the lachrimal sac, dacryocystitis is not uncommon in newborns with lacrimal stenosis prior to repair or spontaneous resolution of the blockage. Complete or partial obstruction of the duct can lead to tear stasis and opportunistic infection. The acute version is most commonly seen in children. The route of infection can be blood-borne, transconjunctival, transneuronal or via direct trauma. Common bacteria that cause dacryoadenitis include *Staph*. sp., *Strep*. sp., *Haemophilus influenzae* and *Neisseria*.

Treatment with broad-spectrum antibiotics should be initiated until



Drug Testing in Children

Before 1970, children and pregnant women were never involved in drug studies, leading to a lack of knowledge of how children process important medications. By the mid '70s, the American Academy of Pediatrics (AAP) argued for the right to include children in well-controlled studies, stating that physicians were forced, without proper data, to conduct uncontrolled experiments on kids when they prescribed medications that had never been tested in this population. In 1977, guidelines established by the AAP stated that children could participate in minimal risk studies if one parent gave permission or higher risk studies with the permission of both parents.[4-8]

cultures, titers and lab studies identify the specific etiology. Appropriate treatment choices include:

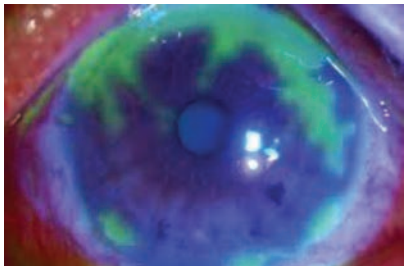
- Amoxicillin/clavulanate: 25mg to 40mg/kg/D in a BID divided dose
- Cefprozil: 15mg/kg/D Q12H, with a maximum dose of 1gm/D
- Azithromycin: 10mg/kg/D x 3D, maximum daily dose of 400mg
- Clarithromycin: 7.5mg/kg/D BID[9]

• ***Ocular rosacea.*** More commonly affecting the central face (i.e., nose and cheeks) than the eyes and adnexa, rosacea is an inflammatory dermatologic condition that has a variety of trigger mechanisms, including spicy foods, cold weather, sun exposure and stress. Rosacea is classified into four subtypes by the National Rosacea Society: erythematotelangectatic, papulopustular, phymatous and ocular. It is common for patients to express more than one type, and more than 50% of patients with rosacea also present with ocular rosacea.[9]

Ocular rosacea causes chronic inflammatory changes of the eyelid margins, leading to an uneven "wiper effect" of the eyelid against the corneal surface, as well as poor meibomian function with tear film instability and evaporative dry eye. The inflammatory mediators released from the lid also commonly lead to conjunctivitis and keratitis.[9]

The cornerstone of medical therapy for ocular rosacea has been the tetracyclines and the macrolide antibiotics, which have been shown clinically effective in reducing signs and symptoms of both facial and ocular rosacea. However, tetracyclines are absolutely contraindicated in younger children. Their use produces permanent tooth discoloration and bone abnormalities. In children older than age 12, the dose of doxycycline is 50mg to 100mg BID. Fortunately, children younger than 12 can use the macrolides. The newest broad-spectrum macrolide agents include clarithromycin (Biaxin, Abbott Laboratories) and azithromycin (Zithromax, Pfizer). Clarithromycin is usually dosed in individuals that weigh less than 40kg at 7.5mg/kg BID. Because azithromycin has a 68-hour half-life, it is dosed at 10mg/kg/D for only three days per week. Treatment commonly lasts for months.[9]

• ***Herpes simplex.*** The majority of the population will be exposed to herpes simplex virus (HSV) by a young age. Transmission of the virus occurs by direct contact with infected lesions, salivary droplets, fomites and healthy, asymptomatic carriers. The incubation period between contact and expression is approximately three to nine days.



Ocular manifestations of HSV-1 include: blepharitis, conjunctivitis, corneal epithelial disease and stromal disease.

While the primary infection may be mild or subclinical in nature, the virus may reactivate and ultimately involve various ocular structures. Potential manifestations include blepharitis, conjunctivitis, corneal epithelial disease, stromal disease, neurotrophic ulceration, endotheliitis, trabeculitis, uveitis, retinitis, or a combination of these. Active viral replication, pathogenicity of the virus and host immune responses are major factors influencing disease recurrence.[9]

There are two types of HSV that affect humans. HSV-1 commonly causes labialis ocular manifestations, while HSV-2 is usually genital in nature and is acquired by sexual contact or, in the case of children, is acquired during vaginal delivery. The specific treatment is dependent on the tissues affected. Ocular epithelial disease is not treated with steroids, whereas inflammatory forms include topical and systemic antiviral therapy and topical steroids.[9]

When prescribing oral antivirals, the dosage depends on the agent. The usual adult dose of acyclovir is 400mg five times daily. Pediatric dosing using the 200mg/5cc suspension is 10mg/kg Q8H.

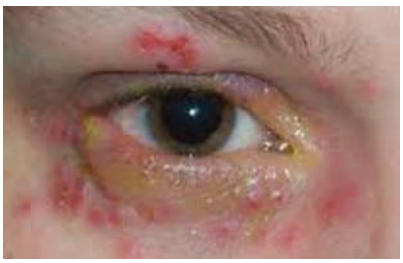
• ***Varicella zoster virus (VZV).*** Varicella is the primary infection from VZV, resulting in inflammatory as well as immunologic reactions. VZV is responsible for the systemic varicella infection (chickenpox) upon initial exposure to the virus and for herpes zoster (shingles) upon reactivation. The primary VZV infection results in an exanthematous reaction of the

skin resulting in the formation of pustules ("pocks") that contain live virus. The lesions are typically self-contained and tend to resolve over a period of one to two weeks. The incidence of childhood VZV has decreased due to the vaccine.

Typically, ocular involvement results in mild to moderate ocular hyperemia with a watery discharge. Symptoms include foreign body sensation, occasionally blurred vision and photophobia. Signs can include a dendriform keratitis and are distinctly different from those seen in herpes simplex. Treatment is supportive, although more severe cases may require oral acyclovir. Pediatric dosing using the 200mg/5cc suspension is 10mg/kg Q8H.[9]

• ***Allergy/immune disease.*** Typical seasonal allergic eye disease is managed with topical agents. However, children experiencing significant ocular anaphylactic symptoms (i.e., moderate to severe immediate hypersensitivity reactions triggered by exposure to antigens such as animal dander, peanuts or medications that commonly include penicillins, sulfonamides and opiates) may require systemic intervention. Children with asthma are particularly vulnerable to significant allergic events. The speed of onset of antihistamine agents is directly related to their lipid solubility. Unfortunately, highly lipid-soluble agents also cross the blood-brain barrier and can lead to significant anticholinergic side effects. These include sedation as well as dry eye and mouth symptoms. The most important rapid-onset oral antihistamine is diphenhydramine. The less sedating agents, unfortunately, have a much slower onset of action and are reserved for prophylactic use.

Diphenhydramine, the drug of



Chickenpox often involves ocular hyperemia with a watery discharge.

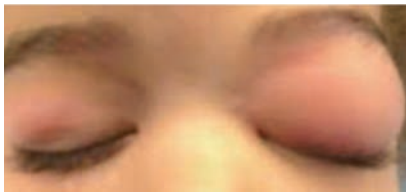
choice for acute mast cell mediated allergic reactions, is available in over-the-counter adult and pediatric forms. The pediatric syrup is available with 12.5mg/5cc. The pediatric dosage is 1mg/kg/D up to TID, with a maximum of 50mg/D. Overdosage can lead to convulsions and seizures.

Pharmacology

Knowing more about the agents you are prescribing to your pediatric patients can help ensure the most appropriate dosage. Here are many of the common systemic medications used in the pediatric population.

• ***Penicillins.*** Amoxicillin is a semisynthetic antibiotic with a broad spectrum of bactericidal activity against many gram-positive and gram-negative microorganisms. It is, however, susceptible to degradation by β-lactamases, and the spectrum of activity does not include organisms that produce these enzymes. Clavulanic acid is a β-lactam structurally related to the penicillins that can inactivate a wide range of β-lactamase enzymes commonly found in microorganisms resistant to penicillins and cephalosporins. In particular, it has good activity against the clinically important plasmid-mediated β-lactamases frequently responsible for transferred drug resistance.[10]

The amoxicillin and clavulanic acid formulation in Augmentin



Ocular anaphylaxis is often treated with a rapid-onset oral antihistamine.

(GlaxoSmithKline) protects amoxicillin from degradation by β-lactamase enzymes and effectively extends the antibiotic spectrum of amoxicillin to include many bacteria normally resistant to amoxicillin and other β-lactam antibiotics. Thus, Augmentin possesses the properties of a broad-spectrum antibiotic and a β-lactamase inhibitor. Amoxicillin/clavulanic acid has been shown to be active against most strains of bacteria that produce pediatric ocular disease, including *Staph*. and *Strep*. species and, most importantly, *Haemophilus influenzae*.[11]

• ***Macrolides.*** Azithromycin represents the new broad-spectrum keto-macrolide antibiotics. They act by binding to the 50S ribosomal subunit of susceptible microorganisms and interfering with microbial protein synthesis. Nucleic acid synthesis is not affected. It is bactericidal and effective against *Staph*. sp., *Strep*. sp. and, most importantly in children, *Haemophilus*. This structural change increases their spectrum of activity over erythromycin. The keto-macrolide agents also include clarithromycin.

Azithromycin differs from clarithromycin in that it has a much longer half-life; therefore, the dosing schedule is markedly different from antibiotics that are used for seven to 10 days. Most pediatric dosing regimens of azithromycin last for only three days, with the drug remaining active in the body for over 10 days. This long half-life improves compliance and negates the need for prescription refills. The most important parameter for safe use is proper pediatric dosing following the guidelines based on age and weight. Overdosage affects the liver, and children are particularly susceptible because of their relative reduced renal function, so patients with liver disease should be prescribed a markedly reduced dosage. The macrolides are particularly valuable as a replacement for individuals allergic to penicillins.

• ***Cephalasporins.*** Cefprozil is an oral second-generation cephalosporin. The second-generation drugs are particularly valuable in the pediatric population for two reasons: (1) They are less susceptible to destruction from penicillinase-producing bacteria, and (2) they show greater activity against *Haemophilus influenzae*. The recommended oral dose of cefprozil is 15mg/kg/dose BID. It is available in liquid dosage forms of 125mg/5ml and 250mg/5ml. The maximum dose is 1gm/day.

• ***Tetracyclines.*** Recently, the use of doxycycline in the management of ocular disease has focused on its ability to reduce ocular inflammation. The indications for its use include ocular rosacea, dry eye, recurrent erosion and bacterial keratitis. A recent study by ASCRS recommends it be used in place of topical steroids for reduction of inflammation secondary to certain forms of bacterial keratitis.[12] The AAP recommends its use for ages eight and older. The major adverse effect of this class of drug in younger children is permanent tooth staining and abnormal bone development. The recommended oral dose for children ages eight and older is 2.1mg/kg/D BID. It should never exceed the adult dose, which is 100mg BID.

• ***Antiviral therapy.*** Acyclovir is a synthetic purine nucleoside analog with *in vitro* and *in vivo* inhibitory activity against HSV-1, HSV-2 and VZV. The inhibitory activity of acyclovir is highly selective due to its affinity for the enzyme thymidine kinase (TK) encoded by HSV and VZV. This viral enzyme converts acyclovir into acyclovir monophosphate, a nucleotide analog. The monophosphate is further converted into diphosphate by cellular guanylate kinase and into triphosphate by a number of cellular enzymes.

In vitro, acyclovir triphosphate stops replication of herpes viral DNA by: (1) competitive inhibition of viral DNA polymerase, (2) incorporation into and termination of the growing viral DNA chain, and (3) inactivation of the viral DNA polymerase. The greater antiviral activity of acyclovir against HSV compared to VZV is due to its more efficient phosphorylation by the viral TK.

The major potential adverse effect is crystalline nephropathy. It is important to calculate a proper dosage and further ensure patients have normal renal function and remain well hydrated during therapy. The drug is extremely safe when the prescriber and parents are conscious of these factors. Of the three major oral antiviral medications—acyclovir, valacyclovir and famcyclovir—only acyclovir is available in a pediatric syrup of 200mg/5cc.

• ***Prednisone/prednisolone.*** The steroid prednisone is popular by virtue of its ability to rapidly control both acute and chronic inflammatory disease. In children it is commonly used to manage acute, type 1 allergic events and asthma. It is used chronically for the management of rheumatoid disorders. Ocular inflammatory

Recommended Pediatric Dosages[11]

- **Diphenhydramine**:
  2-5yrs: 6.25mg every 4-6 hours, max 24 hour dose of 37.5mg/24 hours
  6-11yrs: 12.5 to 25mg every 4-6 hours, max 24 hour dose of 150mg/24 hours
  >11yrs: 25-50mg every 4-6 hours, maximum of 300mg/24 hours
- **Prednisone/prednisolone**: 1mg/kg/day divided up to QID/24 hours
- **Augmentin**: 25mg/kg/day in divided dose of two to three times/day
- **Cefprozil:** 15mg/kg/dose BID
- **Azithromycin**: 10mg/kg daily for three days
- **Clarithromycin**: 7.5mg/kg/dose BID
- **Doxycycline**: 2.1mg/kg/dose BID only in children 8 years or older
- **Acyclovir**: 10 to 20mg/kg/dose every eight hours
- **Acetaminophen**: 15-20mg/kg/dose PRN every 4 hours. Maximum of 5 doses; not to exceed75mg/kg/day or 2.6g total/day
- **Ibuprofen**: 10mg/kg every 6 hours, max dose of 800mg not recommended for children under 6 months
- **Acetaminophen with codeine**: 0.5-1mg/kg of codeine component every 4 hours PRN; caution in children under 3 years of age
- **Acetazolamide**: 5mg/kg/dose every 6-12 hours/24 hours

conditions, including uveitis and allergic eye and periocular skin reactions (hives), also respond well to short-term steroid use. The most important contraindication to even short-term use in any population is diabetes, as glucocorticoids can produce a dramatic rise in blood glucose levels. The dosage for all age groups is 1mg/kg/day in divided dosage three to four times daily with a maximum dosage of 60mg/D. It is available in tablets that range from one to 100mg/dose and a pediatric syrup that contains 5mg/5ml (Pediapred).

Pain Management

It is always important to consider the management of pain associated with ocular injury or inflammation. Adjunctive pain management is most important in those children who may not be able to clearly express their level of discomfort. Dacryocystitis, preseptal cellulitis, corneal abrasion and ocular burns are only a few indications that require short-term pain management. Safety is a major consideration in children managed with analgesics. Liver and kidney damage as well as neurologic adverse effects can occur with inappropriate use of these agents. These side effects must be considered when prescribing these agents. A few pain management options include:

• ***Acetaminophen.*** This OTC drug has long been considered the analgesic of choice for management of pain in special populations such as children and pregnant patients.

For the last several years, the FDA has mounted a significant public campaign to educate both the public and the pharmaceutical industry on the dangers of acetaminophen overdose.

Acetaminophen acute toxicity is a major cause of acute hepatic failure in children. The reasons for overdose include children assuming fruit-flavored syrups and chewable tablets are "candy," and the lack of understanding that using multiple OTC products containing acetaminophen contributes to a cumulative daily dose that exceeds the current recommended maximum adult dose of 2.6g/D, leading to a toxic cumulative dose.

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Advise parents to note the presence of acetaminophen in all products given to their children. The current recommended dose is 15mg/kg every four to six hours PRN for pain with a maximum of five doses/24 hours, not to exceed 75mg/kg/day or 2.6g total/day.

• ***Ibuprofen.*** An oral NSAID, ibuprofen is available in a pediatric dosage of 100mg/5ml. The recommended dose is 10mg/kg/dose every six hours. Do not exceed an average adult dose of 600mg QID. Also, ibuprofen is not recommended in children under the age of six months.

The major contraindication to the use of any NSAID such as ibuprofen is bleeding, as these drugs inhibit platelet activity and increase the risk of hemorrhage in those with injuries, abnormal clotting or in the presence of other drugs that have anti-coagulant properties.

• ***Opiates.*** The most common form of opiate analgesic drugs is a combination agent. Of the common combination agents, acetaminophen with codeine is, by far, the most popular. The use of acetaminophen with codeine is synergistic for pain management; that is, both drugs enhance the other's ability to reduce pain without enhancing toxicity. Each drug in a combination dosage form must be judged separately for its safety in each patient. In addition to acetaminophen's effect on the liver, there is a significant risk of allergic reaction to codeine. All opiates demonstrate cross-sensitivity, so a patient allergic to one opiate is allergic to all of them. Opiates also can produce significant respiratory depression, which is particularly dangerous in children with a history of asthma. The drug can produce anticholinergic side effects, including marked sedation.

The recommended dose of acetaminophen with codeine is based on the codeine content of the medication and should not exceed 0.5mg to 1mg/kg/dose every four hours PRN pain. The liquid dosage form contains 12mg of codeine and 120mg of acetaminophen per 5ml. The dosage is 0.2ml to 0.4ml/kg/dose every four hours PRN pain. The product should be used with extreme caution in children under the age of three.

When treating ocular disease in the pediatric population, always weigh the need for systemic therapy against the associated risks. Specifically, one must determine, based on the clinical history, the specific drug, dosage and frequency of use that will be safe and effective for that particular patient. ■

Dr. Onofrey is a clinical professor and the executive director of continuing education programs at the University of Houston.

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Discern Optic Nerve Head Drusen from True Papilledema

Evaluating a pediatric patient's optic nerve presents particular challenges. Consider these noninvasive techniques and know what you're looking for.

By Pavanjeet Ubhi, OD, Diana Shechtman, OD, and Katherine Green, OD

When a doctor discovers bilaterally elevated optic nerve heads (ONH), she or he faces a particular diagnostic challenge, especially when that patient is a child. Distinguishing congenital etiologies of optic disc elevation (known as pseudopapilledema) from true papilledema is imperative. True papilledema is a medical emergency. However, its incidence among the pediatric population is relatively low—even among patients who doctors initially suspect.[1] According to one case series, three quarters of the pediatric patients referred for papilledema actually had a form of pseudopapilledema, or a variation of normal optic disc.[1] Pseudopapilledema is a benign elevation of the ONH, associated with hypoplastic nerves, tilted nerves, crowded disc or optic nerve head drusen (ONHD).[2]

ONHD are the most common cause of pseudopapilledema. The condition affects 0.4% of the pediatric population, with 75% of cases manifesting bilaterally.[3-5] ONHD are refractile bodies composed primarily of calcium, deposited anterior to the lamina cribrosa. Since drusen may displace the neuroretinal tissue anteriorly, it may cause a false appearance of optic nerve swelling.[6,7] Researchers speculate that smaller-than-average scleral canals may be susceptible to static axoplasmic flow, which causes dysfunction of metabolic capabilities of the axon and results in drusen deposits at the disc.[2]

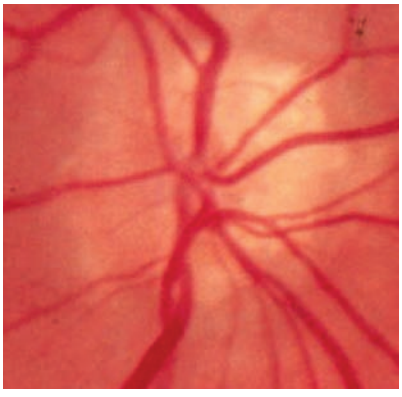

Fig. 1. A chalky, scalloped disc margin characteristic of ONHD is seen on the nasal portion of this optic disc. Note the early vessel branching and trifurcation as the blood vessels exit the nerve.

Papilledema, on the other hand, is a true medical emergency indicated by bilateral swelling of the optic nerve due to increased intracranial pressure.[2] This pressure is transmitted to the subarachnoid space surrounding the optic nerve (ON), causing increased pressure around the ON and resulting in blockage of axoplasmic transportation and edema of the nerve fiber layer (NFL).[8] Causes of papilledema in the pediatric population may include, but are not limited to, Guillain-Barré syndrome, spina bifida, hydrocephalus, intracranial mass, trauma/subdural hemorrhage,

meningitis, subdural venous thrombosis, arteriovenous malformation and idiopathic increase in intracranial pressure.[9-11]

This article reviews how to properly distinguish between pseudopapilledema, such as ONHD, and true papilledema, as well as how to use various diagnostic modalities to make the distinction.

Funduscopic Evaluation

Although no set protocols exist for the evaluation of pseudopapilledema vs. true papilledema, a comprehensive eye exam with a dilated fundus evaluation and the use of fundus photography can pose great value.[12] In some cases, characteristic funduscopic findings may help distinguish ONHD from true papilledema. The presence of a spontaneous venous pulse (SVP) is a significant finding which rules out true papilledema. However, an absence of SVP occurs in 20% of the normal population and, in some cases, the presentation of the SVP may be rather subtle.[13] Furthermore, evaluation for the presence of SVP may be taxing, due to poor cooperation and fixation by pediatric patients.

Visible ONHD show observable calcifications within the disc in addition to a chalky, scalloped disc margin (*Figure 1*). However, visible calcifications are not always evident, since most ONHD in the pediatric population are buried. Although both true papilledema and ONHD will present with elevated disc margins, in cases of ONHD the elevation is confined to the disc. The disc margin may be indistinct and in some cases may possess a scalloped appearance. Elevated disc margins associated with ONHD manifest no vessel obscuration at the disc margin. Anomalous vascular branching patterns

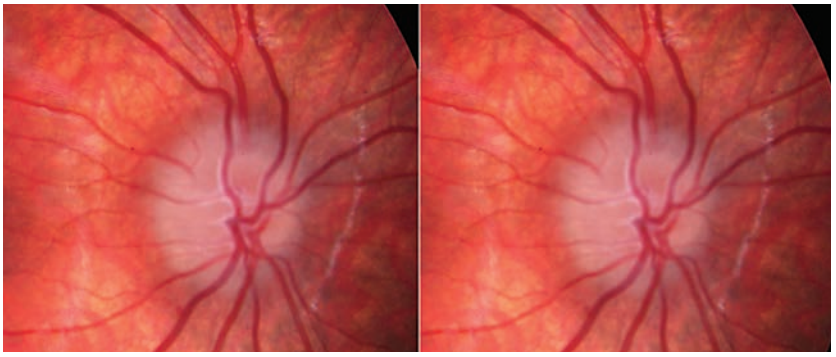

Fig. 2. Stereoscopic fundus photo of ONHD. Abnormal trifurcation is noted, as well as dark appearance deep in the peripapillary tissue.

For a table on the features of ONHD vs true papilledema, visit www.reviewofoptometry.com, or scan the QR code.

depicted as an increased number of retinal vessels, early branching or trifurcation or both (*Figure 1*) are key features of ONHD. Furthermore, a dark gray appearance deep in the peripapillary tissue may be visible in cases of ONHD (*Figure 2*).

In cases of true papilledema, vessel obscuration occurs due to swelling of the NFL, hence elevation expands into the peripapillary retina. The optic disc may be described as hyperemic associated with superficial microvascular dilations and telangiectasia (*Figure 3*). Paton's lines, described as retinal folds or buckling, may also be present.[8] More advanced stages of true papilledema manifest with peripapillary cotton wool spots and retinal hemorrhages.[8] It is important to note that, although not common, vascular changes such as peripapillary retinal hemorrhages or vein occlusion can also occur in ONHD.[14] In addition, patients with ONHD may be at risk for developing choroidal neovascular membranes.[15]

Symptoms such as diplopia, transient visual obscuration, headaches and tinnitus are observed with papilledema. Thirty-three percent of patients with true papilledema are asymptomatic.[2]

Although fundus photography, particularly stereo photos (*Figure 2*), can aid in the evaluation of the optic disc in pediatric patients who have difficulty remaining at the slit lamp, differentiating true papilledema from ONHD may be a challenge using only a static photo.[2]

Non-invasive Imaging Technologies

Doctors have several non invasive imaging options for these situations.

• ***B-scan Ultrasonography.*** B-scan is considered to have high sensitivity and specificity for the detection of ONHD.[2,11] In the general patient, the optic nerve appears as a tubular structure with low homogenous reflectivity. Drusen appear as hyperreflective calcified bodies in the optic nerve and will continue to show increasing brightness even at a low gain. B-scan of true papilledema shows elevation of the optic disc with absence of hyperreflective calcified bodies.[2] A crescent sign representing a ring of fluid around the optic nerve may also be noted in true papilledema.[16] It can be difficult to identify deeply buried drusen on B-scan, as the hyperreflectivity is less evident than

surfaced drusen. Furthermore, the absence of calcifications does not confirm true papilledema, since other causes of pseudopapilledema, such as a crowded disc, may still be present.

• ***Spectral-domain optical coherence tomography (SD-OCT).*** SD-OCT of ONHD will reveal an elevated disc with a characteristic "lumpy-bumpy" appearance. On the other hand, a nerve with true papilledema will reveal a smooth internal contour of the ONH with a characteristic hyporeflective "V" pattern in the subretinal space adjacent to the ONH.[17,18] The "V" contour is presumed to be due to hydrostatic pressure from the ONH leaking into the subretinal space, resulting in an enlarged separation between the neurosensory retina and retinal pigmented epithelium (RPE).[17,18] Investigators report that a small subretinal space also exists when ONHD are present; however, the subretinal space is smaller than that seen in true papilledema.[19]

Blood within blood vessels block the SD-OCT signal, casting a shadow onto the underlying tissues, which can easily be confused for the contoured appearance of ONHD. This is one reason why the SD-OCT image should be compared with ONH photos. Blood vessels are smaller and more superficial than drusen and have presence of reflective tissue completely encircling the blood vessel.[7] Enhanced-depth imaging OCT can detect buried ONHD, as it can image deeper ocular structures than the SD-OCT.[20]

An increase in retinal nerve fiber layer (RNFL) thickness values can further aid in the evaluation of true papilledema. RNFL may appear to be unaffected or thinner in ONHD than in the age-related normative database, but are thicker in

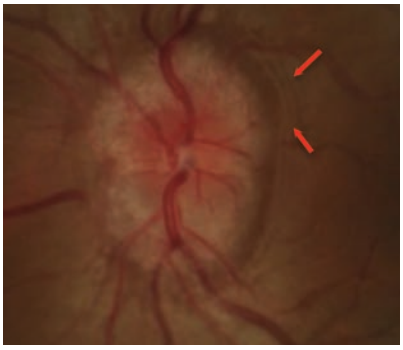

Fig. 3. Papilledema found in a pediatric patient presenting with microvascular dilation of the optic nerve, vessel obscuration and Paton's line (red arrows).

patients with true papilledema.[18,21] However, with the use of SD-OCT alone, many early cases of true papilledema may look similar to ONHD. A recent case study using SD-OCT in the evaluation of ONHD and true papilledema found no difference in RNFL thickness between eyes with mild papilledema and buried ONHD.[17] The concern lies in that potentially life- and sight-threatening conditions may be missed if one relies on a single modality test, as cases of ONHD can easily be confused with mild cases of papilledema.[17]

• ***Fundus autofluorescence (FAF).*** The calcific properties of drusen have inherent autofluorescent ability, thus ONHD will hyperfluoresce on FAF. Research shows this method is effective in diagnosing ONHD in pediatric patients.[22] Remember, however, that autofluorescence of drusen is inversely proportional to its depth, which means deeply buried drusen may be difficult to assess using FAF.[20] In true papilledema, the optic nerve swelling can extend into the surrounding tissue and mask the autofluorescence of the fundus, causing the appearance of an enlarged ONH.[22]

• ***Visual field testing.*** While ONHD is regarded as a benign cause of ONH elevation, functional changes, such as visual field defects, may be noted in rare cases. Visual field defects associated with ONHD include enlarged blind spots, localized focal defects, arcuate visual field defects or generalized constrictions. These may be present in 73% of visible drusen cases in contrast with only 36% of cases associated with buried drusen.[23]

As buried drusen begin to rise to the surface of the optic disc, compression of the neighboring axons can ensue, leading to cell apoptosis and, ultimately, progressive visual defects.[22] This is more commonly noted in the second decade of life.[24] Visual field testing can be used to monitor progression in cases of ONHD. Also, consider baseline testing in the early stages.[5] Clinicians should take into account the maturity and attention span of the pediatric patient when considering visual field testing in the pediatric population. Most children older than eight years can perform a fairly reliable visual field test.

Referral

In any case where the diagnosis of pseudopapilledema is not confirmed, referral to a neuro-ophthalmologist is warranted. Radiological imaging, such as MRI or CT, followed by lumbar puncture (LP) may be necessary. An opening pressure of greater than 280mm H_2O in the pediatric patient older than one year is highly suspicious for increased intracranial pressure.[9]

Although careful funduscopic evaluation of the optic disc can help differentiate pseudopapilledema from true papilledema, the pediatric population provides unique challenges. In the absence of associated intracranial pressure signs and symptoms, multimodal

noninvasive ophthalmic imaging techniques may be used as effective tools to differentiate ONHD from true papilledema—thus, aiding in prompt referrals when necessary, while preventing unnecessary referrals for more invasive testing, and decreasing patient and parental anxieties.[2,4] ■

Dr. Ubhi is an optometric resident in primary care and pediatrics at Nova Southeastern University College of Optometry.

Dr. Shechtman is a professor of optometry at Nova Southeastern University College of Optometry, and an attending optometric physician at the Eye Institute and Diabetic/Macula Clinic.

Dr. Green is an optometric resident in pediatrics and binocular vision at Nova Southeastern University College of Optometry.

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Amblyopia: When to Treat, When to Refer?

This common pediatric condition can be managed successfully with timely intervention. **By Erin Jenewein, OD**

As primary eye care practitioners, we are the first to diagnose and treat a variety of ocular conditions. The role we play becomes even more vital in our youngest patients. Amblyopia, often diagnosed during a patient's first eye examination, can be managed in a general optometric practice to dramatically improve quality of life in the developing child.

Amblyopia is defined as a decrease in the best-corrected visual acuity of one eye, or less frequently both eyes, in the absence of any structural or pathological changes. Also critical to this definition is the presence of a condition that causes the development of amblyopia, including either a significant anisometropic or isoametropic refractive error; a constant, unilateral strabismus; or some form of deprivation occurring before six years of age.[1]

Why is it important, as a primary care optometrist, to know how to diagnose and treat amblyopia? First, it is not an uncommon condition; the prevalence has been reported to be between 1% and 5% of the population, and it is the most common cause of vision impairment in children and young adults.[2-8] Second, it is critical not only to the ocular health but also to the overall well-being of our patients that we correctly diagnose and treat this condition.

It is often difficult to convince patients and parents to invest time and money into treating an amblyopic eye if the patient has good vision in the fellow eye. It is important to educate all parties about what happens if the child loses vision from injury or ocular disease in the non-amblyopic eye. Patients with amblyopia have almost twice the incidence of bilateral visual impairment as those without amblyopia, and their lifetime risk of serious vision loss in both eyes is estimated to be between 1.2% and 3.3%.[9,10] A bilateral loss of visual acuity can impact a patient's ability to drive, maintain employment and function in the world. It is our job, therefore, to not only diagnose and manage this condition, but to also educate our patients on the importance of treating amblyopia.

Causes of Amblyopia

As amblyopia is a rather imprecise term, clinicians must be familiar with the variety of potential etiologies that may be responsible.

• ***Refractive error.*** It is important to understand what is considered a 'significant' refractive error in the diagnosis of refractive amblyopia. Although there are no definitive refractive values at which amblyopia will absolutely occur, there are guidelines we can use to identify refractive errors that put our patients at risk for the development of refractive amblyopia. This also

helps identify patients who do not have a refractive error significant enough, leading us to investigate other causes of decreased best-corrected visual acuity.

Amblyopia can be caused by a significant anisometropia or, less commonly, significant bilateral refractive error. Isoametropic amblyopia is caused by image blur due to a high amount of bilateral ametropia. Refractive errors have to be high enough to prohibit a clear retinal image at any distance. Based on expert consensus outlined in the American Optometric Association's Clinical Practice Guidelines and the American Academy of Ophthalmology's Preferred Practice Patterns—supported by the data from the Multi-Ethnic Pediatric Eye Disease Study—significant refractive error causing isoametropic amblyopia can be defined as a minimum of approximately 6D to 8D of myopia, 4D to 5D of hyperopia and 2D to 2.5D of astigmatism in both eyes.[11-13] Lower values of ametropia can also result in anisometropic amblyopia—in addition to blur, the difference in refractive error can cause abnormal binocular vision and suppression. Patients with 3D of myopic anisometropia or more, 1.5D to 2D of astigmatic anisometropia and only 1D of hyperopic anisometropia are considered at risk for developing refractive amblyopia.[11-13]

• ***Strabismic amblyopia.*** This is caused by a constant, unilateral strabismus. It is important to note that the size of the deviation is not related to the development of amblyopia, nor is the size of the deviation related to the severity of amblyopia, and even a very small-angle, constant, unilateral strabismus can cause strabismic amblyopia. This highlights the importance of a careful cover test to ensure that the patient has a constant, unilateral deviation.

• ***Vision deprivation.*** Finally, amblyopia can be caused by form deprivation. Deprivation amblyopia is caused by a physical obstruction along the line of sight, which prevents a well focused, high contrast image on the retina. This type of amblyopia is relatively rare, found in only about 0.1% of the population, and is associated with conditions such as cataract, hyphema, corneal opacities, vitreous opacities or significant ptosis occurring early in life.[14]

Photo: Kathleen Elliott, OD

A thorough exam can uncover the underlying cause of a patient's amblyopia and help shape the course of treatment.

Diagnosis

When amblyopia is suspected, an initial clinical examination should include visual acuity testing, fixation, refraction, evaluation of ocular alignment and sensory fusion, stereoacuity, accommodation, ocular motility and a thorough ocular health evaluation.[11] Acuity testing can be particularly challenging in amblyopic patients: they generally perform poorly on full chart acuity, but exhibit better results on single-line and single-letter acuity testing due to the crowding phenomenon.[15] It is important, therefore, to use the same type of visual acuity testing on follow up examination to avoid obtaining an artificially high or low visual acuity measurement.[15] It can be difficult to test young children using full-line visual acuity, but using isolated letters may not adequately describe the vision loss seen in amblyopia.

The Amblyopia Treatment Studies use a single HOTV (for children ages three to under seven) or ETDRS (for children ages seven and older) optotype surrounded by crowding bars to evaluate visual acuity, which combines the ease of testing using single letter acuity with the addition of a consistent contour surrounding each letter.[16] Obtaining both single-line and single-letter visual acuity measurements give an estimate of the impact of crowding on the decrease in visual acuity in amblyopic patients.

An objective, and if possible a subjective, evaluation of refractive error should be performed during all initial examinations of patients with amblyopia, both before and after cycloplegia. Two drops of

cyclopentolate 1% are used in children over the age of one for cycloplegic retinoscopy and refraction. If the patient also needs a dilated fundus evaluation, mydriatic drops should be used in addition to cyclopentolate to give adequate mydriasis for a peripheral retinal examination. Subjective refraction can be challenging in patients with amblyopia, and it is important to have a good objective measure of refractive error, such as retinoscopy. When prescribing for patients with amblyopia, take into consideration the refractive error before and after cycloplegia, the amount of anisometropia after cycloplegia and the patient's ocular alignment.

Photo: Kathleen Elliott, OD

Spectacles are the first choice for bilateral refractive amblyopia treatment, considering many patients will improve with spectacle correction alone.

A careful evaluation of ocular alignment is necessary to determine the presence or absence of strabismus, and sensory fusion can be evaluated using random dot stereoacuity and Worth 4-Dot testing. An evaluation of fixation using a visuoscope target should also be done in all patients with amblyopia to evaluate for the presence or absence of eccentric fixation. Finally, a thorough ocular health evaluation is critical for proper diagnosis. Any pathological cause of decreased visual acuity must be ruled out prior to making a diagnosis of amblyopia. Patients with amblyogenic conditions such as strabismus or significant refractive error may also concurrently have an ocular disease process occurring, which may contribute to the decrease in visual acuity.

Treating with Spectacles

When deciding whether or not to treat amblyopia, age should not be a factor; amblyopia has been successfully treated in patients seven to 17 years of age.[17] In a study of amblyopia treatment in children of this age range, 53% of patients treated with spectacle correction, patching and atropine improved by at least 10 letters after 24 weeks of treatment.[17] Although it may be more challenging to treat older patients, all patients—even adults—should be given the option of amblyopia treatment, particularly given the increased relative risk of severe vision loss in both eyes in patients with amblyopia.

The first line of treatment in amblyopia is spectacle correction. Patients should be given spectacles and then monitored regularly, generally every six weeks, for improvement in visual acuity. It is intuitive that patients with bilateral, relatively symmetrical isoametropic amblyopia will have improved visual acuity with spectacle correction alone. In fact, most of these patients will completely resolve their amblyopia with spectacle correction alone within one year.[18] These patients are usually easy to treat and can be monitored until their amblyopia resolves, usually within one year, but often sooner.

Anisometropic and strabismic amblyopia are first treated with spectacle correction, but may require additional, more complex treatment such as penalization therapy if vision does not improve with spectacle correction alone. In one study, approximately one-third of children ages three to under seven with anisometropic amblyopia resolved their amblyopia with spectacle correction alone, and over 75% of patients improved two or more lines of visual acuity with spectacle correction alone.[19]

Surprisingly, another study showed patients with strabismic amblyopia and combined mechanism amblyopia had significant improvement with spectacle correction alone as well; over 75% of patients improved more than two lines of visual acuity, and over 50% improved more than three lines of visual acuity.[20] Overall, approximately 25% of patients with strabismus and combined mechanism amblyopia resolved their amblyopia completely with only spectacle correction over 18 weeks of treatment. Amblyopia still improved in approximately one fourth of children ages seven to 17, who are thought to be more difficult to treat than a younger cohort, with optical treatment alone.[17]

These results highlight the importance of beginning treatment with refraction correction and monitoring. Patients are generally followed every six weeks until the amblyopia resolves, or until their vision plateaus, requiring further treatment.

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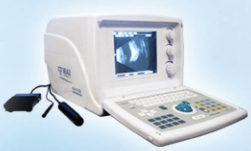

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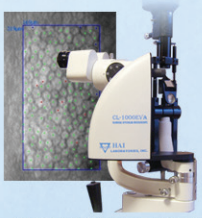

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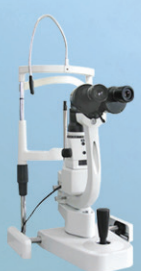

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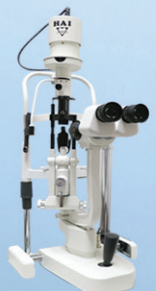
HAI SL-5000s Standard Slit Lamp



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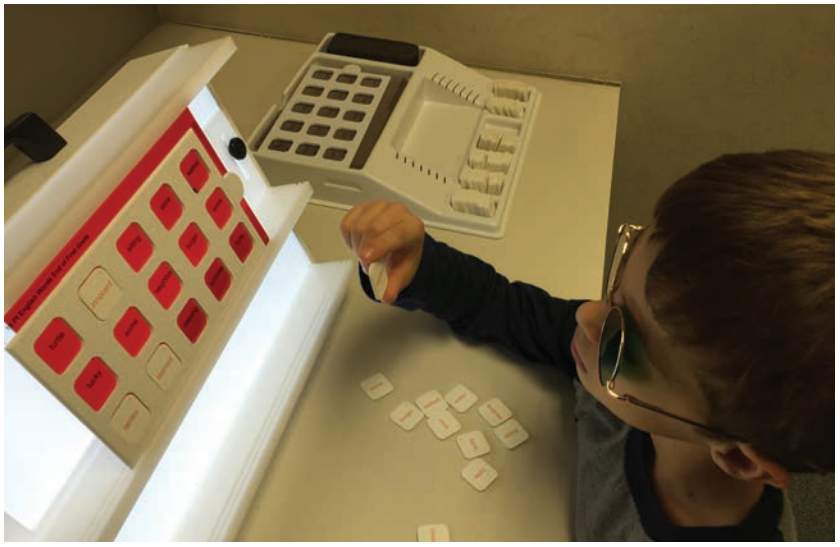

Photo: Marc B. Taub, OD, MS

Advanced amblyopia treatment may include vision therapy, such as anti-suppression therapy using the MFBF matching game.

Advanced Treatment

The next step in amblyopia treatment is penalization of the better-seeing eye. This can be achieved by a variety of methods, the most common being patching or atropine use to blur the sound eye. Patching for two hours per day is recommended for patients with moderate amblyopia (20/80 or better visual acuity), while patching for six hours is recommended for patients with severe amblyopia (20/100 or worse visual acuity).[21,22] Studies have found patients with severe amblyopia improved an average of 4.8 lines of visual acuity over four months with six hours of daily patching, and 62% of patients with moderate amblyopia had either an improvement of three lines of visual acuity or a visual acuity of 20/32 or better after four months of daily patching for two hours.[21,22]

In addition to patching, practitioners may also prescribe at least one hour of near activities per day while patching, such as tasks to improve tracking, fixation and accommodation. One study found no significant difference in visual acuity improvement when comparing near and distance activities during patching; however, the activities in this study were common near tasks and not specifically designed to improve monocular skills.[23] Computer activities, such as Amblyopia iNet (Vision Therapy Solutions), can also be used during patching to help improve monocular skills.

Patching compliance can be an issue when treating amblyopia, as patients and parents are often concerned about its social and cosmetic aspects. Studies using an occlusion dose monitor to objectively evaluate compliance have found that patients are compliant with patching therapy approximately 40% to 60% of the time.[24,25]

The use of atropine for penalization of the better-seeing eye is a good alternative for patients who do not comply with patching, as it has been found to have similar visual outcomes to patching.[26] To make compliance even simpler for patients and families, atropine can be used successfully on a daily basis or on weekend days only to treat amblyopia.[27] Weekend atropine has been found to be as effective as daily atropine for the treatment of moderate and severe amblyopia in children three to under seven years of age.[27,28] Bangerter filters, worn full-time over the sound eye's spectacle lens to degrade the image, have also been used to successfully treat moderate amblyopia.[29] This can also be a good treatment method for patients who are not compliant with patching or atropine penalization.

Another treatment currently under investigation is the use of binocular therapy to treat amblyopia. One approach is to use dichoptic stimuli, in which the amblyopic eye views a high contrast image and the fellow eye views a lower contrast image. This therapy has been implemented using games on an iPad and by having patients view movies with this type of stimulus.[33,34] The Pediatric Eye Disease Investigator Group is currently performing a randomized clinical trial comparing the effectiveness of binocular dichoptic iPad games to patching therapy in treating amblyopia.[35]

Challenges

One of the biggest challenges is the chance of recurrence after completion of treatment. A study of moderate and severe amblyopia treatment found approximately 25% of patients under age seven had a recurrence of amblyopia within the first year of stopping treatment, and children ages seven to 12 had a 7% chance of recurrence (worsening of two lines of visual acuity).[30,36] This recurrence is more common in patients with severe amblyopia who went from six hours of patching per day to no patching.[30] To help prevent this recurrence, patients should be weaned off patching therapy. Additionally, patients with a history of

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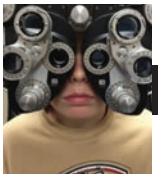

successfully treated amblyopia need continued close monitoring for a recurrence of amblyopia.

Residual amblyopia is another treatment challenge, considering vision does not improve sufficiently with one treatment for some patients. If the patient is currently patching for two hours, increasing patching to six hours per day may help improve visual acuity and treat residual amblyopia.[31] Another option is to switch from patching to atropine or vice-versa to see if acuity improves. Additionally, some practitioners combine therapies, such as using both patching and atropine therapy, although in a study of patients with residual amblyopia, combining patching and atropine did not improve vision more than patients that had patching gradually discontinued.[32]

Next Step: Comanagement

Depending on the type of amblyopia and the patient's treatment outcomes, they may require a referral to a specialist. Patients with strabismic amblyopia should see a specialist who can treat strabismus, once their vision has improved with amblyopia treatment. Additionally, if vision in the amblyopic eye is not improved with spectacle correction or patching treatment, additional testing and possible referral is necessary to rule out any underlying pathological condition that may be causing or contributing to the decreased vision.

If vision improvement plateaus and acuity is still not at the level of the sound eye, a referral to an optometrist who specializes in vision therapy may be appropriate. Vision therapy can help improve fixation, oculomotor skills and accuracy of accommodation, all of which may be poorer in patients with amblyopia. Vision therapy can also help treat suppression and improve binocular skills.

Amblyopia is commonly seen in optometric practice, and initially can be managed in a primary care setting. Many patients will improve their visual acuity, some to the point of resolution, with correction only or with correction and optical penalization. Patients whose amblyopia does not resolve with correction and penalization, as well as patients with strabismus, can be referred to a specialist in binocular vision for further treatment. Proper diagnosis and management of amblyopia in a primary care setting is important to providing amblyopic patients the best care and the best possible visual outcomes. ■

Dr. Jenewein completed a residency in pediatrics and binocular vision at Nova Southeastern University. She is an assistant professor at Salus University Pennsylvania College of Optometry.

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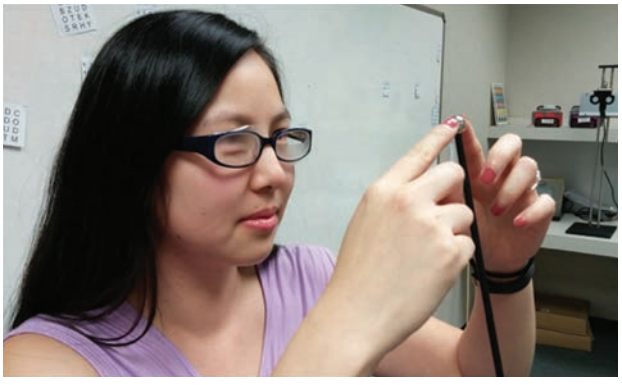
Recognizing TBI-related Vision Disorders

These visual signs and symptoms can help uncover concussions and will guide your approach to intervention. By Christopher L. Suhr, OD, Mark Shust, OD, Ranjoo Prasad, OD, Denise T. Wilcox, OD and Connie Chronister, OD

Because the term *traumatic brain injury* (TBI) encompasses a plethora of situations that range from mild to severe, clinicians often look for greater precision in the definitions and categorization of TBI. Mild traumatic brain injury (mTBI) and concussion are somewhat interchangeable terms used to define "a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces."[1,2] The difference between mTBI and moderate-to-severe TBI can be determined using the Glasgow coma scale (*Table 1*). A 2010 study found that 75% of an estimated 1.7 million people treated for brain injuries in the United States had mTBI.[3] Recently, head injuries among athletes and military service personnel—the two most common patient populations at risk for TBI—have shone a spotlight on the consequences affecting individuals with mTBI.[1]

This article provides an overview of common visual problems associated with TBI, guidelines for diagnostic assessment and management recommendations. An understanding of potential visual and ophthalmic changes following TBI is essential in allowing the primary care optometrist to properly care for affected patients.



This demonstration of oculomotor training shows techniques taught to patients dealing with traumatic brain injury-related vision disorders, notably convergence issues. Here, a patient is asked to locate a stimulus.

Presentations

Clinically, a TBI patient can have any combination of signs and symptoms that may include, but are not limited to, visible trauma, confusion, headaches, memory loss or muscle function problems. Visual disturbances common in mTBI patients can include photophobia, blurred vision, reading difficulties, eyestrain, diplopia, visual field defects, color vision changes and vestibular dysfunctions. Visual symptoms can impair rehabilitation and create significant restrictions with occupational, educational and other activities of daily living.[4-14]

TBI-related damage to the afferent pathway may include the optic nerve, optic tract, chiasm, optic radiation or occipital cortex. Damage to any part of this pathway may decrease visual acuity, which may result in reduced visual function and an inability to perform daily activities. In the worst-case

Table 1. Glasgow Coma Scale

Behavior	Response	Score
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion to pain (also termed decorticate)	3
	Abnormal extension to pain (also termed decerebrate)	2
	No response	1

TBI Classification	Loss of Consciousness	Glasgow Coma Total Score
Mild (mTBI)	Less than 30 minutes	13 and up
Moderate TBI	Greater than 30 minutes, but less than 6 hours	Between 9 and 12
Severe TBI	Greater than 6 hours	8 or less

scenario, there may be such significant loss that the individual ends up legally blind.

Photophobia

Increased sensitivity to light is a common visual symptom following TBI.[4-8] One study found that photophobia was the most common complaint of the TBI population studied.[6] Photosensitivity may be present in all types of lighting or it may be more pronounced in specific conditions, such as indoor fluorescent lighting or outdoor sunlight. The underlying cause of photophobia remains somewhat unclear but some evidence points to an alteration in the visual system's ability to adapt to prolonged light or darkness.[4,6] In addition, abnormal critical flicker fusion frequency—the minimum light flicker frequency for an individual to perceive a steady (non-flickering) presentation of light—may be related to discomfort with fluorescent lighting in some patients.[4,5,7,8,15]

Effective management begins with a thorough history that accounts for the patient's experience of the specific lighting conditions associated with their symptoms. Be sure to rule out any possible ocular inflammatory processes, such as uveitis, that may develop following trauma. Photophobia is typically addressed with filters, visors and attention to proper illumination. Management may also include changing the room lighting from fluorescent to incandescent or LED lightning indoors, employing a light tint in or outdoors and wearing sunglasses outside.

Blurred and decreased vision may occur following TBI as a result of refractive error, or structural ocular changes or both.[4,7,9] Refractive changes may occur with trauma to any refractive ocular structures, including the cornea and crystalline lens, as well as with accommodative dysfunctions, which can follow mTBI. Any ophthalmic examination following TBI must include a meticulous objective and subjective refraction so that any refractive errors can be precisely corrected.

Studies associate TBI trauma with physical changes in various ocular structures that may result in vision blur or distortion.[4,9,16] For example, an altered tear film composition may cause dry eye symptoms, including blink-related blur, distortion or foreign body sensation.

Corneal injury may result in keratitis or scarring, which can lead to loss of vision. The crystalline lens

This oculomotor training technique tests the patient's ability to track a stimulus, in this case a ball.

may develop a cataract or become dislocated in response to traumatic insult, causing vision distortion or diplopia. Vitreoretinal changes can include hemorrhages, detachments, floaters, maculopathies or other pathologies, any of which may lead to visual impairment.[4,9,15,16]

Careful examination of anterior and posterior segment ocular structures, including a dilated exam, is essential to identify and appropriately manage any anomalies associated with mTBI.

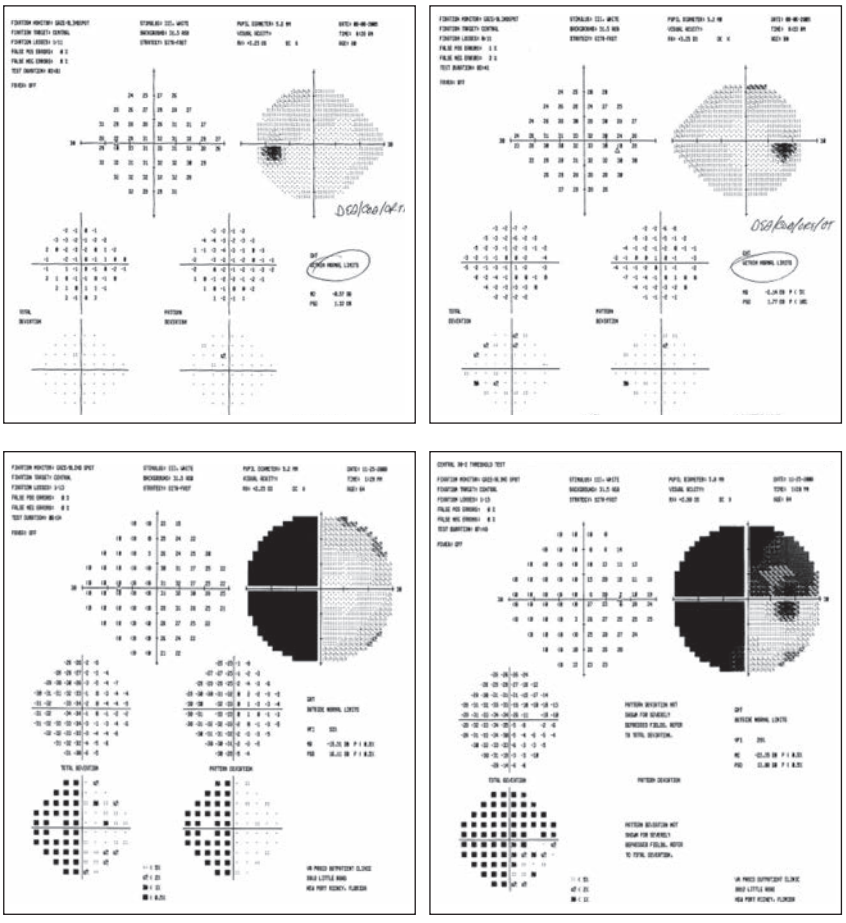
Accommodative Dysfunction

Mild TBI, like any damage to the accommodative pathways or the oculomotor nerve, may lead to accommodative dysfunction. Impaired accommodation can cause variable vision blur, visual fatigue, asthenopia and headaches, particularly with tasks at near distances, such as reading or computer work.[4,7,9,10]

The most common TBI-related accommodative disorder is accommodative insufficiency.[10] Reference values for minimum amplitude of accommodation, typically measured using an accommodative target such as a near-point card with the patient wearing their distance correction, are measured by subtracting 15 from one quarter of the patient's age (0.25[age]-15).

Test each eye monocularly and binocularly. Inconsistent values between eyes may indicate localized pathological changes in one eye, although inaccurate refractive correction in one or both eyes may indicate dysfunction.

Other accommodative disorders may include accommodative infacility and pseudomyopia.[10] Accommodative insufficiency may be treated with reading glasses or bifocal/progressive lenses. Both accommodative infacility and insufficiency may be improved with oculomotor rehabilitation.[9]



These fields are from the same patient. The clear fields, on top, were performed prior to those below (as the patient was a glaucoma suspect). Below you can see a visual field defect that was present after a severe TBI event (motor vehicle accident).

Oculomotor Dysfunctions

Both versional and vergence oculomotor dysfunctions can occur in TBI patients. Versions are conjugate eye movements and include pursuits, saccades and fixation.[4,7,9,16-18] Versional eye movements should be assessed as part of the oculomotor evaluation. Anomalies of the visual fields or damage to various vision-mediating neurological structures can affect these eye movements.[10,16] Dysfunction of versional eye movements may cause symptoms such as significant reading difficulties including reduced reading speed, loss of place when reading and re-reading text.[4,7,9,10]

The King-Devick (KD) test evaluates saccades through rapid number reading on a series of three testing cards, which become progressively more challenging due to variability in spacing between numbers. The test accurately and reliably identifies impaired saccadic function, which may occur following TBI.[19]

Vergence oculomotor functions refer to non-conjugate changes in eye position when viewing objects at varying distances.[4,7,9,17,16] Vergence disorders may be non-comitant (strabismic but the deviation

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- Provides long-lasting tear concentrations[2]
- Proven efficacy against a broad spectrum of pathogens including[1,3]:
  - *S. aureus, S. epidermidis, S. pneumoniae,* and *H. influenzae*
  - *Pseudomonas aeruginosa*

Indication

BESIVANCE® (besifloxacin ophthalmic suspension) 0.6% is a quinolone antimicrobial indicated for the treatment of bacterial conjunctivitis caused by susceptible isolates of the following bacteria: *Aerococcus viridans**, CDC coryneform group G, *Corynebacterium pseudodiphtheriticum**, *Corynebacterium striatum**, *Haemophilus influenzae*, *Moraxella catarrhalis**, *Moraxella lacunata**, *Pseudomonas aeruginosa**, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Staphylococcus hominis**, *Staphylococcus lugdunensis**, *Staphylococcus warneri**, *Streptococcus mitis* group, *Streptococcus oralis*, *Streptococcus pneumoniae*, *Streptococcus salivarius**

*Efficacy for this organism was studied in fewer than 10 infections.

Important Safety Information about BESIVANCE®

- BESIVANCE® is for topical ophthalmic use only, and should not be injected subconjunctivally, nor should it be introduced directly into the anterior chamber of the eye.
- As with other anti-infectives, prolonged use of BESIVANCE® may result in overgrowth of non-susceptible organisms, including fungi. If super-infection occurs, discontinue use and institute alternative therapy.
- Patients should not wear contact lenses if they have signs or symptoms of bacterial conjunctivitis or during the course of therapy with BESIVANCE®.
- The most common adverse event reported in 2% of patients treated with BESIVANCE® was conjunctival redness. Other adverse events reported in patients receiving BESIVANCE® occurring in approximately 1-2% of patients included: blurred vision, eye pain, eye irritation, eye pruritus and headache.
- BESIVANCE® is not intended to be administered systemically. Quinolones administered systemically have been associated with hypersensitivity reactions, even following a single dose. Patients should be advised to discontinue use immediately and contact their physician at the first sign of a rash or allergic reaction.
- Safety and effectiveness in infants below one year of age have not been established.

Please see brief summary of Prescribing Information on adjacent page.

To learn more about BESIVANCE® call your Bausch + Lomb sales representative today.

References: 1. BESIVANCE® Prescribing Information, September 2012. **2.** At 12 hours, the concentration of besifloxacin in tears was >10 µg/mL. Proksch JW, Granvil CP, Siou-Mermet R, Comstock TL, Paterno MR, Ward KW. Ocular pharmacokinetics of besifloxacin following topical administration to rabbits, monkeys, and humans. *J Ocul Pharm Ther.* 2009;25(4):335-344. **3.** Comstock TL, Paterno MR, Usner DW, Pichichero ME. Efficacy and safety of besifloxacin ophthalmic suspension 0.6% in children and adolescents with bacterial conjunctivitis: a post hoc, subgroup analysis of three randomized, double-masked, parallel-group, multicenter clinical trials. *Paediatr Drugs.* 2010;12(2):105-112.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION
This Brief Summary does not include all the information needed to use Besivance safely and effectively. See full prescribing information for Besivance.

Besivance (besifloxacin ophthalmic suspension) 0.6%

Sterile topical ophthalmic drops
Initial U.S. Approval: 2009

1 INDICATIONS AND USAGE

Besivance® (besifloxacin ophthalmic suspension) 0.6%, is indicated for the treatment of bacterial conjunctivitis caused by susceptible isolates of the following bacteria:

*Aerococcus viridans**, CDC coryneform group G, *Corynebacterium pseudodiphtheriticum**, *Corynebacterium striatum**, *Haemophilus influenzae, Moraxella catarrhalis**, *Moraxella lacunata**, *Pseudomonas aeruginosa**, *Staphylococcus aureus, Staphylococcus epidermidis, Staphylococcus hominis**, *Staphylococcus lugdunensis**, *Staphylococcus warneri**, *Streptococcus mitis* group, *Streptococcus oralis, Streptococcus pneumoniae, Streptococcus salivarius**

*Efficacy for this organism was studied in fewer than 10 infections.

2 DOSAGE AND ADMINISTRATION

Invert closed bottle and shake once before use.

Instill one drop in the affected eye(s) 3 times a day, four to twelve hours apart for 7 days.

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Topical Ophthalmic Use Only NOT FOR INJECTION INTO THE EYE.

Besivance is for topical ophthalmic use only, and should not be injected subconjunctivally, nor should it be introduced directly into the anterior chamber of the eye.

5.2 Growth of Resistant Organisms with Prolonged Use As with other anti-infectives, prolonged use of Besivance (besifloxacin ophthalmic suspension) 0.6% may result in overgrowth of non-susceptible organisms, including fungi. If super-infection occurs, discontinue use and institute alternative therapy. Whenever clinical judgment dictates, the patient should be examined with the aid of magnification, such as slit-lamp biomicroscopy, and, where appropriate, fluorescein staining.

5.3 Avoidance of Contact Lenses Patients should not wear contact lenses if they have signs or symptoms of bacterial conjunctivitis or during the course of therapy with Besivance.

6 ADVERSE REACTIONS

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

The data described below reflect exposure to Besivance in approximately 1,000 patients between 1 and 98 years old with clinical signs and symptoms of bacterial conjunctivitis.

The most frequently reported ocular adverse reaction was conjunctival redness, reported in approximately 2% of patients.

Other adverse reactions reported in patients receiving Besivance occuring in approximately 1-2% of patients included: blurred vision, eye pain, eye irritation, eye pruritus and headache.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy Pregnancy Category C. Oral doses of besifloxacin up to 1000 mg/kg/day were not associated with visceral or skeletal malformations in rat pups in a study of embryo-fetal development, although this dose was associated with maternal toxicity (reduced body weight gain and food consumption) and maternal mortality. Increased post-implantation loss, decreased fetal body weights, and decreased fetal ossification were also observed. At this dose, the mean C_{max} in the rat dams was approximately 20 mcg/mL, >45,000 times the mean plasma concentrations measured in humans.

The No Observed Adverse Effect Level (NOAEL) for this embryo-fetal development study was 100 mg/kg/day (C_{max}, 5 mcg/mL, >11,000 times the mean plasma concentrations measured in humans).

In a prenatal and postnatal development study in rats, the NOAELs for both fetal and maternal toxicity were also 100 mg/kg/day. At 1000 mg/kg/day, the pups weighed significantly less than controls and had a reduced neonatal survival rate. Attainment of developmental landmarks and sexual maturation were delayed, although surviving pups from this dose group that were reared to maturity did not demonstrate deficits in behavior, including activity, learning and memory, and their reproductive capacity appeared normal.

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

8.3 Nursing Mothers Besifloxacin has not been measured in human milk, although it can be presumed to be excreted in human milk. Caution should be exercised when Besivance is administered to a nursing mother.

8.4 Pediatric Use The safety and effectiveness of Besivance® in infants below one year of age have not been established. The efficacy of Besivance in treating bacterial conjunctivitis in pediatric patients one year or older has been demonstrated in controlled clinical trials *[see CLINICAL STUDIES (14)]*.

There is no evidence that the ophthalmic administration of quinolones has any effect on weight bearing joints, even though systemic administration of some quinolones has been shown to cause arthropathy in immature animals.

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

12 CLINICAL PHARMACOLOGY

12.1 Mechanism of Action Besifloxacin is a fluoroquinolone antibacterial *[see CLINICAL PHARMACOLOGY (12.4)]*.

12.3 Pharmacokinetics Plasma concentrations of besifloxacin were measured in adult patients with suspected bacterial conjunctivitis who received Besivance bilaterally three times a day (16 doses total). Following the first and last dose, the maximum plasma besifloxacin concentration in each patient was less than 1.3 ng/mL. The mean besifloxacin C_{max} was 0.37 ng/mL on day 1 and 0.43 ng/mL on day 6. The average elimination half-life of besifloxacin in plasma following multiple dosing was estimated to be 7 hours.

12. Microbiology

Besifloxacin is an 8-chloro fluoroquinolone with a N-1 cyclopropyl group. The compound has activity against Gram-positive and Gram-negative bacteria due to the inhibition of both bacterial DNA gyrase and topoisomerase IV. DNA gyrase is an essential enzyme required for replication, transcription and repair of bacterial DNA. Topoisomerase IV is an essential enzyme required for partitioning of the chromosomal DNA during bacterial cell division. Besifloxacin is bactericidal with minimum bactericidal concentrations (MBCs) generally within one dilution of the minimum inhibitory concentrations (MICs).

The mechanism of action of fluoroquinolones, including besifloxacin, is different from that of aminoglycoside, macrolide, and β-lactam antibiotics. Therefore, besifloxacin may be active against pathogens that are resistant to these antibiotics and these antibiotics may be active against pathogens that are resistant to besifloxacin. *In vitro* studies demonstrated cross-resistance between besifloxacin and some fluoroquinolones.

In vitro resistance to besifloxacin develops via multiple-step mutations and occurs at a general frequency of $< 3.3 \times 10^{-10}$ for *Staphylococcus aureus* and $< 7 \times 10^{-10}$ for *Streptococcus pneumoniae*.

Besifloxacin has been shown to be active against most isolates of the following bacteria both *in vitro* and in conjunctival infections treated in clinical trials as described in the INDICATIONS AND USAGE section:

*Aerococcus viridans**, CDC coryneform group G,

*Corynebacterium pseudodiphtheriticum**, *C. striatum**, *Haemophilus influenzae, Moraxella catarrhalis**, *M. lacunata**, *Pseudomonas aeruginosa**, *Staphylococcus aureus, S. epidermidis, S. hominis**, *S. lugdunensis**, *S. warneri**, *Streptococcus mitis* group, *S. oralis, S. pneumoniae, S. salivarius**

*Efficacy for this organism was studied in fewer than 10 infections.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals to determine the carcinogenic potential of besifloxacin have not been performed.

No *in vitro* mutagenic activity of besifloxacin was observed in an Ames test (up to 3.33 mcg/plate) on bacterial tester strains *Salmonella typhimurium* TA98, TA100, TA1535, TA1537 and *Escherichia coli* WP2uvrA. However, it was mutagenic in *S. typhimurium* strain TA102 and *E. coli* strain WP2(pKM101). Positive responses in these strains have been observed with other quinolones and are likely related to topoisomerase inhibition.

Besifloxacin induced chromosomal aberrations in CHO cells *in vitro* and it was positive in an *in vivo* mouse micronucleus assay at oral doses × 1500 mg/kg. Besifloxacin did not induce unscheduled DNA synthesis in hepatocytes cultured from rats given the test compound up to 2,000 mg/kg by the oral route. In a fertility and early embryonic development study in rats, besifloxacin did not impair the fertility of male or female rats at oral doses of up to 500 mg/kg/day. This is over 10,000 times higher than the recommended total daily human ophthalmic dose.

14 CLINICAL STUDIES

In a randomized, double-masked, vehicle controlled, multicenter clinical trial, in which patients 1-98 years of age were dosed 3 times a day for 5 days, Besivance was superior to its vehicle in patients with bacterial conjunctivitis. Clinical resolution was achieved in 45% (90/198) for the Besivance treated group versus 33% (63/191) for the vehicle treated group (difference 12%, 95% CI 3% - 22%). Microbiological outcomes demonstrated a statistically significant eradication rate for causative pathogens of 91% (181/198) for the Besivance treated group versus 60% (114/191) for the vehicle treated group (difference 31%, 95% CI 23% - 40%). Microbiologic eradication does not always correlate with clinical outcome in anti-infective trials.

17 PATIENT COUNSELING INFORMATION

Patients should be advised to avoid contaminating the applicator tip with material from the eye, fingers or other source.

Although Besivance is not intended to be administered systemically, quinolones administered systemically have been associated with hypersensitivity reactions, even following a single dose. Patients should be advised to discontinue use immediately and contact their physician at the first sign of a rash or allergic reaction.

Patients should be told that although it is common to feel better early in the course of the therapy, the medication should be taken exactly as directed. Skipping doses or not completing the full course of therapy may (1) decrease the effectiveness of the immediate treatment and (2) increase the likelihood that bacteria will develop resistance and will not be treatable by Besivance or other antibacterial drugs in the future.

Patients should be advised not to wear contact lenses if they have signs or symptoms of bacterial conjunctivitis or during the course of therapy with Besivance.

Patients should be advised to thoroughly wash hands prior to using Besivance.

Patients should be instructed to invert closed bottle (upside down) and shake once before each use. Remove cap with bottle still in the inverted position. Tilt head back, and with bottle inverted, gently squeeze bottle to instill one drop into the affected eye(s).

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changes depending on the gaze), and may occur with damage to cranial nerves III, IV or VI, or comitant (strabismic and the deviation remains relatively constant). Symptoms of vergence disorders occur only under binocular viewing conditions and may include eyestrain, double vision and headaches.[4,7,9,10,15]

The most common vergence dysfunction in TBI patients is a convergence insufficiency, usually occurring as a result of damage to the oculomotor nerve or the medial rectus muscles, causing symptoms to be most evident when reading or performing other near tasks.[20-22] Patients with convergence insufficiency characteristically have exophoria at near distances greater than far, a reduced nearpoint of convergence and a reduced positive fusional vergence range.[9,10,14] Positive fusional vergence range values that are less than double that of the exophoric posture are likely to be associated with symptomatic convergence insufficiency.[9,10,14]

Vergence testing is conducted using a phoropter, but you may obtain more reliable measurements using prism bars in free space.

Research shows convergence insufficiency often presents itself with other visual dysfunctions, such as accommodative disorders. Each condition may need to be addressed separately.[28] Management of accommodative and vergence dysfunction may include visual therapy to increase fusional range, vergence facility, and accommodative amplitude and facility.[3,21,24] Therapy may also target saccadic and pursuit dysfunctions.

Testing of these areas post-treatment along with subjective improvement of symptoms will indicate outcome and help the clinician develop a means of hopefully improving visual function.

Interventions: Vision Training

Many TBI patients respond well to efforts to improve vision, cope with impairment, or both. In two studies, one research team shows that ocular motor rehabilitation has improved accommodative facility and amplitude, as well as vergence responsivity. This may be due to plasticity of the neural system and "oculomotor learning effects." The improvement in accommodative amplitude "reflects an increase in neuronal firing (through recruitment) and/or better synchronization of the accommodatively based midbrain related neurons," according to research.[23,33,34]

A short-term follow-up study shows persistence or delayed improvement of 62% of the clinical oculomotor parameters tested.[29] A retrospective analysis concluded that their "current clinical sample exhibited either complete or marked reduction in their oculomotor–based symptoms and associated clinical signs with the maintenance of the symptom reduction and sign improvements at the two month to three month follow up."[26]

Investigators noted that it was the combination of motor training with attention training that yielded the best prospect for improved vergence.[35] Protocols developed for training to improve binocular vision included visual targets involving motor perceptual, memory and attention tasks. "Training allows a patient to have heightened attention to manifestation of visual deficits such as blurring of an image, corresponding motor response that compensates for the deficit," say the authors. "With repetition it becomes reflexive."[35]

In other words, the more that a patient has the aforementioned areas stimulated, the better the responses become.

Oculomotor rehabilitation can take place either in office or at home—for instance, in the office first to customize the regimen to the patient's specific needs, and then continued at home. Keep in mind that other, non-ocular dysfunctions may affect the outcome of rehabilitation. Many individuals who have experienced TBI may also have concurrent memory or cognitive issues, or both, which may make ocular motor rehabilitation more challenging.

A review of studies for "evidence regarding the use of oculomotor based vision assessment to identify and monitor recovery from mild TBI" was able to identify "that limitations include small, inadequately described study populations; lack of clearly reported inclusion."[36]

The review also mentioned "inadequate detail regarding study protocols and procedures, lack of detail regarding the diagnosis of mTBI and lack of blinded assessors and limited information on matching control participants."[40]

Visual Field Defects

Various presentations of visual field defects may occur following TBI as a result of damage to any portion of the visual pathway from the visual cortex of the brain onward to the retina.[4,7,25,26]

Defects may include constriction of the fields and either isolated or multiple scattered defects throughout the fields, with or without a generalized decrease in sensitivity.[4,7,25] Lateralized field defects such as homonymous hemianopias may also occur with or without neglect, in which patients are fully unaware of objects located in space within the visual field defects.

Symptoms of visual field defects include mobility issues (e.g., patients bumping into objects), reading difficulties and trouble locating items in tasks of daily living such as eating. Homonymous hemianopias create significant safety challenges, especially when associated with neglect and any activities that require an accurate awareness of one's surroundings.[4,7,25]

Screening for gross field defects by confrontation testing is useful, but more detailed evaluation with

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"This is a freakishly fantastic lens! I was skeptical at first but I have now refit more than a dozen monthly replaceable multifocal lens wearers who were happy with their lenses but wished it could be just a little better. The typical response is elation. One patient even called me a god!"

Michael Ciszek, OD

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"This is not a 'one size fits most' lens. Instead, the design matches what is happening in the maturing eye. With pupil optimization for both age and refractive error, Johnson & Johnson Vision Care, Inc. has done all the work of incorporating information from thousands of eyes to make it easy for us to get it right the first time."

Cheri Vincent-Riemer, OD, FAAO

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1-DAY ACUVUE® MOIST is the #1 globally prescribed daily disposable brand, with an exceptional safety profile and UV blocking.†* With LACREON® Technology, the INFINITY EDGE™ Design, and the comfortable, low modulus of etafilcon A, 1-DAY ACUVUE® MOIST was designed to help keep moisture in and irritation out, which is particularly important for a presbyope.

"The etafilcon A material is an ideal choice for a multifocal lens. Given the dynamics of the lids, tear film and ocular surface in the aging eye, presbyopic patients have a much higher chance of successful contact lens wear in a highly wettable, hydrophilic material."

Clarke D. Newman, OD, FAAO

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

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

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

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Another feature unique to this lens is the hybrid back surface. The back surface has an aspheric center, matching the natural shape of the cornea, to help the lens drape correctly on the cornea and preserve the integrity of the complex front-surface optics. And, it has a spherical periphery to help ensure centration of the optics over the pupil.

In clinical studies, normal eyes with Ks between 38.75 D and 48.50 D could be successfully fit with the single 8.5-mm base curve.

"I have found that patients adapt to this lens even better than I had expected. Perhaps due to the elegant lens design and position of the optics, the near vision is very powerful. In addition, computer and distance vision are fantastic as well.

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

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A lot of science and years of experience have gone into developing a robust fitting guide for 1-DAY ACUVUE® MOIST Brand MULTIFOCAL Lenses. The result is a lens that is easy to fit.

"I admit I am typically the first person to disregard fitting guides, but with 1-DAY ACUVUE® MOIST Brand MULTIFOCAL I have been following the fitting guide exactly. About 75% of the time, I'm successful with the FIRST set of lenses. In another 10% or 15% of cases I just need to make one change."
Charles Clayton, OD

"This lens is super easy to fit in a wide range of patients, from prior multifocal lens wearers to single-vision or monovision wearers to contact lens neophytes and even some post-LASIK patients. The success rate in my first 35 completed fits, which included all those types of patients, was very high: 76.4% were satisfied with their vision and comfort."
Bridgitte Shen Lee, OD

A Lens that Works for Your Presbyopes

Presbyopic contact lens wearers are so eager to stay in contact lenses that many are willing to tolerate big tradeoffs. But what if you could offer them great distance and near vision, all-day comfort, and the health and convenience benefits of a daily disposable lens? With 1-DAY ACUVUE® MOIST Brand MULTIFOCAL Contact Lenses, you can.

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

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

automated or Goldmann perimetry is essential to accurately localize and quantify any suspected defects.

Useful field of vision (UFOV) testing may also be helpful. This computer-based assessment of focused and divided visual function requires the patient to complete three subtests with variable numbers of objects and background noise.[15,26] UFOV may serve as a more effective evaluation of one's visual attention in an environment of distraction, or noise, and may better predict an individual's visual function in real-world situations.

Post-chiasmal afferent damage can cause visual field loss, such as a homonymous hemianopia or a hemianopia of just one eye.[27,28] Patients may complain of bumping into objects on the side of the field loss and a fear of traveling independently. Individuals with loss of inferior field may have greater problems with mobility, as they may have difficulty with stairs and curbs. Rehabilitation goals stress peripheral awareness and may include scanning and compensatory training, or large diopter Fresnel prism placed base out toward the field loss.[7,8]

Other lens options include peripheral prismatic lenses, such as the Gottlieb, Peli or Sectoral prisms. Specialty optical labs may make these devices. Further training with these lenses with a rehabilitation specialist is needed.

Individuals with hemianopic field loss may also have difficulty reading, with left-sided field loss preventing visualization of the first few words of a sentence or right-sided loss obscuring the last few words. Compensatory techniques such as scrolling and tilting the material away from field loss may help. A typoscope or line guide may help with tracking.

Interventions: Low Vision Aids

After performing refraction and BCVA assessment, consider low vision rehabilitation services and devices. For short duration distance-spotting tasks, such as seeing faces and signs, a hand-held or spectacle-mounted telescope apparatus might help. The patient may also find hand-held magnifiers useful for near-vision, such as reading price tags and labels.

For more severe impairments, electronic hand-held magnifiers or closed circuit television may prove beneficial. Non-optical aids, such as large print or auditory devices (for instance, a talking watch), may be considered. Using tactile markers for the stove to help identify burner settings may assist with orientation. This can also be used for clothing to identify and coordinate colors and outfits. Today, low vision smartphone apps can help identify money and colors.

If your office doesn't have the resources to cater to low vision patients, refer them to a nearby specialist. Every state has an agency to provide services for the visually impaired. The individual may have already had low vision rehabilitation services, if the injury was longstanding. While long-term TBI patients may have already seen a low vision specialist, their needs and goals will change over time, as will available low vision technology.

Afferent damage can also reduce contrast sensitivity, which a patient may refer to as "blurred vision." Often, having proper illumination helps increase the contrast to allow for comfortable reading. The amount, type and intensity of illumination are subjective. Another common complaint from patients with contrast sensitivity loss is difficulty with steps and curbs. This typically occurs in severe cases. Refer these patients to an orientation and mobility specialist.

Visual-vestibular Disorders

TBI patients may experience visual-vestibular dysfunctions characterized by dizziness, vertigo, nausea, balance problems, photophobia in fluorescent lighting and increased motion sensitivity.[4,10,29] Words may seem to move around while reading and computer tasks may be problematic due to monitor light flicker or sensitivity to scrolling motions. Visual-vestibular disorders likely occur due to abnormal function of the vestibular-ocular reflex (VOR) that controls gaze stabilization. The VOR stabilizes images on the retina during head movements by producing eye movements opposite that of the head movements.[4,12,16,29-32]

Dysfunction of the VOR may occur with damage to the semicircular canals in the ears, the oculomotor nerve (CNIII), the abducens nerve (CNVI), the acoustic nerve (CNVIII), or any neurological structures integrating these components.[4,10,15,16,22,31,32] Detailed questioning regarding related symptoms in the patient history is especially useful in identifying and addressing potential visual-vestibular disorders that may result from TBI. For these patients, consultation with an ear, nose and throat specialist may be warranted to rule out other pathology. Also, a consultation with an occupational therapist may be beneficial for an evaluation and assistance for mobility purposes.

In our offices, we will all ultimately see patients who have had TBI, whether it be from an athletic or service injury, motor vehicle accident or fall. These patients often have visual concerns that range in complexity and severity. Keeping abreast of both the successes and limitations of recent research can help clinicians tailor a treatment plan to the individual's needs. ■

Dr. Suhr practices at the Corporal Michael J Crescenz Department of Veterans Affairs Medical Center.

Interventions: Tinted Lenses

Since photosensitivity and glare are common complaints in our local population at the Philadelphia (Corporal Michael J. Crescenz) Veterans Administration Medical Center, we conducted a small study to evaluate responses to various tints and filters by 20 patients (age 24 to 64 years; 18 males and two females).

Before prescribing a tint, indoor glare was tested with lights of various intensities affixed to the ETDRS chart, and outdoor glare under bright sunlight, intermittent sunlight and overcast conditions. To accomplish this, the patient stands with their back towards the sun and then turns and walks into the direction of the sun while trialing several tints.

Patient preference for filters varied, with no clear associations noted. However, a wraparound prescription quality frame with the appropriate tint has been quite helpful for our patients.

The results of this unpublished study show that the appropriate tint for each patient resulted in reduction of glare sensitivity for all patients. It is therefore important that the clinician consider multiple options to provide the best comfort possible as each patient may have differing success with different options.

These tinted lenses act as filters to assist patients suffering from a common TBI-related visual symptom, photophobia.

Dr. Shust practices at the Corporal Michael J Crescenz Department of Veterans Affairs Medical Center.

Dr. Prasad practices at the University of Pennsylvania Scheie Eye Institute and the Corporal Michael J Crescenz Department of Veterans Affairs Medical Center.

Dr. Wilcox practices at the Corporal Michael J Crescenz Department of Veterans Affairs Medical Center and works in the Advanced Low Vision Clinic.

Dr. Chronister practices at the Corporal Michael J Crescenz Department of Veterans Affairs Medical Center and works in the Advanced Low Vision Clinic.

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Case Report

Pattern Recognition:

How to Identify and Confirm Multifocal Pattern Dystrophy

This rare condition can be difficult to diagnose. Learn the important differentials and fundamental tools to get your patient on the track to proper management.

By Jonathan R. Hamilton, OD, and Christine L. Burke, OD

Multifocal pattern dystrophy (MPD) is one of the five types of autosomal dominant pattern dystrophies. These dystrophies are rooted in an inherited mutation on the peripherin/retinal degeneration slow (RDS) gene.[1,2] The onset of the presentation of autosomal dominant pattern dystrophies is typically midlife; multifocal pattern dystrophy more specifically presents between the fourth and sixth decades.[3-7] Autosomal dominant pattern dystrophies are characterized by normal to mild visual impairment and some patients report mild visual disturbances. Visual disturbances and deficits typically progress very little, with vision remaining mostly intact throughout life. While mild visual impact is typical, some cases report late vision loss secondary to atrophy, choroidal neovascularization or both.[1,8-10] MPD typically presents with bilateral and symmetrical findings and shows as scattered, yellow, triradiate, fleck-like lesions in the posterior pole and arcades in the fundus.[3,5,11] The following report will discuss a case of multifocal pattern dystrophy, the differentials to consider, additional workup and management.

Case Report

A 58-year-old black male was referred to the eye clinic for a diabetic eye exam without visual or ocular health complaints. His ocular history was pertinent for macular pigmentary disturbance, which was first noted by a previous care provider in 2001 and determined to be congenital. His systemic history was pertinent for:

- ***Type 2 diabetes*** for approximately one year with a hemoglobin A1c of 6.7, controlled with diet and exercise.
- ***Hypertension,*** with the most recent blood pressure reading of 127/79, controlled with amlodipine 5mg, hydrochlorothiazide 25mg and atenolol 100mg, all once daily.
- ***Hyperlipidemia,*** with recent cholesterol of 197, HDL 40, LDL 132.8 and triglycerides 121, controlled with diet.
- ***Esophageal reflux,*** but the patient is currently asymptomatic and not being actively treated.
- Other systemic medications, including aspirin 81mg once daily, ibuprofen 600mg TID as needed and sildenafil 100mg, half a tablet as needed.

The patient had no known environmental or medical allergies. Entrance testing revealed the patient's visual acuity was 20/20 OD and 20/20-2 OS, with pupils equal and reactive to light in both eyes without an afferent pupillary defect. Ocular motility was full and smooth in both eyes and confrontation visual fields were full in both eyes. Upon biomicroscopic

examination, mild meibomian gland stasis was noted in both eyes with all other structures remaining normal. Intraocular pressure (IOP) was taken with applanation tonometry using 0.25% fluorescein sodium/0.4% benoxinate hydrochloride ophthalmic solution and was measured at 18mm Hg OD and 17mm Hg OS.

Dilation was performed with 2.5% phenylephrine hydrochloride ophthalmic solution and 1% tropicamide ophthalmic solution. The dilated exam revealed a 0.3 cup-to-disc ratio both horizontally and vertically with healthy rim tissue in both eyes. The macula was flat and remarkable for subtle pigment disruption, while the posterior pole was remarkable for scattered, well-defined hypopigmented/drusenoid-like formations in both eyes (*Figure 1*). The periphery was unremarkable in both eyes.

Amsler grid testing was normal in the right eye, but revealed a small, less-than-one-degree area of metamorphopsia two degrees temporal to fixation in the left eye. Fundus photos documented the scattered hypopigmented lesions, and optical coherence tomography (OCT) yielded basal lamina drusen-like formations at the level of the outer photoreceptor segments and anterior retinal pigment epithelium (RPE) (*Figure 2*).

At this time, the differential diagnoses were: fundus flavimaculatus, multifocal pattern dystrophy and basal laminar drusen variant. The patient returned for fluorescein angiography (FA) and fundus autofluorescence (FAF) imaging to further refine the differentials. The FA showed hyperfluorescence of the lesions during early phases with normal choroidal flush and residual staining of the lesions in late phases (*Figure 3*). The autofluorescence imaging revealed scattered hyperfluorescent lesions in the posterior pole and hypofluorescence corresponding to the macular RPE disruption *(Figure 4)*.

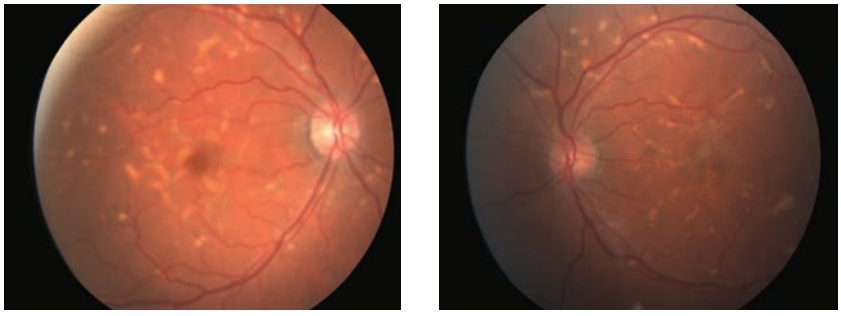

Fig. 1. These fundus images of a 58-year-old black male with multifocal pattern dystrophy in both eyes show a triradiate configuration of flecks within the posterior pole, extending just beyond the vascular arcades.

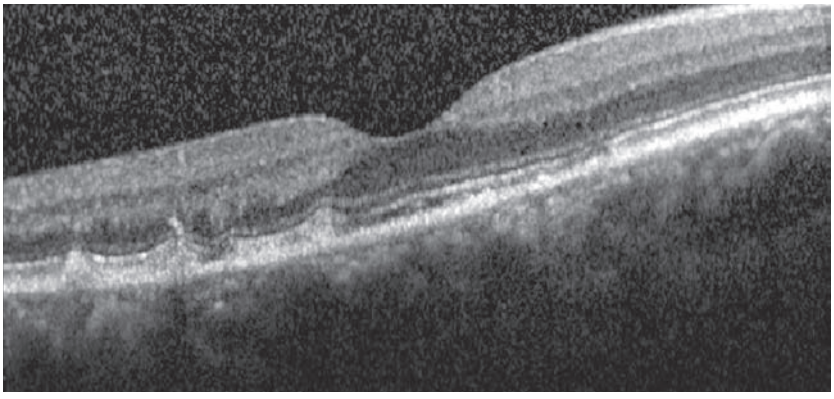

Fig. 2. The patient had basal lamina drusen-like formations at the level of the outer photoreceptor layer and anterior retinal pigment epithelium.

Subsequent to testing and consultation with a retina specialist, the patient was diagnosed with multifocal pattern dystrophy. This conclusion was based on the lack of progression in the fundus findings, FA findings and unaffected vision for more than 10 years from when the retinal findings were first documented. Treatment and management for the patient included regular monitoring of fundus with dilated fundus exam to rule out the formation of choroidal neovascularization, education about the inheritance pattern, and home Amsler grid testing.

Discussion

Autosomal dominant pattern dystrophies can be broken into five groups:

- Adult-onset foveomacular vitelliform pattern dystrophy.
- Butterfly-shaped pigment dystrophy.
- Reticular dystrophy of the RPE.
- Multifocal pattern dystrophy simulating fundus flavimaculatus.
- Coarse pigment mottling in the macula (fundus pulverulentus)

For more, see *Table 1*, next page.

Autosomal dominant pattern dystrophies are characterized by mild, midlife visual disturbances in the presence of various fundus findings.[5,11] These visual disturbances typically progress very little, with the vision remaining mostly intact until late adulthood; however, a

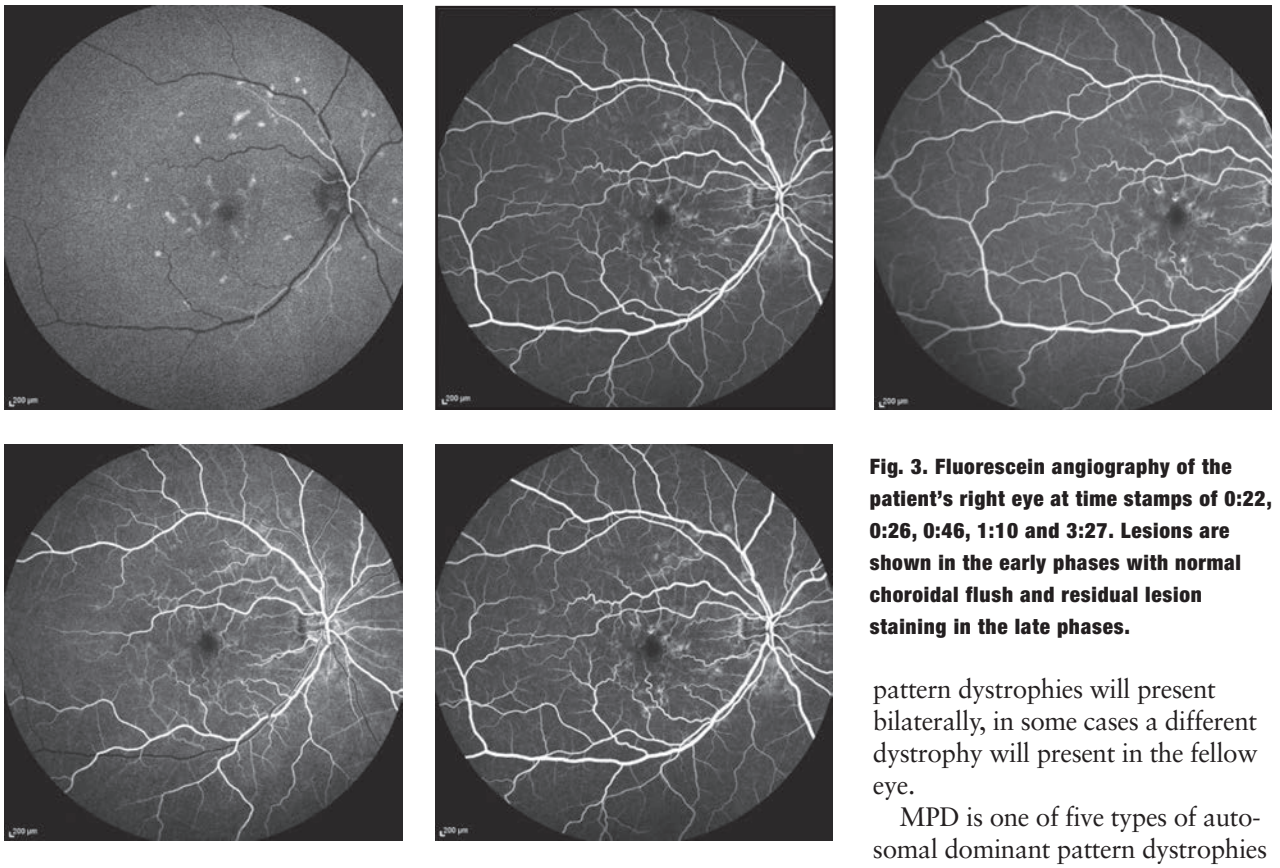

Fig. 3. Fluorescein angiography of the patient's right eye at time stamps of 0:22, 0:26, 0:46, 1:10 and 3:27. Lesions are shown in the early phases with normal choroidal flush and residual lesion staining in the late phases.

minority of patients will experience vision loss secondary to atrophy, choroidal neovascularization or both.[1,8-10] While a majority of pattern dystrophies will present bilaterally, in some cases a different dystrophy will present in the fellow eye.

MPD is one of five types of autosomal dominant pattern dystrophies that have slight variations in vision, onset and retinal presentation *(Table 1)*. These conditions are characterized by deep, intraretinal yellow, orange, or gray pigmentary deposits/changes.[6,11,12] Pattern dystrophies have a variable rate of occurrence in the general population, as it can be inherited dominantly or can occur by various mutations in the peripherin/RDS gene; multifocal pattern dystrophy is inherited through autosomal dominant mutations in the peripherin/RDS gene on chromosome 6.[3,11,13,14]

While genotypically similar to other pattern dystrophies, multifocal pattern dystrophy is phenotypically unique. It can be distinguished from other autosomal dominant pattern dystrophies by its characteristic well demarcated, irregular, yellowish flecks, which take on a triradiate configuration.[3,5,11] These

Table 1. Five Types of Autosomal Dominant Pattern Dystrophies and Typical Characteristics

Dystrophy	Typical Age of Onset	Typical VA	Retinal Presentation
Adult-Onset Vitelliform Dystrophy	4th-6th decades	20/30-20/60	Bilateral, circular, 1/3-1DD
Butterfly-Shaped Pigment Dystrophy	2nd-5th decades	20/20-20/25	Bilateral, triradiate hyperpigmentation
Reticular Dystrophy	5th decade	20/30-20/70	Bilateral, fishnet/ chicken wire hyperpigmentation pattern, 4-5DD
Multifocal Pattern Dystrophy	4th-6th decades	20/20-20/40	Bilateral, multiple yellow fleck-like lesions
Fundus Pulverulentus	4th-5th decades	20/20-20/40	Bilateral, coarse macular pigment mottling

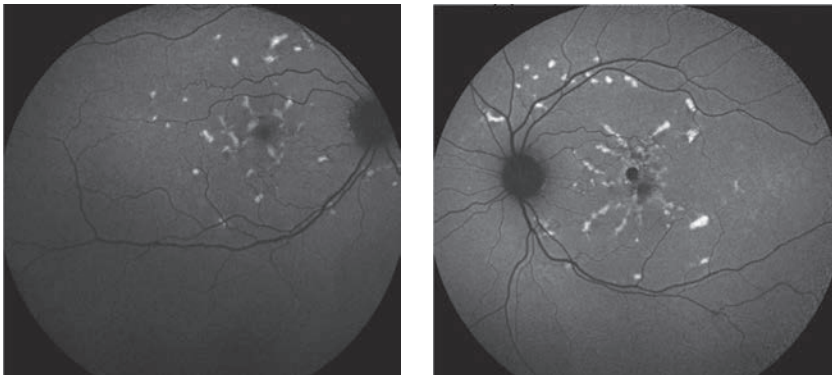

Fig. 4. Fundus autofluorescence images of the right and left eyes, respectively. The autofluorescence imaging revealed scattered hyperfluorescent lesions in the posterior pole and hypofluorescence corresponding to the macular RPE disruption.

flecks are scattered throughout the posterior pole and, in some cases, extend beyond the vascular arcades (*Figure 5*).[3,5,11]

Although multifocal pattern dystrophy will typically present with minimal to no visual effect and visual acuity from 20/20 to 20/40, a small area of central metamorphopsia may be seen with amsler grid testing, although this is more common in non-multifocal pattern dystrophy dystrophies.[3-7,15]

Differentials

Fundus flavimaculatus, Stargardt's disease and a basal laminar drusen variant are three differentials to consider when diagnosing multifocal pattern dystrophy. Fundus flavimaculatus and Stargardt's disease are arguably the most important differentials due to the similarity in retinal presentation.

Fundus flavimaculatus and Stargardt's disease are variants of the same pathology, inherited autosomal recessively through the ABCA4 gene on chromosome 1.[3,16,18,19] Both are characterized by irregular-shaped, triradiate, yellow, fleck-like lesions of variable size and shape within the posterior pole and concentrate around the retinal vascular arcades.[3,18]

While the two conditions are quite similar in terms of etiology and presentation, two important differences exist:

• ***Onset.*** Stargardt's disease typically develops early in life, while fundus flavimaculatus typically develops later in early to mid-adulthood, making the latter a more important differential as it will correlate more closely with age of onset for multifocal pattern dystrophy.[18]

• ***Macular involvement.*** In Stargardt's disease, the macula, paramacular region or both can have a central "beaten bronze" appearance.[18] Patients afflicted with Stargardt's disease will present with retinal abnormalities and experience vision loss much earlier in life, typically in the first or second decades.[18] The vision loss associated with Stargardt's disease is typically in the range of 20/100 to 20/200, or worse.[16]

Vision loss also occurs in fundus flavimaculatus, but occurs later in life and typically remains mild; it can decrease to 20/200, where it will typically stabilize.[15]

Stargardt's and fundus flavimaculatus differ from multifocal pattern dystrophy in key ways:

• ***Onset.*** While Stargardt's disease typically presents in the first or second decades, multifocal pattern dystrophy presents in the fourth to sixth decades.

• ***Effect on vision.*** Patients with multifocal pattern dystrophy typically maintain normal to mild reduction in acuity, whereas patients with Stargardt's disease and fundus flavimaculatus see more severe visual impairment.

• ***Fluorescein angiography.*** The diagnostic feature of Stargardt's disease and fundus flavimaculatus is the dark choroid on FA due to the lipfuscin storage in the RPE cells.[15,17,18,20,21] This does not happen in multifocal pattern dystrophy. Rather, a normal choroidal flush is present on FA (*Figure 5*).

The second differential to consider is a variant of basal laminar drusen. The retinal presentation of basal laminar drusen was first described in 1977 as small, raised, subretinal yellow lesions, and then further qualified in 1985 as focal areas of thickening in the RPE basement membrane.[22-24] Around this same time, researchers qualified basal laminar drusen as cellular aggregations.[22,25] These subretinal lesions can be widespread in the retina and mimic multifocal pattern dystrophy (*Figures 6a and 6b*).[22]

The best way to differentiate between multifocal pattern dystrophy and basal laminar drusen is through OCT, which will allows the clinician to qualify the type of deep retinal lesion by comparing images (*Figures 6a and 6b*).

Visual acuity also correlates between the two conditions, as basal laminar drusen may have minimal to no effect on vision; however, basal laminar drusen are also associated with pseudovitelliform macular detachments, which can result in subretinal fluid and mimic choroidal neovascular membranes.[22,24] Basal laminar drusen formation can

subsequently result in a progressive reduction in vision.[22] Progressive vision loss is rarely seen in multifocal pattern dystrophy; choroidal neovascular membranes rarely occur.[5,17]

Treatment and Management

The treatment and management of multifocal pattern dystrophy centers around its etiology, visual effect and role as a risk factor for the development of choroidal neovascularization. In the rare case the patient develops choroidal neovascularization, the standard of care is a referral to a retina specialist for consideration of anti-vascular endothelial growth factor (VEGF) therapy.

The key to managing the genetic component of the condition is patient education, which may entail a referral for genetic counseling. Through education, the patient and their family will be aware of the condition, its progression and prognosis.

Managing the visual effect of multifocal pattern dystrophy is as important as genetic counseling. Most cases of multifocal pattern dystrophy will retain normal to mildly reduced vision, in the 20/20 to 20/40 range; however, in rare instances vision loss can be more severe.[3-7,15] When vision is not 20/20, it is important to manage its effect on the patient's life. The clinician should address the patient's ability to read a newspaper or book; read aisle signs at the grocery store; complete basic household repairs; and participate in hobbies such as playing cards, building models, etc.

The effect of vision loss on the patient's life is best managed through low vision rehabilitation. Patients with multifocal pattern dystrophy can achieve the acuity needed for reading a newspaper or book and distance spotting through low magnification.

The diagnostic test of choice is fluorescein angiography, which shows normal choroidal flush in multifocal pattern dystrophy and the pathognomonic dark choroid in fundus flavimaculatus and Stargardt's disease.

For reading, patients will likely benefit from single-vision reading glasses and may require monocular occlusion, depending on the add, acuity discrepancy and preferred retinal locus.

Monocular telescopes can be used for distance spotting, whether in the grocery store or walking down the street. For those patients who want to be able to take care of household repairs or continue with hobbies, such as building models, optivisors can improve visual performance. Optivisors can be used to provide an intermediate add while also providing lighting; a 10D loupe can increase magnification when needed. For close work coupled with intermediate vision, a pair of bifocal glasses can be used with an add in the distance portion for intermediate vision and an add in the bifocal portion for close work.

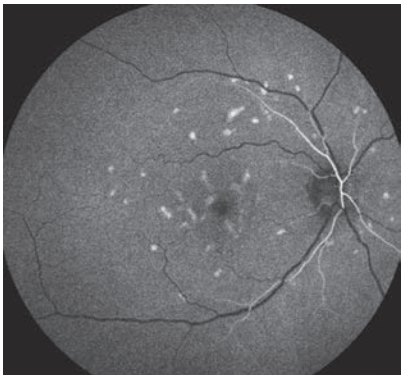

Fig. 5. Fluorescein angiography showing normal choroidal flush at 10 seconds during the early arteriole phase of the FA.

In addition to using conventional optical tints, which provide selective light filtering, task lighting can enhance contrast and reduce glare. Tinting is most successful with blue blocker tints (yellow, orange, amber), which reduce the glare experienced by patients due to the scattering of light on surfaces and by the ocular media.[26-29] Tints can also improve vision quality and contrast sensitivity by decreasing light scattering and veiling glare to improve the retinal image.[26-31]

Conclusion

While genotypically similar to other pattern dystrophies, MPD is phenotypically unique with a close resemblance, funduscopically, to fundus flavimaculatus and Stargardt's disease. The test of choice to aid in diagnosis is fluorescein angiography, which shows normal choroidal flush in multifocal pattern dystrophy and the pathognomonic dark choroid in fundus flavimaculatus and Stargardt's disease. This delineation is key because of the better visual prognosis associated with multifocal pattern dystrophy.

A better prognosis is also found with multifocal pattern dystrophy when compared with basal laminar drusen. The test of choice in distinguishing these two conditions is OCT, because it allows for the identification of the different structures of the deposits and lesions.

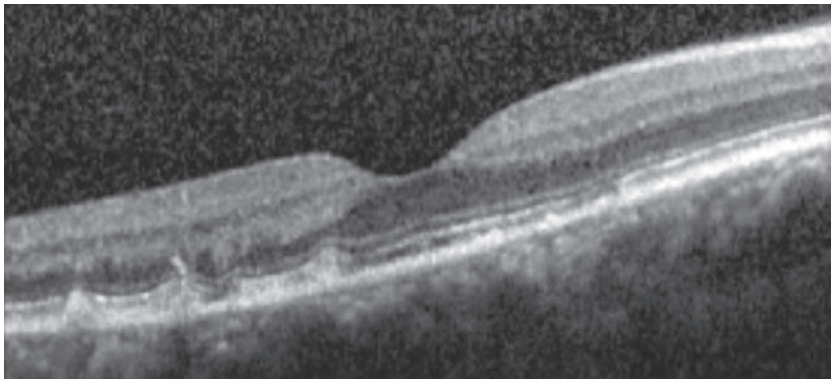

Fig. 6a. OCT image of our patient shows lesions at the level of outer photoreceptors and anterior retinal pigment epithelium.

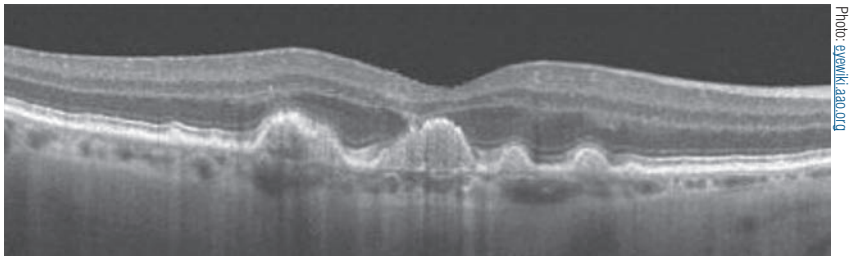

Photo: eyewiki.aao.org

Fig. 6b. Optical coherence tomography of basal laminar drusen.

This differentiation becomes important because patients with basal laminar drusen are at an increased risk of developing a pseudovitelliform macular detachment, which can cause subretinal fluid that mimics choroidal neovascularization; in patients with multifocal pattern dystrophy, it is rare to have progressive vision loss or development of choroidal neovascularization.

Despite the better visual prognosis for multifocal pattern dystrophy over the two leading differentials, there is still the possibility of mild vision loss, since there is no current treatment for the condition. Management of the dystrophy involves patient education, referral to a retina specialist and treatment of a choroidal neovascular membrane, in the rare instance that it occurs. In the event of impaired vision, low vision rehabilitation to aid in activities of daily living is also critically important. ■

Dr. Hamilton, a residency-trained optometrist, is the director of Inpatient Low Vision Optometry and director of the Low Vision Optometry Externship Program at the Connecticut VA Healthcare System, West Haven.

Dr. Burke is a residency-trained low vision optometrist at the Connecticut VA Healthcare System.

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What a Year It's Been

Optometrists have been rolling with the punches all year.

By John Rumpakis, OD, MBA, Clinical Coding Editor

The year 2015 may go down as one of the most tumultuous in terms of health care reform changes. Optometrists have found themselves feeling a bit like Smokey the Bear, putting out fires and trying to maintain control. Let's look back at a few of the issues that have made the biggest impact.

Winter

In January, we were once again dealing with Medicare's flawed sustainable growth rate (SGR) formula and a 21% decrease in reimbursements. Also in January, the Centers for Medicare and Medicaid Services (CMS) released its goal of moving from a traditional fee-for-service system based on volume to a system based on quality or value through alternative payment models such as ACOs or bundled payment arrangements—with the hope of obtaining 85% of all payments by 2016 and 90% by 2018.

Spring

In April, the Supreme Court ruled that health care providers cannot sue to increase Medicaid reimbursements. Specifically, it said "neither the Constitution nor federal law authorizes doctors and other healthcare providers to go to court to enforce the law's directive that the reimbursement rates set by states be 'sufficient to enlist enough providers so that care and services are available' to Medicaid recipients just as they are to the general population."

April also brought us a fix for the flawed SGR, and the bill blocked a 21% cut in payments due to take effect that month. It also provided financial incentives for physicians to bill Medicare patients for their overall care, not individual office visits—again, a move to value not volume.

Summer

With the summer heat of July and August came legislation tying future payments to quality outcomes, as well as studies showing that consumer health care spending is accelerating faster than the past decade due to the Affordable Care Act (ACA) and an improving economy. Health insurance companies also began filing for premium increases across the country, with some as high as 58% year over year. We also learned that as individual income rose, fewer were actually purchasing insurance; many were more willing to pay the tax penalty.

The summer was also merger mania. Aetna merged with Humana and Anthem agreed to buy Cigna, effectively reduced the big Five to the big Three.

Fall

Of course, October brought ICD-10, which came out with more of a whimper than a bang. Studies were also released indicating that health insurance deductibles are growing six times faster than wages. IBM and CVS partnered to provide automated solutions for chronic disease care delivery, and CMS announced a $585 million grant to develop a system to provide higher quality outcomes for less cost. More recently, some high profile insurance carriers such as Highmark and UnitedHealthcare have suggested they may not offer ACA plans due to increasing costs and lower profits.

Optometry Updates

Our industry was also reshaped by a number of significant events:

- Essilor's acquisition of Vision Source and PERC/IVA.
- VSP opening up VSP-branded clinics on company campuses and partnering with CVS.
- Luxottica partnering with Macy's to put 500 LensCrafters into prominent Macy's locations around the country.
- More private equity money coming into optometry and acquiring practices nationwide.

Health care reform will be a primary focus in 2016 as the employer mandate rolls out, Meaningful Use stage 3 becomes more refined and we begin to learn new acronyms such as VBM (value-based modifier) and MIPS (merit incentive-based payment system). So as you ring in the New Year, don't forget that, in our dynamic environment, change is the rule, not the exception. In order to thrive, you must be aware of your market and nimble enough to change your paradigm. Next year's Coding Connection column will highlight the issues shaping our health care system and discuss how to safely implement changes into your practice. ■

Send questions and comments to ROcodingconnection @gmail.com.

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Taking the Bull by the Horns

In the case of a bacterial corneal ulcer, what oral action—if any—should be taken?

Edited by Joseph P. Shovlin, OD

Q I recently saw a patient with a significant *Pseudomonas* ulcer in which the edge had reached the limbus. The ulcer is 3.5mm by 4mm with at least 50% thinning. The corneal specialist added an oral antibiotic. Is this necessary, and what might the best option be drug-wise?

A "There is no data that specifically supports how much of an oral antibiotic reaches the ocular surface," Paul Karpecki, OD, of Koffler Vision Group, says. "That being said, there is also no data to say that it won't help the ocular surface." In the case of an aggressive *Pseudomonas* ulcer, oral antibiotics are worth considering despite their unknown distinct impact on the cornea. Besides providing added antibiotic coverage, many oral antibiotics also carry anti-inflammatory properties and, since a topical steroid would be contraindicated in this case, the oral medication could be used to help manage inflammation.

Certain corneal ulcer situations—such as ones carrying the potential for endophthalmitis, in the case of concurrent treatment for systemic diseases like gonorrhea, or if the infection spreads to the sclera—necessitate the use of oral antibiotics, Eric Donnenfeld, MD, of Ophthalmic Consultants of Long Island and Connecticut, says.

In this particular instance, "the progression of isolated infectious keratitis toward the limbus suggests that the infection has been undertreated or incorrectly treated and is associated with a poor prognosis," says James P. Dunn, MD, director of the uveitis unit at Wills Eye Hospital. Patients with this presentation often require enucleation or evisceration.[1] Thus, Dr. Dunn explains, it is critical to treat such infections aggressively using culture and sensitivity results as a guide; however, he suggests keeping in mind that *P. aeruginosa* in particular is extremely difficult to remove once it invades the avascular scleral lamellae. Debridement, scleral wall resection and patch grafting can be used in severe cases.[2] He adds that residual scleromalacia is typical.

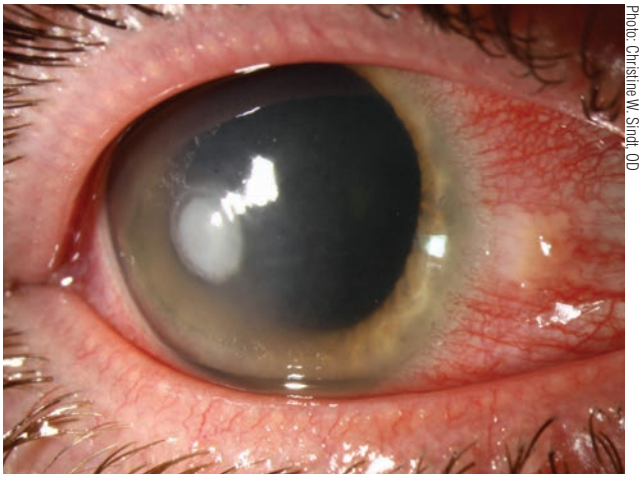

Photo: Christine W. Sindt, OD

***Pseudomonas* ulcers, like the one pictured here, must be aggressively treated to prevent permanent corneal damage.**

James Aquavella, MD, of the University of Rochester Medical Center, recommends the use of fortified topical antibiotics—specifically tobramycin 15mg/ml every hour around the clock alternating with cefazolin 50mg/ml. "If there is threatened perforation, systemic drugs may have a place, but even here I prefer a surgical approach with intracameral antibiotics," he adds.

In agreement, Dr. Dunn notes the use of combination intravenous ceftazidime and gentamicin in addition to topical antibiotics may be more effective than treatment with a single intravenous agent.[3] Subconjunctival aminoglycosides may also be beneficial.[4] Regarding oral antibiotics, he says, fluoroquinolones and imipenem do seem to achieve good intraocular penetration and may be as effective as intravenous antibiotics, though controlled studies are not forthcoming. ■

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The Opposite of "Red Disease"

A second opinion for a 65-year-old glaucoma suspect leads to a discussion of technology, imaging and, ultimately, glaucoma. **By James L. Fanelli, OD**

In October, a 65-year-old Caucasian female presented as a new patient. She had moved to the area about one year earlier, and her previous eye care provider, apparently, had followed her closely as a glaucoma suspect and left her with the impression that she was probably going to need medical intervention at some point in the future.

However, upon moving to the area, she felt her new provider had too quickly dismissed her concerns. She came to us for a second opinion.

Presentation

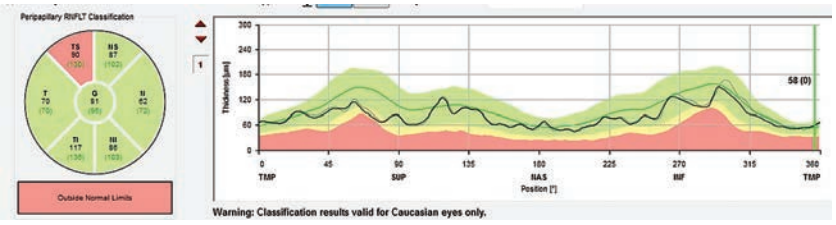
On initial presentation, entering visual acuities were 20/30 OD and 20/25- OS, and best-corrected acuities were 20/20- OU through mildly myopic astigmatic and presbyopic correction. Her pupils were round and reactive to light and accommodation, with no afferent pupillary defect. There was 0.5mm anisocoria that was stable in light and dark.

Her medical regimen included 81mg acetylsalicylic acid QD, and an unknown generic statin and an unknown antihypertensive medication, which she believed was lisinopril. She reported no known allergies to medications.

She had no family history of glaucoma, but reported her older sister has macular degeneration.

Evaluation

A slit lamp examination of her anterior segments was completely unremarkable. There was fine SPK noted centrally following instillation of anesthetic and fluorescein, but otherwise all was normal. Angles were judged to be wide open at the slit lamp. Applanation tensions were 20mm Hg OD and 19mm Hg OS at 10:45am. Central corneal thickness measurements were 531µm OD and 542µm OS. Threshold 24-2 visual fields were relatively clear, but with non-contiguous defects in the arcuate areas in both eyes, and reliability indices were moderate at best.



Note the TSNIT and global optic nerve sector analysis of a patient similar to the one discussed in this column. There is depression of the superior temporal RNFL of enough substance to be reflected in the corresponding global optic nerve sector as aberrant. Smaller RNFL defects would be seen on the RNFL TSNIT scan, but might not show up on the sector analysis. In that case, the entire sector analysis would be erroneously flagged as normal.

Through dilated pupils, her crystalline lenses were characterized by mild anterior cortical and nuclear cataracts, with vacuoles in both eyes. There were bilateral posterior vitreous separations.

Stereoscopic evaluation of her optic nerves demonstrated average-sized discs with a cup-to-disc ratio of 0.55 x 0.70 OD and 0.55 x 0.65 OS. The superior temporal neuroretinal rim of each disc was somewhat thinned. There appeared to be an NFL wedge defect in the superior temporal region of the RNFL in the right eye extending to the disc. The retinal vasculature was characterized by mild arteriolar sclerotic retinopathy in both eyes. Both maculae were characterized by fine RPE mottling, with no evidence of subretinal neovascularization. There was a small ERM perifoveally in her left eye. Her peripheral retinal evaluations were unremarkable in both eyes.

Multimodal 30 degree and 55 degree imaging of both posterior poles was obtained which readily showed the ERM in her left eye, as well as a well-defined RNFL wedge defect superior temporally in her right eye.

OCT imaging was also obtained for both eyes. The TSNIT graph for her right eye demonstrated a depression in the area of the wedge defect, but was otherwise normal, and that of the left eye was essentially normal in the double hump appearance. Global indices of the

optic nerve and RNFL scans were normal in both eyes, as were the macular thickness maps centered on the ETDRS grid.

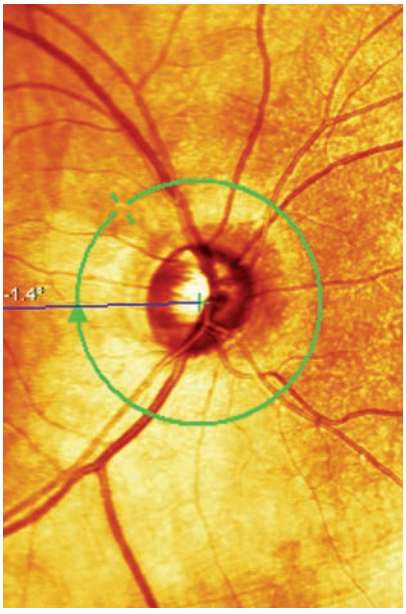
Discussion

After the initial examination, I discussed with the patient my concerns that she did, in fact, have glaucoma in the right eye—primarily manifest as the NFL defect extending to the optic nerve—and that she was a suspect in the left eye. She was rather articulate in her questioning as to why, specifically, I felt she had glaucoma and more so as to why I felt the best course of action would be medical intervention for her right eye.

Unless there are compelling reasons to do so, I prefer not to medicate a new patient who has glaucoma at the first visit. This mostly has to do with fostering a feel for diurnal variations and developing a stronger doctor/patient relationship.

Accordingly, at the completion of the initial examination, I asked to see her back in approximately a month to obtain several other measurements, in particular a HEP FDF visual field, HRT-3 optic nerve imaging and another IOP reading.

During the discussion, the patient posed an awkward question about the clinical capabilities of the clinician who, she felt, dismissed her situation. As I have mentioned before in this column, it serves no purpose to disparage another practitioner, especially when the encounter is painted through the articulation of the patient. In situations like these, I simply fall back on the fact that each clinician is different, and while we may be looking at the same disease, we look at it through different experiences and clinical intuitions.



This image shows a wedge defect in the RNFL, a similar condition to that of the patient in this case.

Follow-up

When the patient returned for follow-up, her IOPs were 22mm Hg OD and 20mm Hg OS. FDF field testing did reveal an arcuate defect inferiorly in her right eye, consistent with the RNFL defect, and HRT-3 imaging correlated well with the clinical appearance of the optic nerve. I had also obtained a copy of her medical records at the patient's request.

In reviewing her previous records, and in particular the OCT, I saw what may have been a deciding piece of information for the previous clinician in calling the patient normal—a lack of "red disease." All indices on the earlier OCT scan were flagged as "green" which is what our eyes tend to gravitate toward when we evaluate optic nerve and retinal scans.

Red Disease

In earlier columns, I discussed the issue of "red disease;" that is, when a doctor looks at a particular instrument's normative database for an assessment of whether or not disease is present. The unfortunate impact of having normative databases is in the quick clinical assessment of the patient when either red or green shows up, implying either the presence or absence of disease.

It is incredibly important to realize that normative databases are simply statistical representations of the patient population used to gather the normative data. In other words, the normative database looks at a typical bell curve distribution of a particular index, concentrating on the center of the bell curve. As we all know, there are always outliers that are, frankly, normal. Just because a database may flag a particular index as red does not necessarily mean disease is present. Conversely, if a normative database flags a study as green, that does not mean no disease is present.

In looking at the earlier OCT, as well as the current OCT, the macular thickness maps were all "normal" (read as green). The RNFL scan was also green.

Perhaps the previous provider quickly glanced at the scans and saw all green and quickly determined that the patient was fine. I don't know, but that is certainly a possibility.

We noted an RNFL defect, but a normal appearance in the global RNFL scan data and macular scans, similar to the scan provided on page 77. That pertains to a significant enough wedge defect to skew the data of the entire sector. In this patient's case, the wedge defect was small and not of enough substance to affect the whole sector of the RNFL scan.

And therein lies the main crux of this case: statistics do not a disease make, nor do they absolve one from disease. ■

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When the Stars Align

Can connecting the dots between symptoms and imaging explain this patient's vision loss?

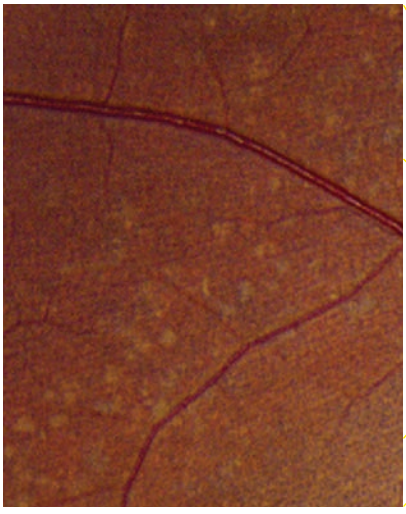
By Mark T. Dunbar, OD, and Alison Bozung, OD

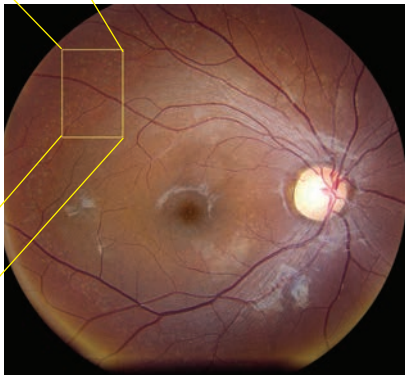
An eight-year-old Asian male was referred to our clinic for unexplained vision loss in both eyes. Starting about five months earlier, his parents stated, he began sitting closer to the board at school and pulling books in closer to read. They noted he was also having some trouble differentiating colors.

His overall health was unremarkable. He was not taking any medications, nor did he have any medication allergies. His family medical and ocular history was non-contributory.

Upon examination, his visual acuity measured 20/100 OU with no improvement after mild hyperopic refractive error was trialed. Extraocular motilities showed full range of motion. His confrontation fields were full to careful finger counting in both eyes. Pupils were round and reactive to light with no afferent defect. He was able to see the test plate on color vision, but scored 0/10 in each eye.

Slit-lamp examination of the anterior segment yielded nothing remarkable. Posterior segment examination revealed optic nerves with moderate, physiologic cupping and mild temporal pallor, in his right eye more than in his left. The fundus photos depict what was observed clinically (*Figures 1a, 1b and 1c*). Fundus autofluorescence (FAF) (*Figures 2a and 2b*) and intravenous fluorescein angiography (IVFA) (*Figure 3*) were performed and is available for review.





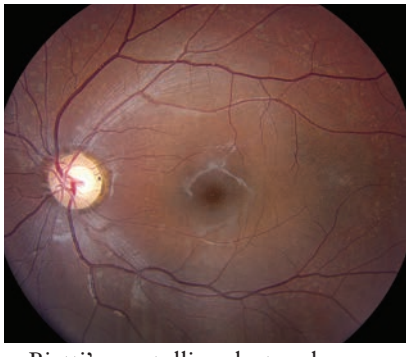


Fig. 1a-c. What do the fundus images below tell you about our eight-year-old Asian male patient? Take special note of the magnified area to the left.

Take the Quiz

1. What do the discolored lesions in both eyes' fundus photos represent, and where are they located?
a. Lack of melanin granules; within the RPE.
b. Drusen; under Bruch's membrane.
c. Lipofuscin; between RPE and photoreceptors.
d. Lipofuscin; within the RPE.

2. On FAF, how would one describe the pattern seen?
a. Foveal hypoautofluorescence with punctate hyperautofluorescent lesions.
b. Scattered window defects with normal autofluorescence.
c. Unremarkable.
d. Normal macular autofluorescence with peripheral lesions.

3. What is the likely diagnosis?
a. Progressive cone dystrophy.
b. Stargardt's macular dystrophy.
c. Bietti's crystalline dystrophy.
d. North Carolina macular dystrophy.

4. Which two features does the IVFA for our patient's condition typically manifest?
a. Late leakage near fovea from leaky vessels.
b. Silent choroid from RPE blockage defect.
c. Early punctate hypofluorescence surrounding arcades
d. Late staining of retinal flecks.

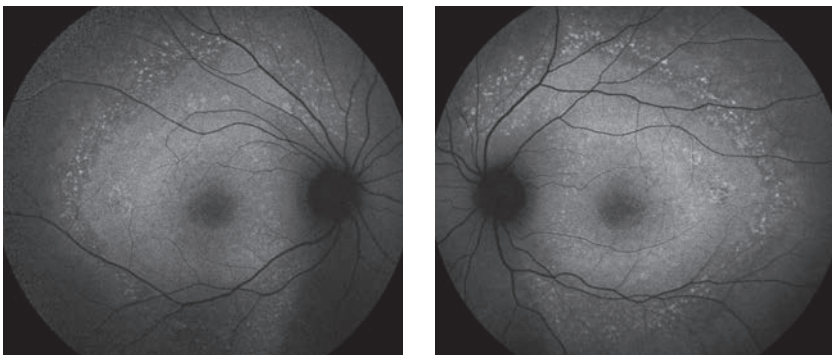
Discussion

Our patient had reduced central vision, a silent choroid on fluorescein angiography, and an early bulls-eye pattern on fundus autofluorescence. Though he lacked the stereotypical "pisciform" or "triradiate" retinal flecks, his diagnosis was most consistent with autosomal recessive Stargardt's macular dystrophy (or Stargardt's disease).

Stargardt's disease is the most commonly inherited childhood autosomal recessive retinal dystrophy. Less commonly, some Stargardt's disease mutations have an autosomal dominant transmission.[1] The genes typically affected are ABCA4, ELOVL4 or PROM1. This condition affects the retinal pigment epithelium (RPE) and photoreceptor layer and typically has an onset in childhood or young adulthood. Some patients can develop symptoms of visual acuity loss as late as the fourth or fifth decade of life.

Diagnosis of Stargardt's disease can be straightforward in the event that the patient presents in such a fashion. However, there is variability in the shape/formation or even presence of the retinal flecks, the age of onset, the decline in visual acuity, and the OCT findings—all of which can complicate the diagnosis.

Clinical testing for this condition can be extremely helpful, especially because vision can be reduced before any retinal signs are present. Recent research suggests one of the earliest signs is an abnormally thickened external limiting membrane (ELM) on OCT.[2] The ELM is located between the photoreceptor cell bodies (outer nuclear layer) and the photoreceptor inner segments, and its thickening indicates early photoreceptor damage. As the disease progresses, OCT reveals a clear dropout of the outer retinal layers, as seen in our patient.



Figs. 2a and 2b. Can you use these FAF images to explain the patient's symptoms?

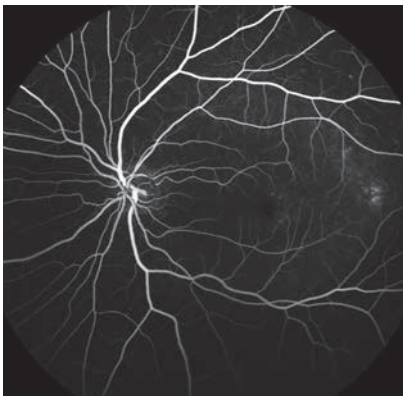


Fig. 3. This is the venous phase of the fluorescein angiogram. What does it reveal about our patient's condition?

The FAF lights up in the areas where there is loss of RPE and overlying photoreceptors on OCT. The areas of hyperautofluorescence relate to areas where there is still functioning, but defective, RPE. The overall area of abnormal FAF has been found most specifically to relate to IS/OS junction loss that may extends beyond the damaged retina visible on direct observation.[3]

IVFA may not be necessary for diagnosis, but if performed, shows a key feature: the "silent choroid." The absence of choroidal flush is not actually a problem with the choroid. The hypofluorescence seen is due to signal blocking from high-grade lipofuscin accumulation within the RPE lying above. Foveal patches of hyperfluorescence are window defects with increased signal transmission through defective RPE. The flecks themselves present as early blockage and late, non-progressive staining.[4] Frequently, a bulls-eye pattern will be present as well.

Unfortunately, there is no current treatment for Stargardt's disease. There are, however, ongoing studies in early phases for deuterium enrichment of vitamin A to slow lipofuscin formation and storage, subretinal transplantation of stem cell-derived RPE cells and gene therapy.

Handling progressive vision loss in a child is a difficult topic to discuss, so both our patient and his parents were carefully counseled about his condition. We discussed future prognosis and assistance programs for school. He is not currently enrolled in any studies, but may be a candidate in the future at our facility. ■

This case was written by Alison Bozung, OD, optometric resident at Bascom Palmer Eye Institute.

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Sowka Down Under... Again

Don't be so quick to discount so-called "natural" therapies.

By Joseph W. Sowka, OD

Readers familiar with this column have, at times, seen me comment upon ocular therapeutic issues that I have gleaned while travelling abroad. I recently returned from lecturing at the Tasmania Lifestyle Congress in Hobart, Tasmania off the coast of Australia. This is one of the finest conferences that I have ever attended, and it was a pleasure and an honor to be invited to present.

My epiphany for this column did not come from the actual conference, but rather from touring Australia. At one wildlife park, I had the chance to hold a koala. It was a wonderful experience, but when I put this hairy-bowling-ball-with-claws down, I noticed that I was covered with the scent of eucalyptus. Koalas are herbivorous and most of their diet consists of eucalypt leaves. The oils seep from their pores and coat their fur, giving koalas that distinct eucalyptus scent.

I found myself reflecting upon how many natural compounds have found their way into modern medicine. The eucalyptus oil used by humans comes from *Eucalyptus*, a diverse genus of flowering trees and shrubs. The oil is steam-distilled from the plant's leaves and has many uses, such as an industrial solvent, antiseptic, decongestant to open clogged nasal passages, and is used in some foods such as cough drops.

There are numerous home remedies, potions, lotions and alternative therapies that are often discounted by modern medicine. However, some natural products do indeed have medicinal properties. Considering where some commonly used ophthalmic medications have arisen from, it would not be wise to totally discount natural therapies.

The author and his wife, Lori Vollmer, OD, making friends in Australia.

From a Shrubbery

Pilocarpine is a parasympathomimetic medication that works topically to lower intraocular pressure (IOP) in glaucoma and orally to increase saliva production in patients with xerostomia. In the eye, it causes constriction of the iris sphincter muscle, resulting in pupillary miosis. In angle-closure patients, the miotic effect can move the pupil off the mid-dilated state where pupil block is most pronounced, as well as physically stretch the iris, pulling it away from the pigmented trabecular meshwork. In open-angle situations, the iris contraction puts stress on the scleral spur, temporarily altering the structure of the trabecular meshwork, allowing aqueous to flow better through this drainage pathway.

Few know our oldest topical glaucoma treatment, topical pilocarpine, is obtained from the leaves of tropical South American shrubs from the genus *Pilocarpus*.

Commercial production is derived entirely from the leaves of *Pilocarpus microphyllus*. Topically applied pilocarpine is extracted from powdered leaf material in a multi-step process involving acidification, ammonification and chloroform extraction.

A Good Nightshade

Topically applied atropine is an anticholinergic medication that blocks the neurotransmitter acetylcholine at the muscarinic receptors of the neuromuscular junction. Atropine is in the belladonna alkaloid family and has been used for hundreds of years to cosmetically enlarge the pupils. Atropine has numerous systemic uses. It can be used to treat symptomatic bradycardia as well as reduce salivation and sweating. By blocking the action of acetylcholine, atropine can be used as a treatment for poisoning by organophosphate insecticides and nerve gases.

In the eye, topically applied atropine will induce mydriasis and cycloplegia by blocking acetylcholine at the nerve junctions in the pupil sphincter and ciliary body, respectively. Due to atropine's long duration of action, it is not a preferred mydriatic agent, but its deep penetration and full blocking effects makes it an extremely useful cycloplegic agent in uveitis management.

Visual blur induced by atropine makes it a useful alternative to patching in amblyopia therapy

While we are accustomed to atropine in a bottle as a topical solution, it occurs naturally in a number of plants of the nightshade family, including deadly nightshade, Jimson weed and mandrake. I recall a patient who accidentally cyclopleged herself for two weeks by handling the wrong plant at a flower show.

The cassowary may look like a rough customer, but she's actually quite harmless. The same is true of some ophthalmic medications.

Power Plants

Mirtogenol (LifeExtension) is a combination formula of a bilberry extract called Mirtoselect and Pycnogenol French maritime pine bark extract. It is a non-prescription supplement promoted to both increase ocular blood flow as well as reduce IOP in patients with glaucoma and ocular hypertension.

In a blood flow study, 38 subjects with ocular hypertension were either given Mirtogenol (20 subjects) or were not treated (18 subjects).[1] The visual acuity, IOP and ocular blood flow were measured at two, three and six months. Nineteen of the 20 patients taking Mirtogenol had a decreased IOP after three months. Based upon color doppler imaging, ocular blood flow in the central retinal, ophthalmic and posterior ciliary arteries significantly ($p<0.05$) increased both in the systolic and diastolic components, as compared with both the baseline and control groups.[1]

In an IOP study of Mirtogenol's effects, 79 patients with ocular hypertension were randomly assigned to three groups to receive either Mirtogenol, latanoprost or both in combination. Mirtogenol lowered IOP in patients almost as effectively as latanoprost, though it took much longer (24 weeks vs. four weeks) to do so. Mirtogenol alone lowered IOP from baseline 38.1mm Hg to 29mm Hg after 16 weeks, whereas latanoprost alone lowered IOP from baseline 37.7mm Hg to 27.2mm Hg within four weeks. Mirtogenol potentiated the IOP lowering effect of latanoprost; the combination of latanoprost and Mirtogenol was more effective for lowering IOP than either alone. The combination of Mirtogenol/latanoprost lowered IOP from 38mm Hg to 27.3mm Hg after four weeks, and further decreased IOP to 24.2mm Hg after six weeks.[2]

Mirtogenol has shown promise in these small studies, but since it appears no further research has been conducted on this product, I do not actively recommend this product. However, I will discuss the known research with patients who ask about natural and over-the-counter adjuncts for glaucoma treatment.

While it is easy to dismiss products made from barks, plants, extracts, seeds and the like, it is important to remember that some of these so-called natural products can have valuable medicinal properties. Some of these remedies have even undergone professional compounding and rigorous scientific scrutiny and have been approved and classified as medicines. Really, the only difference between a home potion or remedy and a medicine is approval by the FDA.

Which Has Killed More People: Cassowaries or Chloramphenicol?

No column based upon travels to Australia is complete without my usual chloramphenicol diatribe. Chloramphenicol has a broad spectrum of both gram-positive and gram-negative antibacterial activity.[3-7] Since the inception of systemic chloramphenicol, there has been a reported association with several blood dyscrasias, the most notable of which is aplastic anemia. While topical chloramphenicol use is widespread throughout the world, this possible association with aplastic anemia has curtailed use of the drug in the United States. Practitioners have long known that this topical medication has resulted in patient deaths and fear the lethal potential of topical chloramphenicol.[3-7] But how deadly is this drug?

Stories of the deadly potential from topical chloramphenicol are similar to those I have always heard, and believed, about the 'world's most dangerous bird,' the Australian cassowary, a large emu-like flightless bird with strong claws. Every time I have been in Australia, I have heard about this bird's ability (even proclivity) to kill humans. However, during my most recent trip, a biologist actually gave me the facts about the "world's most dangerous bird." Apparently, during the approximately 200 years that the species has been recognized, only two people have been killed, presumably in defense of a nest or young (W61.92. is the ICD-10 code for this, by the way). This actually approximates the number of people who have been killed by topical chloramphenicol. The first case of aplastic anemia associated with topical use was reported in the 1960s.[8] In 1980 and 1982, the first two cases of fatal aplastic anemia that were thought to be associated with topical chloramphenicol use were reported.[9,10] While chloramphenicol has not been used in the United States for many years due to perceived risks of aplastic anemia, the Australians have no such worries; topical chloramphenicol is over-the-counter in Australia. In fact, I walked into a pharmacy and bought myself a bottle to take home. ■

Like the cassowary, chloramphenicol is behind only two known deaths.

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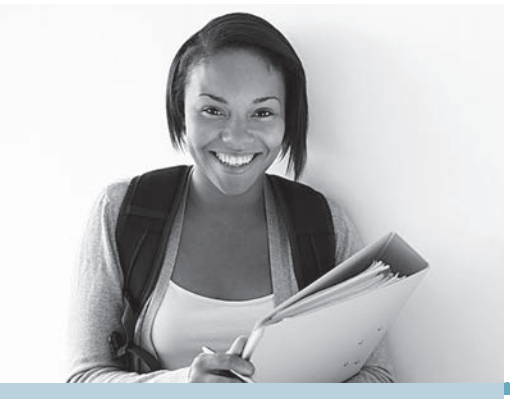

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Edited by Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

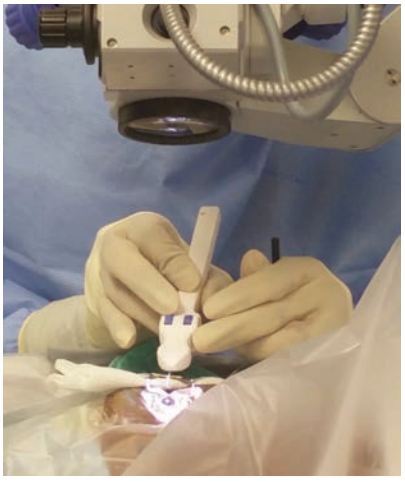
Expanding Outflow with MIGS

A new procedure, Visco360, can be a great stand-alone option, or it can be combined with cataract surgery. **By Constance Okeke, MD, MSCE**

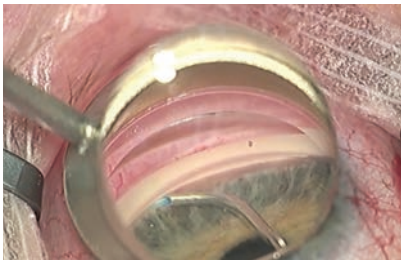
One of the newest options for minimally-invasive glaucoma surgery (MIGS) is Visco360 (Sight Science), which was released this year and provides a new approach to viscocanaloplasty. While research from the last 10 years shows that ab externo canaloplasty can effectively lower intraocular pressure (IOP) in open-angle glaucoma patients, the procedure can be technically challenging.[1] This new surgical option allows clinicians to perform viscocanaloplasty with an internal approach through the same small corneal incision used in cataract surgery, making it a much faster, less intrusive procedure that spares the conjunctiva and can lower IOP. It facilitates the delivery of small, controlled volumes of viscoelastic fluid in the space of Schlemm's canal behind the trabecular meshwork (TM) to improve outflow. This can separate compressed tissue planes where there could be adhesions, which dilates the canal and the more distal collector channels, improving aqueous outflow.

Surgical Technique

If combined with cataract surgery, Visco360 is performed first. The device is primed by instilling viscoelastic prior to the start of the procedure. A corneal incision with a blade is made temporally, and the anterior chamber (AC) is filled with viscoelastic. The device is advanced across the AC and, with the help of a gonioprism, the tip of the device



Dr. Okeke using the Visco360 device.



The advancement of a flexible injection tube past the TM into Schlemm's canal.

makes an initial incision through the TM to provide a track for entry into Schlemm's canal. Rotating a control wheel allows advancement of a flexible injection tube into Schlemm's canal for up to 180 degrees. The injection tube is then retracted and an infusion pump slowly releases the viscoelastic. After this is completed in one direction, the device is turned to repeat the process in the other direction for up to another 180 degrees. There may be a small egress of heme from Schlemm's canal with the initial incision though the TM, but it is minimal. Once completed, if a stand-alone procedure, the remaining viscoelastic is removed from the AC, and the corneal wound is hydrated with a watertight seal, typically without the need of any sutures. If combined, the surgeon proceeds with the cataract surgery.

Advantages and Disadvantages

Unlike other MIGS procedures, Visco360 tissue, does not require permanent placement of an implant and treats up to 360 degrees of the outflow system. It is indicated as a stand-alone procedure in phakic or pseudophakic patients and can be easily combined with cataract surgery. It has a disposable, single-use instrument and typically takes less than 10 minutes. There is minimal hyphema, and there is an already established procedure code, which improves its insurance coverage.

One major disadvantage is that it is an extremely new device, and the clinical trial results are sparse so far. Time will tell if this device will remain a viable MIGS option. ■

Dr. Okeke is an assistant professor of ophthalmology at Eastern Virginia Medical School and a glaucoma specialist and cataract surgeon at Virginia Eye Consultants in Norfolk, Va.

1. Lewis RA, von Wolff K, Tetz M, et al. Canaloplasty: three-year results of circumferential viscodilation and tensioning of Schlemm's canal using a microcatheter to treat open-angle glaucoma. J Cataract Refract Surg. 2009 May;35(5):814-24.

Practice Management

New EHR Software

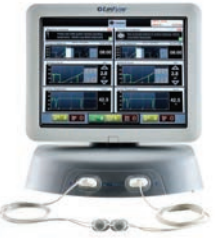
Optometrists can now choose from two new software offerings from ManagementPlus: Cloud and Revenue Cycle Management. ManagementPlus Cloud backs up your EHR system to secure, remote servers, minimizing IT costs and protecting patient data, the company says. It is also designed to centralize EHR and other applications, to make them easier to manage.

According to the company, Revenue Cycle Management simplifies the revenue stream with claims reporting and analysis, collection on past-due balances, and patient eligibility verification.

Visit www.managementplus.com.

Dry Eye Treatment

New MGD Treatment Pricing



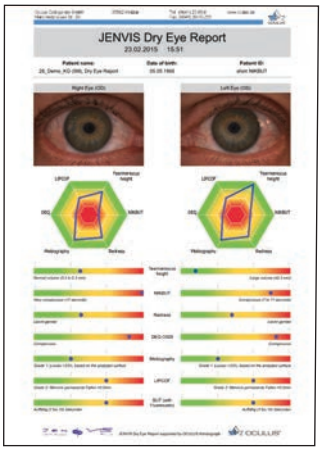
TearScience has announced a 50% price cut for single-use activators used in its Lipiflow device. LipiFlow applies heat to the inner eyelid and pressure to the outer eyelid during an in-office procedure, and has been shown safe and effective for the treatment of MGD, according to the company.

Visit www.tearscience.com.

Diagnostic Technology

Dry Eye Report

Optometrists now have a new way to show patients how dry eye is affecting their eyes.



The Jenvis Dry Eye Report, unique to the Oculus Keratograph, supports dry eye diagnosis, Oculus says. The Dry Eye Report summarizes all the data from a dry eye questionnaire, slit-lamp measurement of lid parallel conjunctival folds, meibography and measurements performed with the Oculus Keratograph 5M. The report gives patient-specific recommendations and explains abbreviations and technical terms to improve patient engagement, according to Oculus.

Visit www.oculus.de/us/products.

Electroretinography and VEP Vision Testing System

To help optometrists with early detection and patient management, Diopsys now offers the Argos electroretinography and VEP vision testing system as a tabletop version of its office-based visual electrophysiology.

The system features the new full-field electroretinography (ffERG) testing protocols, which use a handheld mini-Ganzfeld dome to stimulate retinal cells. The results indicate a patient's level of functional loss, allowing optometrists to better manage the patient and their potential for IOL procedures, according to the company.

Visit www.diopsys.com/diopsysargos.

IS-5500 Instrument Stand

Practioners looking for a new instrument stand can now consider Topcon's IS-5500, which combines new functions—like its EZ-Lock braking mechanism—with traditional Topcon quality and durability.

The IS-5500 features:

- White LED overhead lamp.
- More compact design.
- Illuminated liquid-proof membrane control panel for full control of the stand, chair and room light functions.

Visit www.topconmedical.com.

Contact Lenses

Monthly Silicone Hydrogels for Presbyopia

Bausch + Lomb says it will offer a multifocal option in its Ultra contact lens line in 2016.

The monthly replacement lens will combine the Ultra lens' high wettability with three-zone progressive design found in Bausch + Lomb's daily disposable multifocals.

The lens uses polyvinylpyrrolidone, allowing it the highest Dk/t (163) and lowest modulus (70) compared to the leading silicone hydrogel lens, Bausch + Lomb says. The lens also has aspheric optics for exceptional vision even in low light conditions, according to the company.

Visit www.bausch.com/our-products. ■

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Paul Karpecki, OD

Bermuda
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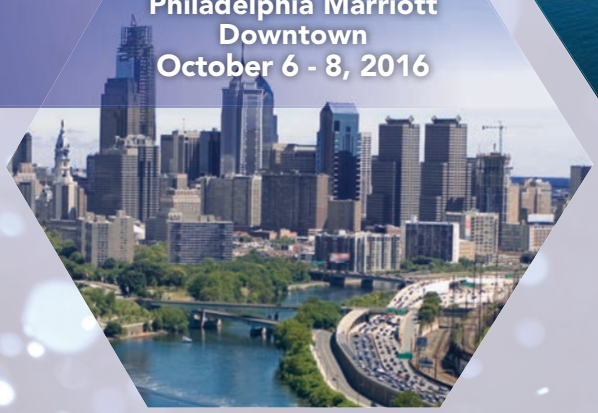
June 9 - 12, 2016

Philadelphia
Philadelphia Marriott Downtown
October 6 - 8, 2016



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December 2015

■ **26-Jan. 2.** *Seminar in Paradise*. MSC Cruise Line's Divina, Western Caribbean Cruise, Departs Miami, FL. Hosted by: Dr. Travel Seminars/The New Jersey Society of Optometric Physicians. CE hours: 16. To register, email Robert Pascal at info@DrTravel.com, call (800) 436-1028 or go to DrTravel.com.

January 2016

■ **9.** *Glaucoma Symposium 2016*. Willows Lodge, Woodinville, WA. Hosted by: Pacific University College of Optometry. Key faculty: Howard Barnebey, Murray Fingeret. CE hours: 7. To register, email Martina Fredericks at frederim@pacificu.edu, call (503) 352-2207 or go to www.pacificu.edu.

■ **9.** *2016 Coding Update*. Embassy Suites Little Rock, Little Rock, AK. Hosted by: Arkansas Optometric Association. Key faculty: John McGreal. CE hours: 5. To register, email Vicki Farmer at aroa@arkansasoptometric.org, call (501) 661-7675 or go to arkansasoptometric.org.

■ **11.** *2016 Legislative Conference*. Capitol Plaza Hotel, Jefferson City, MO. Hosted by: Missouri Optometric Association. Key faculty: Alan Cleinman. CE hours: 3. To register, contact Lee Ann Barrett at moaed@moeyecare.org, call (573) 635-6151 or go to www.moeyecare.org.

■ **16-17.** *Gold Coast Educational Retreat*. Hyatt Regency Pier 66, Fort Lauderdale, FL. Hosted by: Broward County Optometric Association. Key faculty: Randall Thomas, Ron Melton, Tim Murray, Roger Prouty, Cory Collier, Joseph Sowka, Joseph Pizzimenti. CE hours: 17. To register, email Rachell Snell or Brandon Cornishat bcoa@browardeyes.org or go to www.browardeyes.org.

■ **16-18.** *Kraskin Invitational Skeffington Symposium on Vision (KISS)*. Hyatt Regency, Bethesda, MD. Hosted by: Optometric Extension Program Foundation (OEPF). CE hours: 19. To register, email Jeffrey Kraskin at jlkraskin@rcn.com, call (202) 363-4450 or go to www.skeffingtonsymposium.org.

■ **17.** *IOA Winter CE Series*. Westin Chicago North Shore, Wheeling, IL. Hosted by: Illinois Optometric Association. Key faculty: Len Messner. CE hours: 6. To register, email Charlene Marsh at ioabb@ioaweb.org or go to www.ioaweb.org.

■ **17-23.** *2016 Island Eyes Conference*. Sheraton Maui Resort, Lahaina, Maui, HI. Hosted by: Pacific University. Key faculty: Denise Goodwin, Nathan Lighthizer, Leo Skorin, Stanley Teplick, Samuel Kim. CE hours: 29. To register, email Jeanne Oliver at jeanne@pacificu.edu, call (503) 352-2740 or go to www.pacificu.edu/IslandEyes.

■ **24.** *IOA Winter CE Series*. Hyatt Regency O'Hare, Rosemont, IL. Hosted by: Illinois Optometric Association. Key faculty: Paul Karpecki. CE hours: 6. To register, email Charlene Marsh at ioabb@ioaweb.org or go to www.ioaweb.org.

■ **30.** *Georgia Optometric Association Super CE*. Georgia International Convention Center, College Park, GA. Hosted by: Georgia Optometric Association. CE hours: 8. To register, email Vanessa Grosso at VanessaGOA@aol.com, call (770) 961-9866 x-1 or go to http://hleachgoa.wix.com/goaeyes.

■ **31.** *VOA 1 Day CE Conference*. Richmond Marriott West, Glen Allen, VA. Hosted by: Virginia Optometric Association. CE hours: 4. To register, email Bo Keeney at Office@thevoa.org, call (804) 643-0309 or go to www.thevoa.org.

February 2016

■ **3-4.** *Michigan Optometric Association Winter Seminar*. Kellogg Hotel and Conference Center of Michigan State University, East Lansing, MI. Hosted by: Michigan Optometric Association. Key faculty: Steven Ferrucci, Marc Bloomenstein. CE hours: 12. To register, email Amy Root at amy@themoa.org, call (517) 482-0616 or go to www.themoa.org.

■ **8.** *IOP Winter CE*. The Grove Hotel, Boise, ID. Hosted by: Idaho Optometric Physicians. CE hours: 4. To register, email Randy Andregg at execdir@iopinc.org, call (208) 461-0001 or go to Idaho.aoa.org.

■ **12-14.** *Heart of America Contact Lens Society*. Sheraton Crown Center, Kansas City, MO. Hosted by: HOACLS. Key faculty: Paul Ajamian, Michael Chaglasian, Joseph Sowka, Valerie Kattouf, Jeffry Gerson. CE hours: 77 total, 15 per OD. To register, email Ron Fiegel at registration2@thehoalcls.org or go to www.hoacls.org.

■ **12-16.** *SkiVision*. Westin Snowmass Resort, Snowmass Village, CO. Hosted by: SkiVision, *Review of Optometry*. Key faculty: Murray Fingeret, John Flanagan, Ian Ben Gaddie, Jack Schaeffer, Jay Haynie, Kathy Dumbleton. CE hours: 20. To register, email Lois DiDomenico at ldidomenico@jobson.com, call (610) 492-1018 or go to www.skivision.com.

■ **13.** *OAL Mid-Winter CE Conference*. DoubleTree Hotel, Lafayette, LA. Hosted by: Optometry Association of Louisiana. CE hours: 8. To register, email Jim Sandefur at optla@bellsouth.net, call (318) 613-1392 or go to www.optla.org.

■ **13-20.** *Innovations in Eye Care*. Western Caribbean Cruise from Fort Lauderdale, FL. Hosted by: Dr. Travel Seminars, LLC. Key faculty: Robert Wooldridge. CE hours: 16. To register, email Robert Pascal at DrTravel@aol.com, call (800) 436-1028 or go to www.drtravel.com/optometristsSeminars.html.

■ **14.** *IOA Winter CE Series*. Marriott Bloomington-Normal Convention Center, Bloomington/Normal, IL. Hosted by: Illinois Optometric Association. Key faculty: Michael Chaglasian. CE hours: 6. To register, email Charlene Marsh at ioabb@ioaweb.org, call 217-525-8012 or go to www.ioaweb.org.

■ **14-24.** *AEA Cruises Canary Islands Optometric Cruise Seminar*. Aboard NCL Epic, Barcelona, Spain. Hosted by: AEA

Cruises. CE hours: 10. To register, email Marge McGrath at aeacruises@aol.com, call (888) 638-6009 or go to www.optometriccruiseseminars.com.

■ **19-21.** *32nd Annual Palm Beach Winter Seminar.* Hilton West Palm Beach, Florida. Hosted by: Palm Beach County Optometric Association. CE hours: 20+. To register, email PBWinterSeminar@gmail.com or go to www.pbcoa.org.

■ **20-27.** *AEA Cruises Eastern Caribbean Optometric Cruise Seminar.* Aboard NCL Escape, Miami, FL. Hosted by: AEA Cruises. CE hours: 10. To register, email Marge McGrath at aeacruises@aol.com, call (888) 638-6009 or go to www.optometriccruiseseminars.com.

■ **25-27**. *MOA Winter Educational Symposium.* Huntley Lodge, Big Sky, MT. Hosted by: Montana Optometric Association. Key faculty: Andrew Morgenstern, Maynard Pohl. CE hours: 13. To register, email Sue Weingartner at sweingartner@rmsmanagement.com, call (406) 443-1160 or go to www.mteyes.com.

■ **25-27.** *Third Party/Practice Management Seminar.* Embassy Suites, Portland Airport, Portland, OR. Hosted by: Oregon Optometric Physicians Association. Key faculty: John McGreal, Elizabeth Cottle, Steve Farebrother, Ronald Guerra, Shelly Sneed. CE hours: 15 total, 13 per OD. To register, email Lynne Olson at lynne@oregonoptometry.org, call (800) 922-2045 or go to www.oregonoptometry.org.

■ **28.** *OptoWest South Newport Beach.* Newport Beach Marriott Hotel and Spa, Newport Beach, CA. Hosted by: California Optometric Association. Key faculty: Leo Semes, Todd Severin. CE hours: 12 total, 6 per OD and 6 per staff member. To register, email Sarah Harbin at sharbin@coavision.org, call (916) 266-5022 or go to www.coavision.org.

■ **28.** *IOA Winter CE Series.* Tinley Park Convention Center, Tinley Park, IL. Hosted by: Illinois Optometric Association. Key faculty: Mark Dunbar. CE hours: 6 regular or TQ. To register, email Charlene Marsh at ioabb@ioaweb.org, call (217) 525-8012 or go to www.ioaweb.org.

■ **28-March 4.** *30th Annual Eye Ski Conference.* The Lodge at Mountain Village, Park City, UT. Hosted by: Timothy Kime and James Fanelli. Key faculty: Joe Pizzimenti, Alan Berman, Leonard Messner, James Fanelli. CE hours: 20. To register, email Timothy Kime at tandbkime@bex.net, call (419) 475-6181 or go to www.EyeSkiUtah.com. ■

To list your meeting, please send the details to:

Rebecca Hepp
Senior Associate Editor
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Diagnostic **Quiz**

Lids That Shake, Rattle and Roll

By Andrew S. Gurwood, OD

History

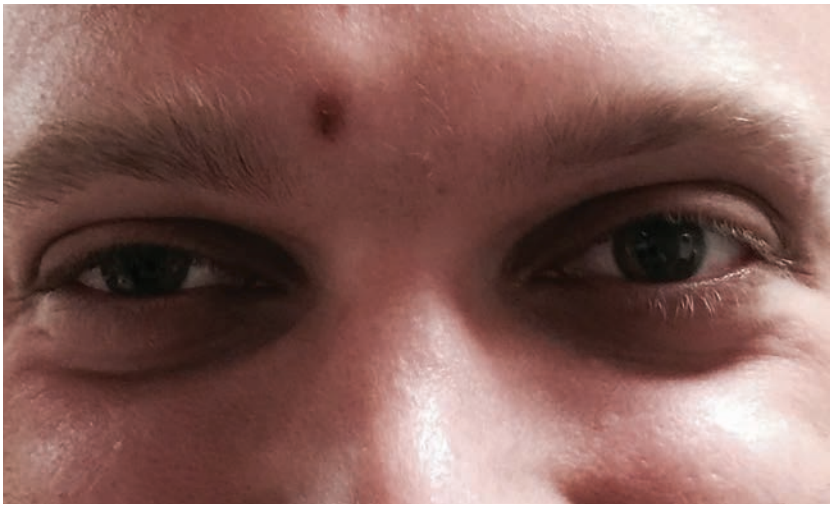
A 56-year-old white female presented for an urgent visit with a chief complaint of a twitching eyelid. She had been seen in the past for seasonal ocular allergy and mild dry eye syndrome for which she was medicating with Pataday (olopatadine, Alcon) and artificial tears, as needed. She had no systemic history and denied any exposure to foreign body or harmful substances. She reported no allergies to any medication.

Diagnostic Data

Her best-corrected visual acuity was 20/20 OU. External examination was normal with no evidence of afferent defect. Observation during the history revealed intermittent quivering and twitching of the temporal aspect of the right superior eyelid.

Refraction uncovered mild hyperopia with negligible changes to her habitual spectacle prescription. Biomicroscopy revealed normal lids and lashes OU with normal anterior segment structure.

Intraocular pressure (IOP) was measured at 16mm Hg. A dilated funduscopic examination found quiet grounds and normal posterior poles in both eyes.



This 56-year-old patient suffers from seasonal allergies and mild dry eye symptoms, but her chief complaint is an annoying eyelid twitch. Can you identify her diagnosis?

Your Diagnosis

Does this case require any additional tests? What does this patient's history and clinical findings tell you about her likely diagnosies? How would you manage this patient? To find out, please visit www.reviewofoptometry.com.

Retina Quiz Answers *(from page 82):* 1) d; 2) a; 3) b; 4) b & d.

Next Month in the Mag

In January, *Review of Optometry* presents its 9th Annual Pharmaceuticals Report. Topics include:

- *Pregnancy Precautions: How to Prescribe Safely For New and Expectant Mothers*
- *Psych Meds and the Eye: What You Should Know*
- *An Update on Systemic Meds for ODs*
- *Optometric Study Center: Principles of Ocular Pain Control* (earn 2 CE credits)

Also in this issue:

- *Hands-on Series: How to Perform Selective Laser Trabeculoplasty in Early Glaucoma*

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REFERENCES: 1. Data on file. Bausch & Lomb Incorporated. Fit Data. Q4 2014 **2.** Results from a 22-investigator, non-masked multi-site switching study of Bausch + Lomb ULTRA® contact lenses with MoistureSeal® technology, on 327 current silicone hydrogel lens wearers. After 7 days of wear, subjects completed an online survey. Subjects rated performance across a range of attributes. Preference comparisons represent only those subjects expressing a preference. Ratio is based on the average across the silicone hydrogel lenses represented in the study.

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

‡UV-blocking percentages are based on an average across the wavelength spectrum.

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

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