Earn 2 CE Credits: Are You Missing These Optic Nerve Disorders? *p. 66*

REVIEW® OF OPTOMETRY

FEBRUARY 15, 2017

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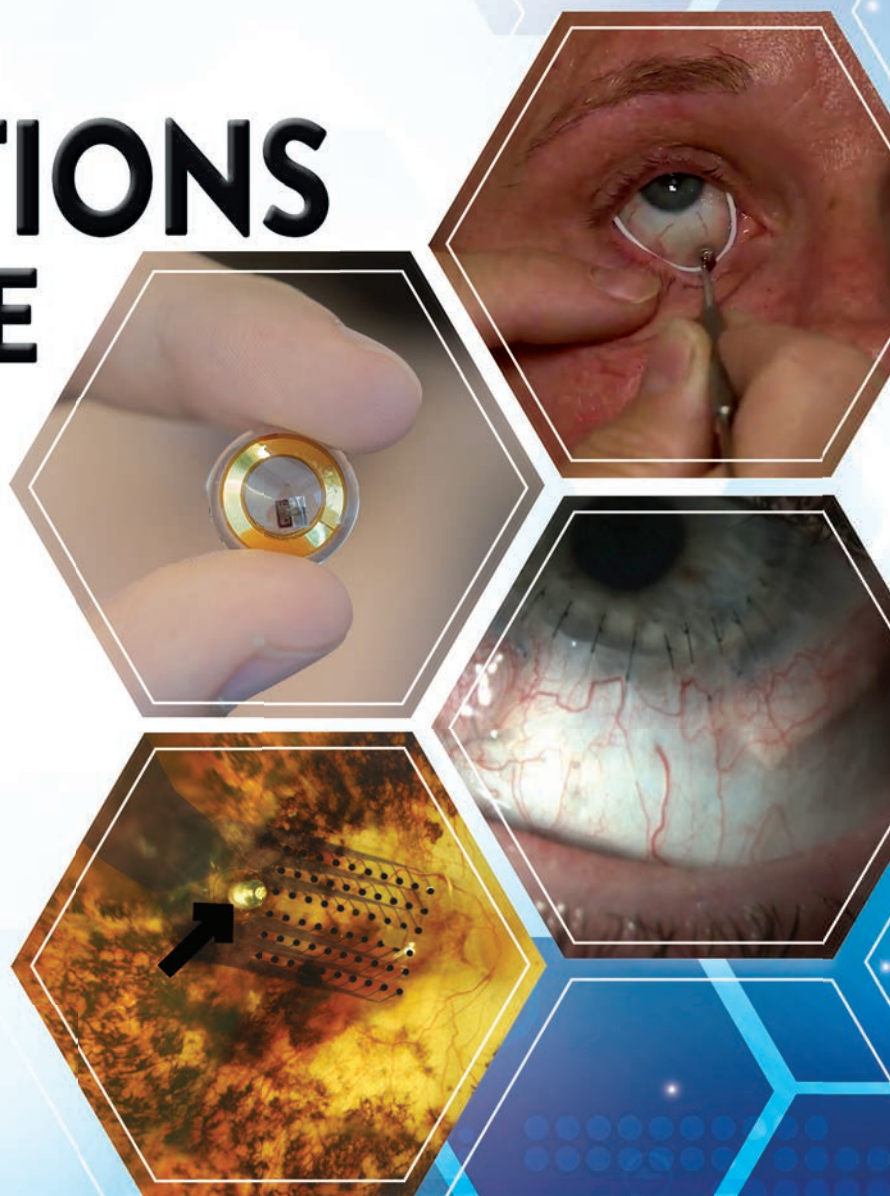
# INNOVATIONS IN EYE CARE





ALSO INSIDE


Sam desires the replacement cycle and affordability of monthly wear but wants to avoid declining comfort over the month, which may be caused by a change in lens hydration.

*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.

## Because I know Sam wants a full month's use from his lenses,

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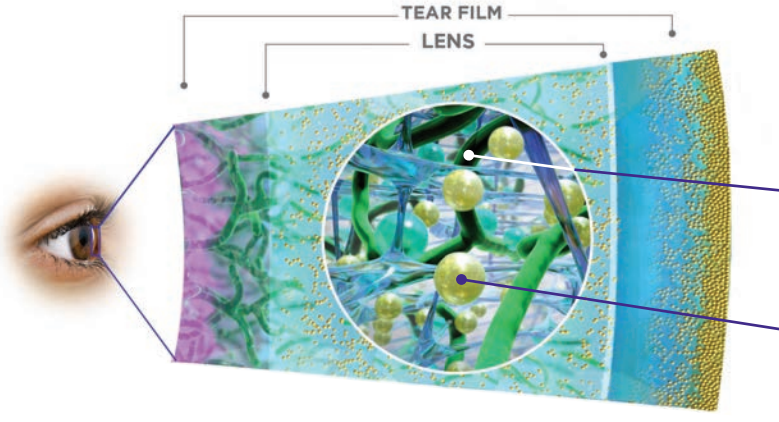


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VOL. 154 NO. 2 ■ FEBRUARY 15, 2017

## IN THE NEWS

**Lucentis** (ranibizumab, Genentech) 0.5mg recently received **FDA approval** for the treatment of patients with **myopic choroidal neovascularization (mCNV)**. Lucentis is the first FDA-approved anti-vascular endothelial growth factor (VEGF) therapy to treat mCNV in the United States—the fifth FDA-approved indication for Lucentis since its launch in 2006. A phase 3 study demonstrated treatment with Lucentis provided superior visual acuity gains in patients with mCNV compared with verteporfin photodynamic therapy.

Researchers recently used confocal adaptive optics scanning light ophthalmoscopy to **image individual retinal ganglion cells (RGCs)** in an animal model. By collecting multiple images, varying in size and location, and combining them, they could visualize not just individual RGCs, but also structures within the cells, such as nuclei. Investigators hope this imaging modality may one day help **assess glaucoma in humans before the retinal nerve fiber thins**.

Ahad Mahootchi, MD, medical director at The Eye Clinic of Florida Same Day Surgery Center, recently performed the first **pre-Descemet's endothelial keratoplasty corneal transplant** procedure with an **eye bank-prepared Descemet's membrane graft**, delivered preloaded in the injector. The tissue, prepared by The Lions Eye Institute for Transplant & Research in Tampa, "made a very difficult surgery very easy. Having the tissue prestamped, prestained and preloaded in the injector saved me over an hour of time," Dr. Mahootchi said in a news release.

# When Technology Makes Us More Human

For some, Internet apps are improving connections with low vision patients. **By Bill Kekevian, Senior Editor**

When a 12-year-old Syrian refugee showed up to the Eye Institute at Salus University in Philadelphia, Erin Kenny, OD, had no way to communicate with her. The young girl spoke no English, was severely visually impaired and was accompanied by a teacher—from St. Lucy's School for the Blind in Philadelphia—who spoke no Arabic. In previous encounters, Dr. Kenny communicated with a friend of the girl's family who could translate, but he was unavailable at this visit.

"She was extremely introverted, didn't make eye contact and was so shy," Dr. Kenny reports. All she knew was the patient's vision had clearly worsened. Dr. Kenny remembered a former colleague—a low vision specialist like herself—who had since moved to Canada and might be able to translate. She grabbed her iPad and reached out to Youssef Neema, OD, using Apple's FaceTime application.

"You could just tell, the moment he started speaking Arabic, her [the patient's] face lit up. He could look at her and she could look at him and they had a real conversation," Dr. Kenny says. "So much can be lost in a language barrier." The patient was so receptive, the girl's teacher even asked Dr. Neema to make some inquires on her behalf. "There were things her teacher needed to know that she couldn't ask her," Dr. Kenny says.

**Dr. Kenny uses apps in practice to help communicate with low vision patients.**

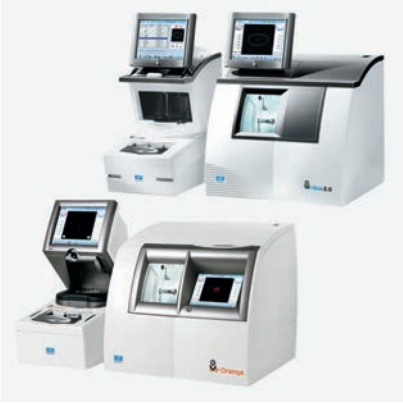
After consulting with the patient, both doctors agreed she had extremely reduced central acuity and inferior visual fields, necessitating the use of a mobility cane. A lot of pediatric patients reject aids such as mobility canes for cosmetic reasons, but for refugees fleeing a war zone, cosmetic reasons may not be so trite. "She was uncomfortable with it because ISIS preys on those who seem weak," Dr. Kenny explains.

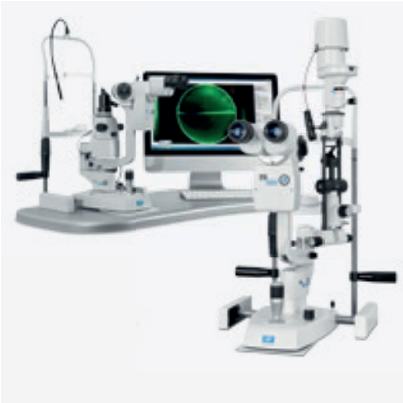
### A Doctor's Eyes and Ears

This isn't the first time Dr. Kenny has turned to high-tech gadgetry for help in a complex situation. In another instance, a patient who was deaf presented with low vision, making communication a unique challenge. Without an aide to accompany the patient, "the exam won't go anywhere," she says. But when the patient turned to an iPad

*continued on page 7*

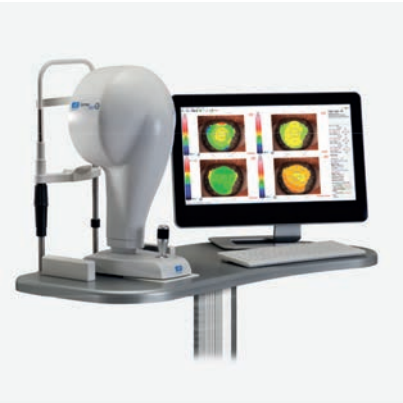
essilor
INSTRUMENTS



# We do more than just lenses



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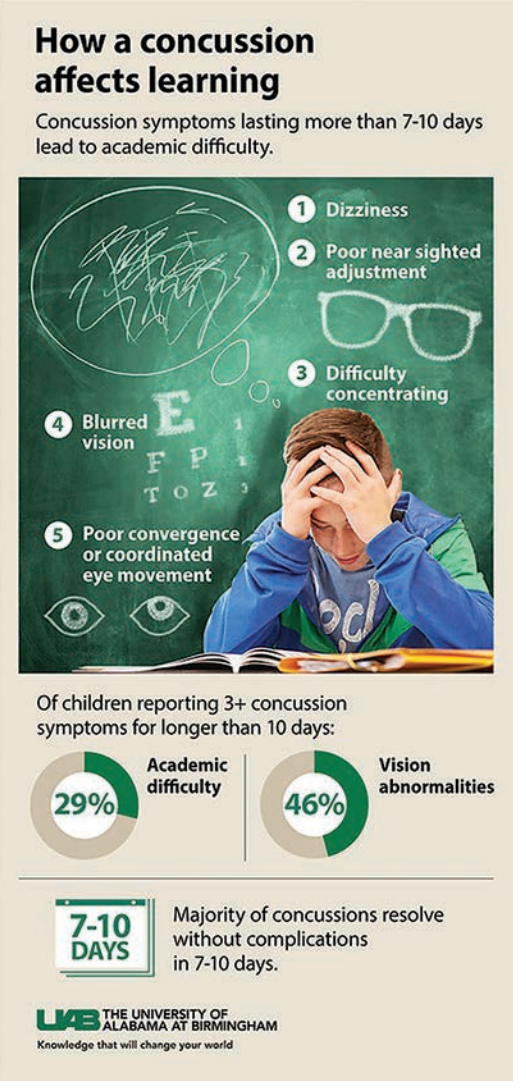
# Post-concussion Vision Symptoms Can Keep Kids out of the Classroom

Students with concussion-related vision problems should undergo comprehensive vision assessments before returning to the classroom, new research suggests.

Although researchers recommend both 'return-to-learn' and 'return-to-play' protocols to evaluate concussion patients' readiness to resume such tasks, less attention has been paid to the return-to-learn protocol than the return-to-play protocol—and patients with prolonged concussion symptoms report academic difficulties.[1]

"We focus so much on returning players to the field, but we lose focus on what happens to them the rest of the time," says Marc Taub, OD, chief of vision therapy and rehabilitation at Southern College of Optometry.

Return-to-learn protocols rarely include vision evaluations in the decision-making process, despite evidence that suggests oculomotor tasks are affected in a majority of pediatric patients and convergence insufficiency yields higher total symptom scores.[1]




**Researchers created an infographic to help explain the effects a concussion can have on a child's ability to return to the classroom.**

Researchers from the University of Alabama (UAB) at Birmingham set out to determine if a link exists between vision symptoms and reports of academic difficulty in pediatric patients suffering from prolonged post-concussive symptoms. Using data collected from Children's of Alabama Concussion Clinic over a period of roughly seven years, researchers studied academic difficulties in a cohort of 276 children ages five to 18, who reported three or more concussion-related symptoms for 10 days or more, as well as a primary symptom of vision problems. Of these, 29% reported difficulties in school and 46% reported vision abnormalities, according to the study.[2]

Traumatic brain injuries impact near focus and eye movement coordination, leading to blurred vision and discomfort.[2] "Because these problems are related to near vision, distance visual acuity testing conducted during a standard eye test may not show problems occurring with deskwork," according to a press release.[2]

"Moving forward, physicians treating concussed patients should consider the damage done to the brain, specifically the vestibular and oculomotor functions, and how long this will affect a child's progression and learning," Mark W. Swanson, OD, lead author of the study, said in the press release.

While specific visual complaints and symptoms are acknowledged, Dr. Taub says, possible findings during a comprehensive vision evaluation and the most appropriate treatment options to enhance the ability to return to learn still need to be addressed further. "We see in the study population a correlation between concussion, visual symptoms and academic difficulty," Dr. Taub says. "This study represents a step toward identifying the links between vision and learning."

1. Swanson MW, Weise KK, Dreer LE, et al. Academic difficulty and vision symptoms in children with concussion. Optom and Vis Sci. 2017 Jan;94(1):60-7.
2. Rohan A. Vision symptoms following concussion can limit a child's ability to return to the classroom. UAB News. Available at www.uab.edu/news/innovation/item/7864-vision-symptoms-following-concussion-can-limit-a-child-s-ability-to-return-to-the-classroom. Accessed 24 Jan 2017.

# Apps in Clinic

*(continued from page 4)*

app to communicate with staff, Dr. Kenny was quickly able to identify the cause of his visual distress.

## App to the Future

Dr. Kenny, who completed her low vision residency last summer, is among the many optometrists and other practitioners who are embracing the use of these so-called "smart" devices in the exam lane.[1-3] In fact, she's incorporated smartphone applications into her daily routine. "I have a list of apps that we've tried in our office that I give to patients at the end of each low vision exam," Dr. Kenny explains. The list "breaks them down into categories—'money reader' apps and color identifiers, for instance—and I star the ones I like."

Some apps are designed to step in for low vision aids, such as portable video magnifiers, which she says can help eliminate the stigma of living with low vision.

"Portable video magnifiers are amazing, but sometimes the costs are prohibitive for our patients. Smartphones can allow that magnification, the change in contrast and other things they need."

Incorporating smartphone apps into regular practice has given low vision a "facelift," says Dr. Kenny. "Our population is only going to get older, and the number of low vision patients is only going to increase." These apps, she says, are helping bolster their quality of life.

1. Lewerenz D. The iPad as a low vision tool. Lecture presented at the AOA annual meeting, Philadelphia, June 28, 2014. Available at www.optometrysmeeting.org/documents/handouts/3115.pdf. Accessed January 11, 2017.
2. Wu R, Rossos P, Quan S, et al. An Evaluation of the Use of Smartphones to Communicate Between Clinicians: A Mixed-Methods Study. J Med Internet Res. 2011 Aug 29;13(3):e59.
3. Whitman L. Hospital clinicians' iPad use: an interim report. Med Ref Serv Q. 2012;31(4):433-8.

# CL Wear: Risky Business

A new survey may help identify risky behaviors that can lead to issues for contact lens (CL) wearers.[1] The Contact Lens Risk Survey was developed to take a closer look at known and potential risk factors associated with soft CL wear among patients. It was also designed to determine differences in soft CL use for those with serious and significant conditions triggered by CL use, those with other red eye events not triggered by CLs and healthy control patients.

In the study's results, patients with serious and significant conditions showed a higher propensity for risky behaviors compared with the other two groups, including overnight wear, internet purchasing, two-week replacement, failure to discard leftover solution on a daily basis and napping in lenses. The survey also introduced a new risk factor among patients with serious and significant conditions: sharing a bedroom. This was initially included in the survey because of reports that confined living situations potentially heighten disease transmission.[2-4] The survey's results instead suggested the opposite, possibly because not sharing a bedroom is different than living alone.

"We hope that in the future the survey will be used to help researchers and practitioners identify patients who are exhibiting risky behaviors associated with soft contact lenses," says Heidi Wagner, OD, one of the study's authors. "It would potentially be used to characterize the participant's risk profile and provide individualized feedback regarding risky soft CL behaviors and assess interventions aimed at reducing soft CL-related complications." ■

1. Sorbara L, Zimmerman AB, Mitchell GL, et al. Multicenter testing of a risk assessment survey for soft contact lens wearers with adverse events: a contact lens assessment in youth study. Eye & Contact Lens. October 13, 2016. [Epub ahead of print].
2. Rimza EM, Kirk GM. Common medical problems of the college student. Pediatr Clin North Am. 2005;52:9-24.
3. Round A, Evans M, Salmon RL, et al. Public health management of an outbreak of group C meningococcal disease in university campus residents. Eur J Public Health. 2001;11:431-6.
4. Turner JC. How colleges can plan for the bird flu. Chronicle Rev. 2005;52:B20.

## Optometry's Role in 21st Century Cures Act

The 21st Century Cures Act, a new law that aims to invigorate medical research and speed up the nation's drug regulation system, includes $6.3 billion in new funding for research and alters the FDA approval process to hopefully make approvals smoother and quicker.

While the impact will likely differ across medical fields, optometry played a part in shaping the act to ensure it makes sense for eye care. The American Optometric Association (AOA) "helped shape the bipartisan 21st Century Cures Act and prevent an irresponsible expansion of telehealth," says Andrea P. Thau, OD, AOA president.

Early proposals looked to expand telehealth service into Medicare, and one proposal even suggested new authority to replace face-to-face care. The AOA spoke out strongly against this, and it paid off. The final law simply calls for the Department of Health and Human Services to study possible effects of telehealth expansion. "From the very start, we made clear that, while we fully embrace appropriate uses of new and evolving technologies, we will do everything in our power to beat back those seeking to undermine the standard of care our patients need and deserve," Dr. Thau says. "We were able to fight back and win against those seeking to downplay and discount the essential, in-person care provided by America's doctors of optometry."

Bonamici S. H.R.34 - 21st Century Cures Act. Available at www.congress.gov/bill/114th-congress/house-bill/34/text. Accessed January 25, 2017.

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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

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

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

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

### Nursing Mothers

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

### Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

### Geriatric Use

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

## NONCLINICAL TOXICOLOGY

### Carcinogenesis, Mutagenesis, Impairment Of Fertility

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

## PATIENT COUNSELING INFORMATION

### Administration

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

### Risk of Contamination

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

### Contact Lens Wear

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

### Risk of Secondary Infection

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

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

US Patent No. 5,800,807



LGX.0114.USA.16
Based on 9269101/9269201 Revised: 08/2016

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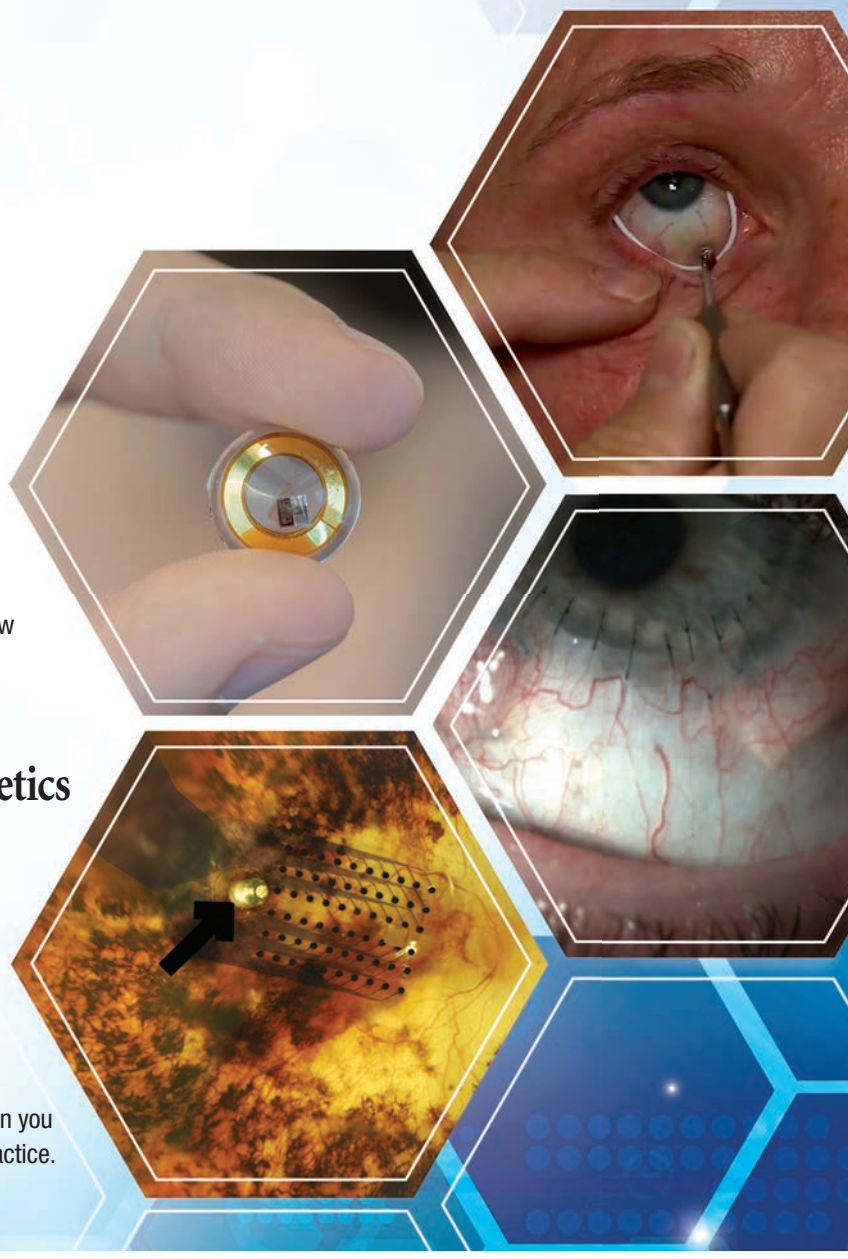

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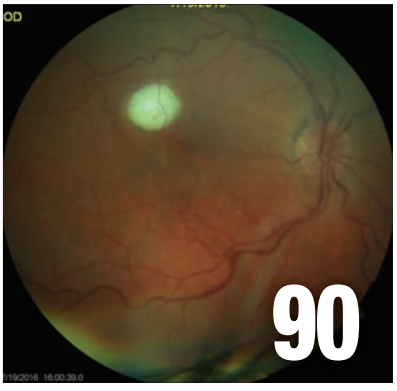
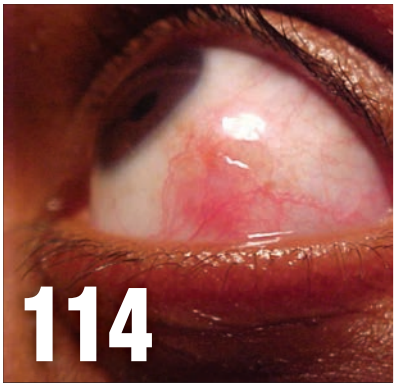
Review of Optometry February 2017





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JOBSON MEDICAL INFORMATION LLC

Outlook

By Jack Persico, Editor-in-Chief

# We Have the Technology

Our innovation issue celebrates science with a look at medical breakthroughs here today and on the way.

Those of us who grew up in the 1970s (and were of a geeky mindset) remember the famous voice-over that opened every episode of *The Six Million Dollar Man*: "Steve Austin, astronaut. A man barely alive. Gentlemen, we can rebuild him. We have the technology. We have the capability to build the world's first bionic man. Steve Austin will be that man. Better than he was before—better, stronger, faster."

I've been a lifelong admirer of and advocate for science, in no small part because of that show and other idealistic portrayals of science's transformative effect. Sci-fi posits a medical world that can conquer disease, trauma and degeneration, extend our lifespan and even augment human capabilities. It fires up the imagination and gives researchers the audacity to believe they can change the world.

*Do* we have the technology? For Steve Austin, no—at least not yet. But we've made progress on many fronts that gives tangible benefits today and optimism for tomorrow.

Ophthalmologist Mark Humayun has spent nearly 30 years working to advance the cause of restoring sight in severe low vision patients. His retinal implant may not match Steve Austin's bionic eye, but it gives some functional vision to retinitis pigmentosa patients who otherwise would be completely sightless. We're honored to have a contribution from him in this issue on innovation. On page 46, he and his colleagues explain the arduous work and hard-won gains achieved in developing the Argus II epiretinal implant.

So-called smart contact lenses—another staple of science fiction—may actually deliver on their slate of promises. Already, the Sensimed Triggerfish lens can measure ocular changes in glaucoma that correlate with IOP. Well underway are efforts to deliver on-eye biometric monitoring of glucose levels in diabetes patients and real-time accommodation for presbyopes. Some of the more ambitious—and, for privacy advocates, troubling—proposed uses include image and video recording embedded in a contact lens and the ability to add a layer of augmented reality to the wearer's field of view. On page 54, optometrists Brian Chou and Jerome Legerton report on this fast-moving frontier.

Technology looks ready to finally crack the compliance problem, with sustained-release glaucoma devices that would obviate the need for patient-administered medications. Shira Kresch, OD, profiles many exciting projects in her article on page 40. While some topics featured this month are moonshots, this one looks capable of delivering real-world results to millions—soon.

Advances that enable cutting-edge stem cell therapy for corneal healing are the focus of the article by James Thimons, OD, on page 33. Patients once destined for graft surgery might benefit from a safer, simpler procedure. "It is rare in a clinician's lifetime to witness the birth of a new technology, but with today's stem cell advancements, we stand at the dawn of a true paradigm shift," says Dr. Thimons in his feature. "Welcome to the future." ■

# If You Can't Beat 'Em, Join 'Em

You optometry bullies know who you are, and I have a plan to get back on top.

**By Montgomery Vickers, OD**

Before we begin, I want to take this opportunity to thank all the bullies I faced as a kid. I just read that dirt is actually good for you, as the *Mycobacterium vaccae* we accidently ingest can increase serotonin levels, reduce depression, increase learning and help slow myopia progression. So thanks, bullies, for all that dirt you made me eat.

Bullies don't go away just because you grow up. In fact, even optometrists can bully one another. The worst bullies are CE presenters. Ohhh, don't play dumb. You tell us stuff that we never heard of before, and we are so frightened of your obviously superior intellect we can't even think of a good question to ask, except "How much do you charge for your contact lenses?" You think we are all losers!

That's right. You bully us. We need to defend ourselves and quit letting you optometry bullies make us look dumb. Here's my approach to toughen up my image:

(1) I plan to become a world expert in something—anything. If you can do it, why not me? After listening to one lecture on myopia control, that field kinda spoke to me. After all, to be a world expert in myopia control, all you have to do is answer every question with, "We really are not sure." Seems doable to me. Feel free to call if you need to know the facts on how myopia control works because, as a world-class myopia expert, I am really not sure.

(2) To increase my fame, I'll write a best-selling book Tom Hanks casually picks up at LaGuardia while waiting for a late flight back to LAX. He reads it all the way home, then calls me and gives me tons of money for the rights to make a movie. When the movie wins a stack of Oscars, I go to the after parties where I meet Sir Paul McCartney, who asks if I know anything about rock 'n' roll. I answer, "I am not really sure" and, since that's how his career started, he recognizes me as a world expert on rock and roll and hires me to join his new band, which I expertly name the "Odeatles." There's no reason this won't work. Tom, Paul: call me.

(3) I'll only attend CE meetings in states that allow open carry of firearms. Since I don't actually own a gun, I'll wear an NRA hat and stick a retinoscope in my holster. Young doctors have never seen one, so they'll assume I'm packing and let me sit wherever I want—near the free breakfast banana muffins. Man, it feels good to intimidate…

## I've Already Joined 'Em

Writing *Chairside* all these years has already given me some street cred I don't deserve.

That's why I never got that teardrop tattoo below my eye. Don't need it. My colleagues somehow presume I know something important when all I really know is that I have no clue. But, in today's touchy-feely world, having no clue is a sign of strength. Maybe that's the secret. Consider myopia control world experts, for example.

Finally, to firmly establish myself as a force to be reckoned with, I want everyone to know I can answer that age-old question: "How much are your contact lenses?" Bully CE presenters just can't answer that. They are too busy studying bearded gnat eyes to determine why they never get macular degeneration. (It's because they get resveratrol from sucking the blood of winos.)

So how much are my contact lenses? Free. The sales rep hooks me up. Why, do you have to buy yours?

What losers… ■

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Through **My Eyes**

**By Paul M. Karpecki, OD, Chief Clinical Editor**

# The 'Eye-tech' Practice

In 2017, expect new technologies with potential to change eye care and patient's lives.

The pace of innovation seems to gain speed each year, and 2017 may provide even more breakthroughs than ever. This month and next I'll highlight a few, first with a focus on treatment modalities below and then a look at diagnostic technologies in March.

• ***Corneal collagen crosslinking.*** Approved last year after more than a decade of international experience, this procedure offers nonsurgical strengthening of the cornea for keratoconics, and perhaps a way to prevent post-LASIK ectasia. Some ODs are already wondering if this fairly low-risk procedure might one day make its way to optometric practice. Whether as providers or comanagers, ODs will play a key role.

• ***Dry eye.*** The success story of 2016 was the launch of Xiidra (lifitegrast, Shire), which is already changing how we practice. New treatments likely in 2017 include TrueTear, a neurostimulus device from Allergan that can activate the trigeminal nerve to trigger tear production (likely all three layers), and Dr. i-Coach, an overlay placed on a mobile device to train patients to blink frequently and properly. It also encourages proper eye elevation ergonomics, illumination and relaxation of the accommodative system.

• ***Glaucoma therapy.*** New drugs anticipated include latanoprostene bunod (Vyzulta), a nitric oxide-donating prostaglandin with two outflow pathways that shows improved IOP-lowering capacity. Three new-class therapeutic agents in Phase III trials include the Rho kinase inhibitor netarsudil mesylate 0.02% (Rhopressa), the norepinephrine transporter inhibitor trabodenoson (a highly selective adenosine type 1 receptor agonist) and Roclatan, which combines netarsudil mesylate with latanoprost; in recent clinical trials, it showed more IOP lowering than any drug to date. Watch for these in late 2017 or early 2018.

But 2017 may be the year of minimally invasive glaucoma surgery (MIGS). Recent approvals of the Xen implant from Allergan and Cypass from Alcon will continue to boost awareness of MIGS.

• ***Drug delivery systems.*** Expect a wave of innovation here to begin this year. Only about 10% of a topical drug typically penetrates the cornea, and compliance is a major issue for all patients. Both could potentially be addressed with devices we could see in 2017, including Dextenza, a punctal plug that houses dexamethasone. In Phase III trials, it showed a statistically significant improvement over placebo in absence of pain and inflammation after cataract surgery. Glaucoma and dry eye medications are also being studied via this system.

• ***Cataract surgery innovations.*** AMO's Symfony IOL provides good near and intermediate vision along with distance correction, plus a toric option. It has been a great recent addition for patients interested in better post-op vision. Its more forgiving nature has set a high bar.

Alcon's just-approved AcrySof IQ PanOptix Toric is another new way to correct for near, intermediate and distance vision, plus astigmatism.

A new technology in the final steps of FDA clinical trials may also dramatically alter the landscape in 2017 if approved. The Calhoun Vision light adjustable lens allows surgeons to adjust the final IOL power post-surgically, using an ultraviolet laser to fine-tune the refraction. If the patient has an IOL calculation 'surprise' and ends up +1.00 after surgery, they can have the lens altered with the laser to correct it to plano. This technology eliminates the influence of the inherently unpredictable healing response.

• ***Wearabless and telemedicine.*** Consumer and professional products are changing our visual experience of the world. Eyeglass-mounted cameras from PogoTec and Snap will capture consumer interest in wearable tech. For surgeons, technologies like TruVision Systems improve ocular surgeries like cataracts and especially retinal surgery by providing 3-D enhanced imaging and the ability to overlay angiography or OCT images for precise manipulation of tissue.

Finally, a group of optometrists (CoolDoctors) have advanced the area of telemedicine for our profession with an impressive platform that will allow HIPAA-complaint follow-up visits and acquisition of new patients digitally, billable in your state of licensure. Expect this trend of remote access to patients to advance in 2017 and beyond. ■

*Relevant financial disclosures for Dr. Karpecki: Aerie Pharmaceuticals, AMO, Alcon, Allergan, Avedro, Bausch + Lomb, Calhoun, CoolDoctors, Eyes4Lives, Glaukos, Ocular Therapeutix, PogoTec, Shire.*

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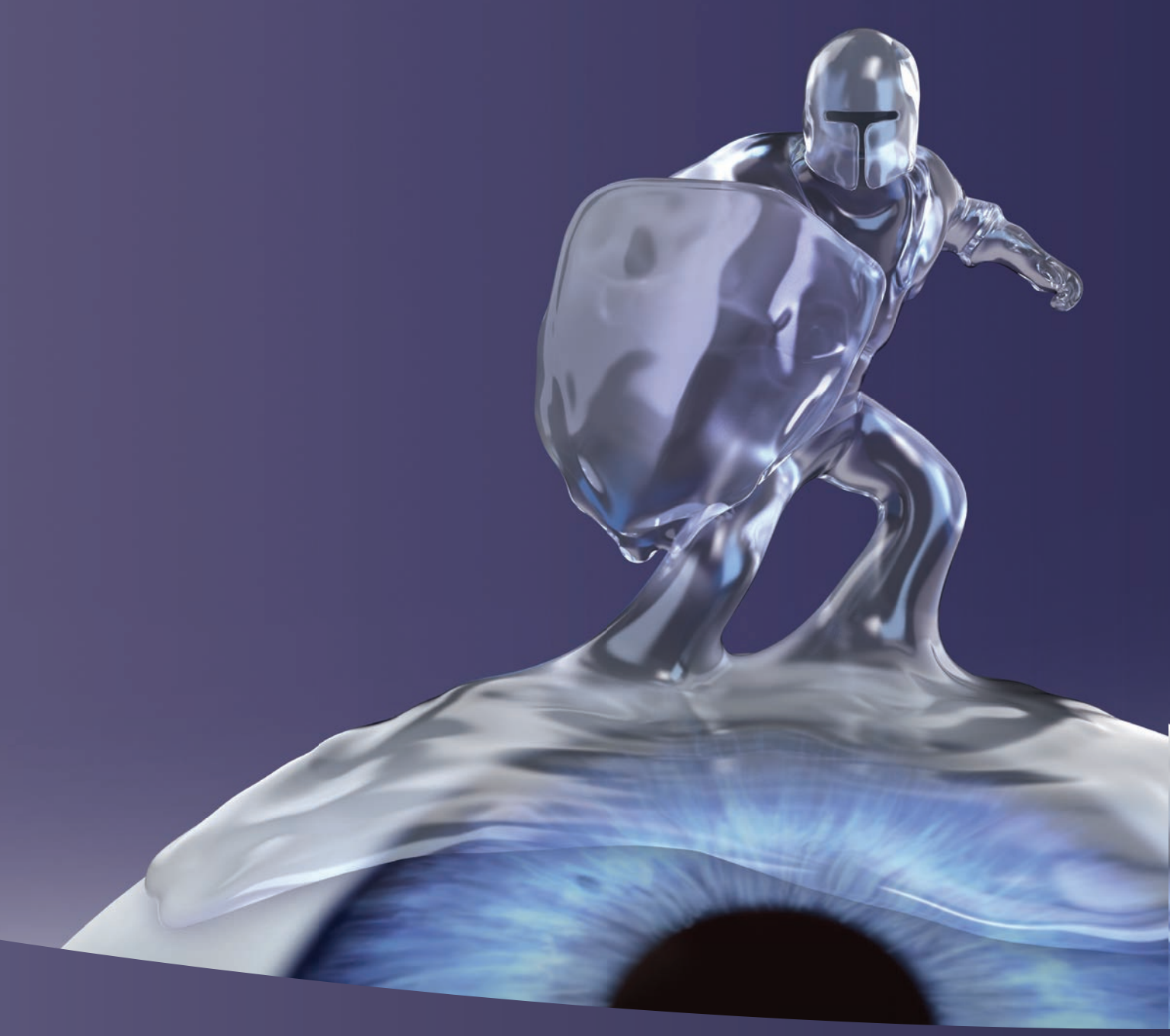

**Indications and Usage**
BromSite™ (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

**Important Safety Information**

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

  It is recommended that BromSite be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- Use of topical NSAIDs may result in keratitis. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular

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surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

- BromSite should not be administered while wearing contact lenses. The preservative in BromSite, benzalkonium chloride, may be absorbed by soft contact lenses.
- The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

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

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

NSAID=nonsteroidal anti-inflammatory drug.

**References: 1.** BromSite [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016. **2.** Hosseini K, Hutcheson J, Bowman L. Aqueous humor concentration of bromfenac 0.09% (Bromday™) compared with bromfenac in DuraSite® 0.075% (BromSite™) in cataract patients undergoing phacoemulsification after 3 days dosing. Poster presented at: ARVO Annual Meeting; May 5-9, 2013; Seattle, Washington. **3.** Bowman LM, Si E, Pang J, et al. Development of a topical polymeric mucoadhesive ocular delivery system for azithromycin. *J Ocul Pharmacol Ther.* 2009;25(2):133-139. **4.** ClinicalTrials.gov. Aqueous humor concentration of InSite Vision (ISV) 303 (bromfenac in DuraSite) to Bromday once daily (QD) prior to cataract surgery. https://clinicaltrials.gov/ct2/show/results/NCT01387464?sect=X70156&term=insite+vision&rank=1. Accessed July 18, 2016. **5.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66.



SUN OPHTHALMICS

# BromSite™ (bromfenac ophthalmic solution) 0.075% Brief Summary

## INDICATIONS AND USAGE

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

## DOSAGE AND ADMINISTRATION

**Recommended Dosing**

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

**Use with Other Topical Ophthalmic Medications**

BromSite should be administered at least 5 minutes after instillation of other topical medications.

**Dosage Forms and Strengths**

Topical ophthalmic solution: bromfenac 0.075%.

## CONTRAINDICATIONS

None

## WARNINGS AND PRECAUTIONS

**Slow or Delayed Healing**

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

**Potential for Cross-Sensitivity**

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

**Increased Bleeding Time of Ocular Tissue**

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

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

**Keratitis and Corneal Reactions**

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

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

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

**Contact Lens Wear**

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

## ADVERSE REACTIONS

**Clinical Trial Experience**

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

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

## USE IN SPECIFIC POPULATIONS

**Pregnancy**

Risk Summary

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

Clinical Considerations

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

Data

*Animal Data*

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

**Lactation**

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

**Pediatric Use**

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

**Geriatric Use**

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

## NONCLINICAL TOXICOLOGY

**Carcinogenesis, Mutagenesis and Impairment of Fertility**

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

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

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

## PATIENT COUNSELING INFORMATION

**Slow or Delayed Healing**

Advise patients of the possibility that slow or delayed healing may occur while using NSAIDs.

**Concomitant Topical Ocular Therapy**

If more than one topical ophthalmic medication is being used, advise patients to administer BromSite at least 5 minutes after instillation of other topical medications.

**Concomitant Use of Contact Lenses**

Advise patients not to wear contact lenses during administration of BromSite. The preservative in this product, benzalkonium chloride, may be absorbed by soft contact lenses.

**Sterility of Dropper Tip/Product Use**

Advise patients to replace the bottle cap after use and do not touch the dropper tip to any surface as this may contaminate the contents.

Advise patients to thoroughly wash hands prior to using BromSite.

**Rx Only**

**Distributed by:** Sun Pharmaceutical Industries, Inc. Cranbury, NJ 08512



SUN-OPH-BRO-017 09/2016

SUN OPHTHALMICS

# Managing a Cat-tastrophe

A bacterial infection transferred from a pet can lead to urgent eye issues.

By Nicole Stout, OD, Abby Gillogly Harsch, OD, and Richard Mangan, OD

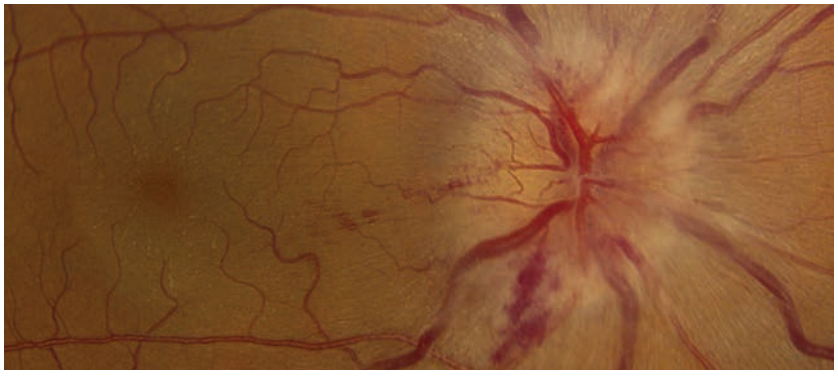
Cat scratch disease (CSD) is caused by the gram-negative bacillus *Bartonella henselae*, and is most commonly transmitted through a lick, bite or scratch by a flea-infested cat (usually a kitten).[1-3] A red papule may arise at the site of infection within one week, and is typically followed by regional lymphadenopathy. Patients may experience general malaise, fever and pain in the area of the affected lymph node. CSD predominantly affects the pediatric population, and can lead to serious systemic sequelae if disseminated, particularly in the immunocomprised.[1-3] While CSD affects approximately 22,000 Americans, only 5% to 10% of cases involve ocular tissue.[1,2]

## Anterior Complications

The most common ocular complication is parinaud oculoglandular conjunctivitis, presenting with unilateral conjunctival redness, foreign body sensation, serous or purulent discharge and one or more granulomatous nodules on the conjunctiva. These ocular signs occur in addition to an ipsilateral swollen preauricular, submandibular or cervical lymph node.[4] Antibiotic treatment in these presentations is considered optional, as it is typically self-limited and resolves in two to six weeks.[1,3]

## Posterior Complications

CSD affecting the posterior segment, however, is a different story. Cat-scratch neuroretinitis is considered an atypical, serious presentation of CSD and occurs in approximately 1% to 2% of cases.[3,5] Although rare, cat scratch neuroretinitis is the most common form of infectious neuroretinitis.[6] One study found that *Bartonella henselae* is the most common cause of neuroretinitis with 64.3% of tested patients with neuroretinitis having positive serological analysis for *Bartonella henselae*.[7] Presenting symptoms of cat scratch neuroretinitis include acute central vision loss (88%), which is typically unilateral, with the associated systemic complaints discussed above.[6] Vision loss is typically painless; however, eye pain has been reported to occur in about 8% of cases.[6] Color vision changes, including red desaturation, may be present on examination.



**This patient displays optic nerve swelling and a neurosensory retinal detachment in the right eye. If you look closely at the macula, you'll see exudate in a "macular star" pattern. This presentation most likely represents a neuroretinitis caused by *Bartonella henselae*, or cat-scratch disease.**

Inflammation of the posterior segment can indicate a potential case of CSD. The classical description of neuroretinitis is optic disc edema with the presence of a macular star (ODEMS). The macular exudates are a result of serous leakage and may not be present on initial examination. These findings often resolve to 20/40 or better central vision.[6] More site-threatening complications of CSD include retinal vasculitis and vascular occlusions. A relative afferent pupillary defect may be noted in unilateral cases. A serous retinal detachment or vitritis, or both, may also be present.[1] Discrete, white chorioretinal lesions have recently been a more common posterior segment finding related to CSD.[1] Performing an OCT can be useful in confirming or ruling out subtle amounts of intraretinal fluid or a mild/early serous detachment.

## Diagnosis Pearls

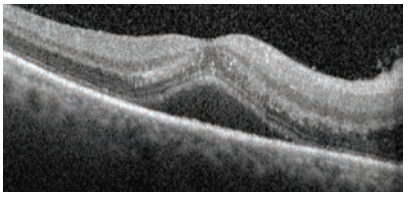
Apart from the urgency of treating moderate-to-severe CSD, consider

the often-urgent differentials. Other conditions known to cause posterior uveitis and retinitis such as lyme disease, sarcoidosis, syphilis, and toxoplasmosis should be considered. Malignant hypertension and diabetic papillopathy must also be ruled out as both conditions can cause ODEMS. Optic nerve edema also necessitates consideration of intraocular mass, increased intracranial pressure and viral infections. Rare cases of bilateral CSD can mimic pseudotumor cerebri.[1] CSD can also appear clinically similar to Vogt-Koyanagi-Harada syndrome.[1,2,6]

In cases of possible CSD, an important step in diagnosis is asking for known history of exposure to kittens or cats. Although patients will typically report close contact with a cat, cases have been reported where no history of contact with a cat have been reported.[10] CSD should always be considered when a child presents with neuroretinitis. If CSD is suspected, serology testing can be performed for IgM and IgG antibodies. Initial negative serology testing does not rule out CSD and can be repeated even after treatment is initiated.[8] One group of researchers showed an MRI localizing enhancement to the optic nerve-globe junction has much higher reliability than serology testing.[8,9] A biopsy of the affected lymph node can also yield *Bartonella bacillus* for diagnosis. Typically, treatment is initiated based on clinical signs and a positive history of exposure.

## Treatments

As previously mentioned, CSD is typically self-limiting; however, most ocular findings in CSD are indicative of a moderate-to-severe infection. In these cases, antibiotic treatment is recommended. Controlled, prospective studies of treatment in ocular manifestations of CSD are lacking in the literature; however, one group did publish recommendations in 2004.[3] They emphasized the marked difference in immunocompromised versus immunocompetent patients' improvement with antibiotic therapy. In the absence of posterior segment disease, the authors felt that antibiotic therapy in otherwise healthy patients was not strongly recommended. Immunocompetent patients with retinitis appeared to improve with doxycycline or rifampin treatments, or both.[1,3,6] Ciprofloxacin has also been suggested as a treatment in adults who are not pregnant.[6] Azithromycin displayed a faster resolution of lymphadenopathy than no treatment; however, doxycycline demonstrates better penetration of intraocular tissues. Investigators suggest erythromycin for pediatric patients.[1,3]



**This OCT scan shows the same eye as on page x. An OCT scan can help detect intraretinal fluid of early serous detachment in CSD patients.**

Researchers also found that a four-to-six week course of oral doxycycline (100mg BID) and rifampin (300g BID-TID) promoted resolution of *Bartonella henselae* neuroretinitis, in addition to the systemic infection.[11] They found that once antibiotic therapy was initiated, no further progression of ocular inflammation was noted and disc swelling was reduced within a few days.[11] The study concluded that visual recovery was most rapid if the time between symptom onset and initiation of antibiotic therapy was minimized.[11] It is for the above reasons that it is so important to recognize the signs and symptoms of cat scratch neuroretinitis and initiate treatment promptly.

The decision to initiate treatment may be made clinically or in consultation with the patient's primary care provider. As discussed, moderate-to-severe CSD poses risks of systemic dissemination, encephalopathy, and liver, spleen and heart complications.

After the successful diagnoses and treatment of cat-scratch neuroretinitis, it is vital encourage patients who have been diagnosed with cat-scratch neuroretinitis to be monitored long term for signs of late onset optic-neuropathy.[11] ■

*Dr. Stout is a clinical instructor at the University of Waterloo School of Optometry and Vision Science.*

*Dr. Harsch is in practice at Nittany Eye Associates in State College, PA.*

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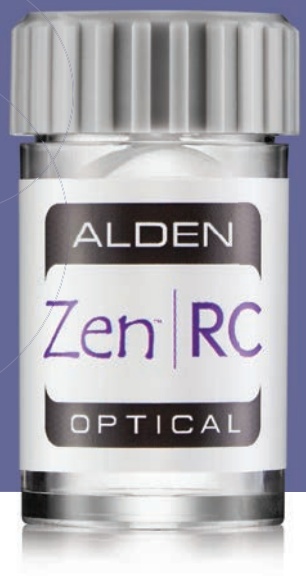


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# Got Milk?

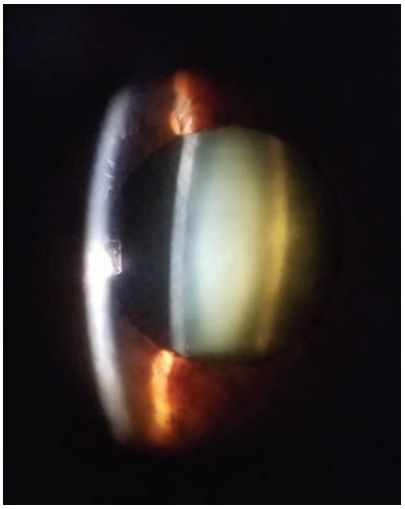
A patient presents with bilateral vision loss and nothing obvious to explain it. What gives? It could be a milky nuclear sclerotic cataract. **Edited by Paul C. Ajamian, OD**

**Q A 52-year-old Caucasian male presented with unexplained vision loss in both eyes. He's a 10-diopter myope, which of late has increased by two diopters, and the referring doctor noted an "early" cataract. The patient was sent for a retina consult and possible neuroimaging. Is the answer hiding in plain sight?**

A This doctor should be commended for raising red flags on vision not correctable to 20/20 without an obvious explanation. "When I get referrals for unexplained vision loss, I initially think about occult corneal irregularity, milky NS, subtle retinal conditions like an epiretinal membrane or lamellar hole, and neurological issues," says Edward Wasloski, OD, clinical director at Omni Eye Specialists of Baltimore. Once you have ruled out corneal problems with the slit lamp and topography, and retinal problems with a dilated exam and OCT, turn your thoughts to a diagnosis of milky nuclear sclerotic cataract or 'milky NS,' says Dr. Wasloski.

The majority of the time, taking a thorough history and listening to the patient will lead you to the proper diagnosis. "The first thing I ask about are halos, monocular ghost images, or diplopia," explains Dr. Wasloski. "Double vision is not something any of us necessarily like to deal with, but when you can demonstrate it in one eye only, the culprit is usually either cornea or cataract,."

Dr. Wasloski says that, since milky NS cataract is different in appearance and clinical findings than cortical or brunescent NS presentations, failing to recognize it early on can lead the patient on a wild goose chase. "The practitioner will put the patient through test after test—from retinal consults to neuroimaging—all of which return normal results and above-normal frustration."



**In the absence of posterior and anterior segment findings, think about a diagnosis of "milky NS.'**

Key characteristics include a central dark reflex on retinoscopy and the characteristic biomicroscopic appearance of a 'lens within a lens.' The opalescent or milky white appearance of the nucleus with no surrounding brunescence or cortical spoking is pathognomonic.

Often, a shift in nearsightedness is noted, ranging from one to six diopters. "The patient will also be complaining of significant glare and haloes around lights, especially at night. On dilated exam, note the bowing of the fundus beam that almost appears as if a staphyloma is present wherever the beam is placed on the retina," says Dr. Wasloski. This is most apparent through the old Hruby lens, rarely used today, but can still be seen to a lesser degree with handheld lenses behind the slit lamp."

Dr. Wasloski says that the view through these cataracts is often very clear, which is what can throw you off. The more common brunescent or posterior subcapsular cataracts will obscure your view into the eye, which will be consistent with the patient's view out. "Always think milky NS when the cataract doesn't look like it can cause the level of acuity reduction that you have measured," he says. Milky nuclear sclerotic patients are typically younger, with no associated medical conditions or medication use. The cataracts are often asymmetric between eyes.

## Treatment

Dr. Wasloski says cataract surgery is the easy answer, and by catching the condition early, you're saving your patients the worry and frustration associated with unexplained vision loss. And, don't forget to review the various options for lens implants and standard vs. laser-assisted cataract surgery before you refer them. "Patients are looking for your advice; so, before referring, give your opinion on the options that will best suit them and their lifestyle." ■

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# Ups and Downs of Innovation

Beware of these common pitfalls when integrating new technology into your practice.

**By John Rumpakis, OD, MBA, Clinical Coding Editor**

Innovative technology is advancing at a breakneck pace, and nowhere is that more apparent than in the realm of ophthalmic care. Every day there is news of new diagnostic and treatment devices, new procedures and new medications, to name only a few.

But therein lies the challenge from a coding and reimbursement perspective. There may be limitations that impede your ability to use new technologies in your clinical setting. Actually, I should be more specific: there may be limitations with a third party medical carrier when it comes to paying for this innovative care. Here are two pitfalls to look out for:

***1. Who's Footing the Bill.*** We have a hard reality to face, especially due to the changes in the medical insurance environment. Insurance premiums are increasing all across the country, causing deductibles to rise because the consumer dollar doesn't go as far in buying coverage. That means that even for covered services, the patient will generally be the one responsible for the out-of-pocket cost until meeting their deductible. Even then, they may still encounter challenges with coverage and affordability.

Additionally, if something is not a covered service for a specific condition, the out-of-pocket expense for the test or procedure doesn't apply to the deductible, making it even less affordable for most patients.

***2. What's Covered, What's Not.*** The realities of applying new technologies in practice may not always be what was represented by the sales person who sold you the equipment. Consider visual evoked potential (CPT code 95930), for example. This test helps detect glaucoma far earlier in the disease cycle—and I personally think our ability to deliver this test in a day-to-day clinical environment is incredible.[1,2] Unfortunately, there is a disconnect with insurance carrier policy for tests such as this. According to CMS, there are only three CMS carriers that have a coverage policy on 95930.[3] And not a single one allows this test for any diagnosis related to glaucoma.[4-6]

So what does that mean? Do you stop doing the test that assists in the early detection? Most likely not, but it does mean you should use an Advanced Beneficiary Notice of Noncoverage (ABN), knowing the patient will be bearing the cost.

This is a classic example of when technological innovation and third party coverage policy collide, and we will see more of this in the future, as coverage policies become based on outcomes and economics.

Innovations in technology that apply to direct patient care are integral to our ability to diagnose and treat patients effectively. Yet we must strike a balance in how we apply that technology based on the medical necessity for that specific patient and the cost the technology adds to the episode of care.

Patient contributions to their own health care costs are increasing—let's make sure they spend their dollars wisely. ■

*Send questions and comments to ROcodingconnection@gmail.com.*

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## What's an ABN?

The ABN is a written notice you provide to a Medicare beneficiary before items or services are furnished when you believes Medicare will not pay for some or all of the items or services. The most current ABN form and instructions can be downloaded at www.cms.gov/Medicare/Medicare-General-Information/BNI/ABN.html.

In addition, there are four common modifiers that can be appended to CPT codes for the procedures that may be denied by the carrier. Depending on the service provided and the specific circumstances, the modifier can be either required by Medicare or voluntarily appended to the CPT code. While the ABN is a formal document required by Medicare, the concepts here often also apply to other commercial medical carriers as well.

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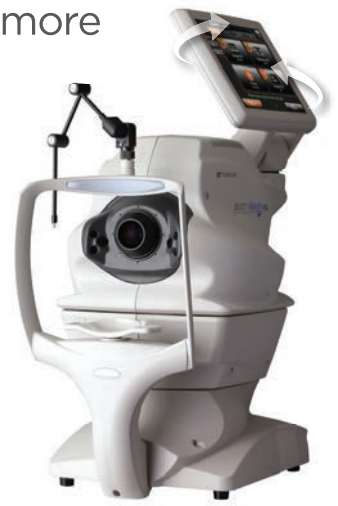

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INNOVATIONS
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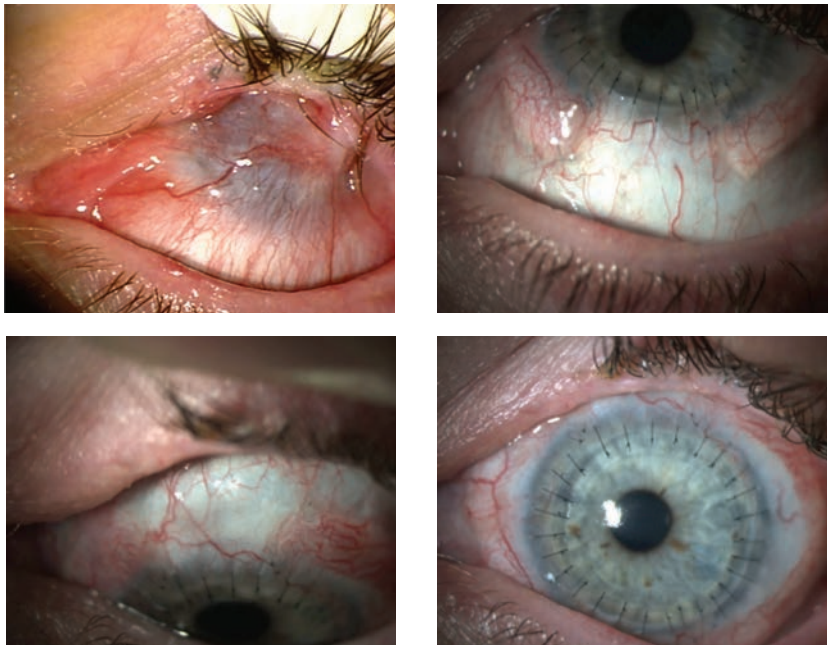
# Stem Cells: The Future of Corneal Rehabilitation

Modern science can provide replacements for dead tissue that's been historically complicated to replace. **By J. James Thimons, OD**

Niels Bohr, one of the preeminent physicists of the 20th century, once said "predicting can be difficult, especially when it involves the future." Twenty years ago, it was more science fiction than reality that stem cell transplantation could treat a variety of vision-debilitating diseases that have historically eluded intervention. The last two decades have seen an explosion of science surrounding stem cell transplantation for the eye, specifically in regards to limbal stem cells and the critical role they play in corneal health.[1]

In the past, stem cells were harvested primarily from cadavers. More recently, they came from autologous sources and live donations from relatives. But today, science has developed the capacity to cultivate limbal stem cells *in vitro*, and transplant them onto the recipient's tissue bed in an effort to manage disease states.

This article provides an overview of these modern advancements.



Photos: Ed Holland, OD

**These images show a patient with epithelial defects before (top left) and after stem cell therapy in conjunction with corneal transplantation.**

## Pathophysiology

Corneal epithelial cells undergo constant mitosis and migration to provide a clear, smooth optical pathway for light to enter the eye and allow for visual stimulation.[2-4]

While this basic process involves numerous levels of cellular activity, its primary responsibility—new cell development—is dependent upon a population of stem cells that reside in the palisades of Vogt at the limbal margin.[2-4] These cells serve as an engine for cellular replication and corneal health.[2-4] Additionally, the limbal margin serves as a barrier to infectious disease and a mediator for anterior segment inflammation.[2-4] When violated, it can succumb to the progression of a disease, permitting it to enter corneal space.[2-4]

Corneal limbal stem cells, unlike other stem cells, are specifically directed toward developing new cellular tissue exactly like the originating cell.[5] The lack of plasticity that is unique to corneal limbal stem cells differentiates them from other stem cell types. It is also

what allows them to be relatively universal in their capacity to differentiate into a variety of tissues, including muscle cells, organ cells and vascular tissue.[6]

## On a Cellular Level

The stem cell network begins its differentiation during fetal development. Adult stem cells start out as embryonic stem cells—which are pluripotent—that is, capable of forming all cell lines and tissues necessary for the continued support to the body. In addition to the embryonic cells, science has now discovered techniques to revert adult stem cells back to a pluripotent state. These are known as induced pluripotent stem cells (iPSCs).[1,5,7]

## Clinical Signs of Disease

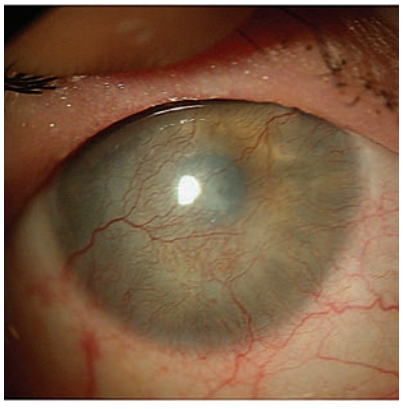
Corneal limbal stem cell disease (LSCD) can be classified as either primary or secondary.[12,13] Primary LSCD may be associated with conditions such as neurotrophic keratopathy, endocrine deficiencies and chronic inflammation.[12,13] It typically does not have observable etiologic factors.[12,13] Secondary LSCD occurs as a result of tissue destruction, such as in the case of contact lens wear, infectious disease (bacterial or viral), surgical trauma and thermal and ultraviolet injuries.[12,13]

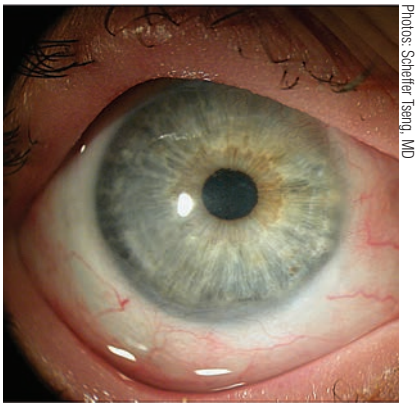
### LSCD Causes

Photo: Paul Karpecki, OD

This LSCD patient could benefit from stem cell therapy.

Etiologically, LSCD can derive from numerous clinical conditions. Inflammatory, infectious or traumatic sources can damage limbal cellular activity at a level which prevents normal re-establishment of physiology.[4,8-11] In addition to chemical burns and Stevens-Johnson syndrome, other causes of LSCD include contact lens-induced keratitis, limbitis, aniridia, multiple ocular surgeries, peripheral ulcerative disorders, chronic neurotrophic keratitis, pterygium, severe microbial keratitis, chronic bullous keratopathy and radiation therapy. [4,8-11]





Photos: Schefter Tseng, MD

**At left, this patient's LSCD was due to Stevens-Johnson syndrome. At right, a CLAL procedure (from her fellow eye) resulted in a healed, smooth corneal surface with no immunosuppression.**

This can present with several clinical signs, such as:[8-11]

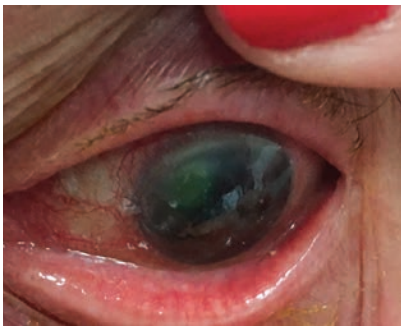
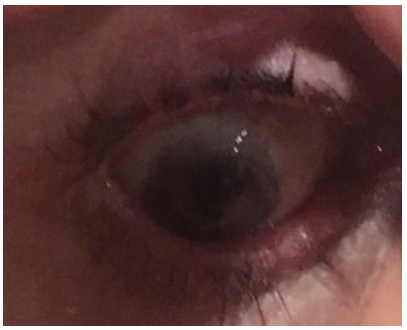
1. Irregular epithelial resurfacing that is specific to a segment of the cornea initiated at the limbus.
2. Sodium fluorescein, lissamine green and rose bengal staining of the cornea in a stippled and sometimes whorl-like irregular pattern.
3. The development of corneal erosions or ocular surface inflammatory disease with possible changes in matrix metalloproteinase-9 findings.
4. Corneal opacities.
5. Neovascularization.

Clinical signs and symptoms of LSCD typically include photophobia, injection, discomfort and pain.[14] Neovascularization is frequently present in chronic cases typically associated with inflammation.[14]

While the diagnosis of corneal LSCD is usually confirmed by clinical signs and patient symptoms, doctors can also employ technologies such as impression cytology and histopathology to look for specific cellular changes relative to LSCD.[14] Doctors can also biopsy tissue at the limbal margin to determine the underlying cause. Fluorophotometry looks at the dysfunction of the tissue at the limbal margin from a physiologic perspective.[15]

## Treating Limbal Stem Cell Disease

Typically, clinicians don't test for LSCD. More commonly, they treat the underlying issue and expect resolution. These treatments usually include cycloplegia and topical NSAIDs for analgesia, topical antibiotics to mitigate infective processes and steroids to mitigate inflammation. When these don't work, clinicians can suspect LSCD. Research shows the use of topical

**This LSCD patient initially presented with 20/400 visual acuity with pinhole.**

steroids may be effective in mild cases.[16-18] Low-dose topical steroids can be applied on a chronic basis, so long as appropriate follow-up is instituted to monitor for their possible complications. Research also shows other agents, such as cyclosporin A and Xiidra (lifitegrast, Shire), can be effective along with nonpreserved tears and ointments.[16-18]

***Mild LSCD.*** The typical rehabilitation timeline for patients with mild LSCD can be anywhere from weeks to months, or longer, and may need to be accompanied by debridement of the corneal epithelial tissue to remove the migrated conjunctival epithelial cells.[16-18] This is best accomplished with a classic debridement technique and placement of an amniotic membrane to enhance surface healing and to encourage normal repopulation of the limbal margin.[16-18]

Once the epithelial surface has regenerated, the underlying cause resolved and the limbal margin returned to a normal population, the condition resolves.[16-18]

***Severe LSCD.*** In patients with more serious presentations, limbal stem cell transplantation surgery can maximize outcomes. As a part of the preoperative assessment, review the periorbital tissue to assure that lid hygiene is optimal and both anterior and posterior blepharitis are treated. Clinicians also need to identify and treat abnormalities of lid position, such as ectropion and lagophthalmos.[8,19,20]

An additional risk includes glaucoma, due to the long-term need for topical steroid use in patients who have undergone limbal stem cell transplantation. Lotemax (loteprednol etabonate, Bausch + Lomb) in this setting is a reasonable treatment to avoid intraocular pressure (IOP) elevation, and is one that can be implemented to minimize risk of steroid-induced IOP rise. Toxicity from topical therapy (steroidal or anti-glaucoma) may decrease success in surgical intervention. This can be mitigated with the use of nonpreserved topical medications, selective laser trabeculoplasty or micropulse laser trabeculoplasty prior to initiating treatment. These procedures are designed to remove the need for chronic topical therapy or at the very least, decrease the dosage levels.

## Surgical Limbal Stem Cell Transplantation

Limbal stem cell transplantation can be accomplished through several modalities.[1,21,22] These include:

1. Conjunctival limbal autograft (CLAU), which involves harvesting healthy donor tissue from an unaffected fellow healthy eye.

2. Conjunctival limbal allograft (CLAL), which uses a donor-matched tissue from a relative (typically indicated in cases of bilateral disease).

3. Keratolimbal allograft (KLAL), in which the tissue is acquired from a cadaver donor.

The method most commonly employed for either CLAU or CLAL involves the harvesting of conjunctival tissue progressing forward to the limbal margin and moving into clear cornea.[23,24] This assures a full complement of limbal stem cells for transplantation. The tissue from the donor eye, or the healthy eye of the patient, is then transplanted to the recipient eye, which has been prepared by removing the damaged tissue to create a bed for the transplanted tissue.[23,24] The use of fibrin glues to secure tissue is the most common method of wound closure.[8]

In conjunctival autograft harvesting, the donor eye could potentially develop LSCD as a result of the surgical manipulation of the tissue and removal of the limbal cellular bed.[25] Identify any clinical complications to decrease trauma long-term to the donating eye, in addition to the treated eye, to enhance outcomes. Because the CLAU is a self-donated tissue, the patient faces no risk of immunologic rejection.[25]

## Allograft Tissue Replacement

When disease states are bilateral, the use of an autograft is not possible. In this circumstance, an allograft from a living relative donor is an effective alternative to cadaveric tissue, studies show.[19,20,26] Since allograft tissue has the same concerns as other donated tissue, the standards for excluding disease states, such as screening for hepatitis and HIV, needs to be implemented, and tissue which is not

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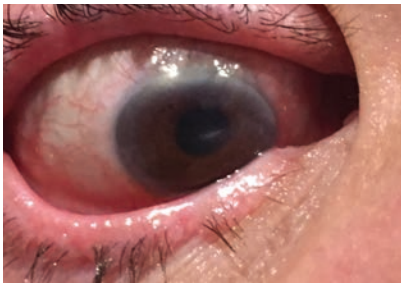
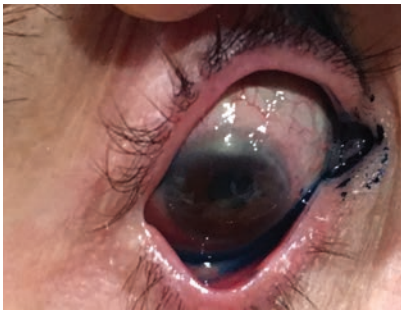


**This image revisits the patient from page 34. Two weeks after initiating limbal stem cell disease therapy, she was down to 20/100 with pinhole.**

healthy should be excluded.[19,20,26]

Allograft tissue replacement has the potential for rejection and requires topical and systemic immunosuppression therapy for long-term success. Because the procedure does not involve a complete 360-degree transplantation, it has limits in patients with a diffuse disease state (for example, burns or neurotrophic keratopathy). These patients are more likely to undergo cadaveric transplantation, while a living relative allograft is better suited for patients with specific zones of limbal stem cell damage.[27-29]

The third procedure, KLAL, which uses cadaveric tissue, was studied approximately two decades ago.[30] Investigators have since evolved the technique to its current level, which requires the preparation of the recipient tissue antecedent to the placement of the corneo-scleral transplant tissue. The donor tissue is prepared through a complex surgical procedure that involves the dissection of both conjunctival, limbal and corneal tissue in preparation for the placement. After placement of the donated tissue, the use of a fibrin glue to secure the transplant, along with the positioning of an amniotic membrane to accelerate the regeneration of surgical wound site, is typical. The success rate varies considerably with this technique, but authors have found that upwards of three-fourths of patients were successful on long-term immunosuppressive therapy, with a variety of agents.[30-32]



**Here is our patient again four weeks after her initial treatment. She is now at 20/40 with pinhole.**

Surgical intervention has been the primary method for addressing this complex problem, but new technology developed over the last decade may change that. The most recent advancement is the use of cultured corneal epithelial stem cells to replace the damaged tissue. This is accomplished with autologous stem cell systems, which eliminate the risk of rejection and the need for immunosuppressive therapy. We also have the ability to culture stem cells through generalized stem cell lines; however, because stem cell lines are specifically directed creates a greater level of difficulty for this technique. While this does not eliminate the need for anti-rejection therapy, nor does it completely block the body's ability to reject the graft, it does present an exciting methodology.

The first series of cases in this area monitored patients with autologous cultured corneal epithelial stem cell transplantations over a two-year period, and described a relatively low rate of complications and notable visual improvement.[31] In cases with more advanced disease states, the procedure was less successful, with less than 50% of the eyes achieving long-term stability without complications.[32]

## Comanagement of LSCD

Initial treatment of surgical limbal stem cell disease patients is not unlike the therapy currently used for other transplanted ocular tissue. Patients are placed on a course of topical antibiotics, along with steroids, and can take additional anti-immune system management drugs, such as cyclosporin A. Clinicians should minimize the use of any and all preservative-based compounds when possible, along with surgical intervention to address abnormal lid positioning or other concerns. These agents can be used for significant lengths of time and may need to be indefinite for some patients to maintain optimized ocular surface status.[33] This presents real risks relative to the development of secondary complications, such as secondary steroid-induced open-angle glaucoma and ocular surface-related issues in the form of toxicity, and can cause degradation of surgical outcomes.[32] Rejection is always possible as both a short- and long-term concern. Depending on severity, oral immunosuppressants may need to be used adjunctively to topical therapy to minimize risk.[33]

It is rare in a clinician's lifetime to witness the birth of a new technology, but with today's stem cell advancements, we stand at the dawn of a true paradigm shift. Welcome to the future. ■

*Dr. Thimons is a founding partner with the Ophthalmic Consultants of Connecticut and an adjunct clinical professor at Salus University.*

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# Reinventing Glaucoma Therapy

The advent of sustained-release drug delivery systems may finally break through the compliance barrier. Here's a preview of what's coming next. **By Shira Kresch, OD, MS**

When the prostaglandin analog became an approved treatment in 1996, it changed the way we approached glaucoma management.[1] With its convenient once-a-day dosing and improved efficacy to decrease intraocular pressure (IOP), prostaglandins quickly became first-line therapy.

However, the increasing prevalence of the disease causes concern, and compliance barriers—long known as a significant hurdle in successful glaucoma therapy—have led to poor compliance with glaucoma medications.[2] In 2013, the global prevalence of open-angle glaucoma (OAG) in people 40 to 80 years of age was 3.54%, affecting nearly 64.3 million people.[3] This number is expected to rise to 76 million by 2020.[4] With less than one-third of glaucoma patients considered "highly compliant," blindness from the disease will inevitably increase and have devastating impacts on health care costs.[5]

Arguably, the most substantial barrier for patients is lack of communication, accounting for about 50% of the barriers identified in one study.[5] Barriers affected by poor communication between the doctor and patient include: disbelief of the necessity to take drops, misunderstanding or naiveté of the visual consequences of glaucoma, failure to acknowledge medication side effects and not receiving adequate reminders to take medications. In addition, patient education often fails to cover the cost of medications, difficulty with traveling, hand tremor, forgetfulness, poor eyesight and poor hand-eye coordination.[6]

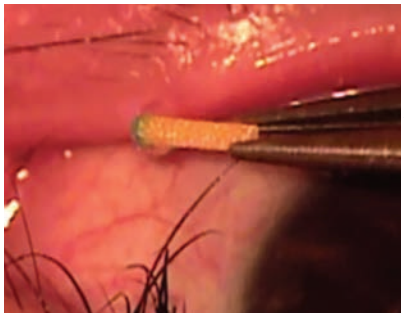

Photo: Ocular Therapeutix

**Fig. 1. This intracanalicular punctal plug from Ocular Therapeutix delivers travoprost to the ocular surface for two to three months.**

However, patients who understand they have a blinding disease, trust their doctors and receive adequate reminders to take their medications often have improved compliance. If a sustained-release drug delivery system was available, these barriers would no longer exist—and fortunately, several are on the horizon.

## Ocular Drug Delivery

On the ocular surface, several biochemical factors play into how and when an adequate amount of a drug reaches its destination without causing toxicity to ocular tissues along the way. Ocular barriers in the anterior segment that affect drug delivery include static barriers such as the corneal epithelium, corneal stroma and blood-aqueous barrier and dynamic barriers such as conjunctival blood flow, lymph flow and tear drainage. Metabolic barriers also affect proper drug delivery to the ocular structures.[7]

In order for a drug to demonstrate superior efficacy, a hydro- and

Photo: Mati Therapeutics

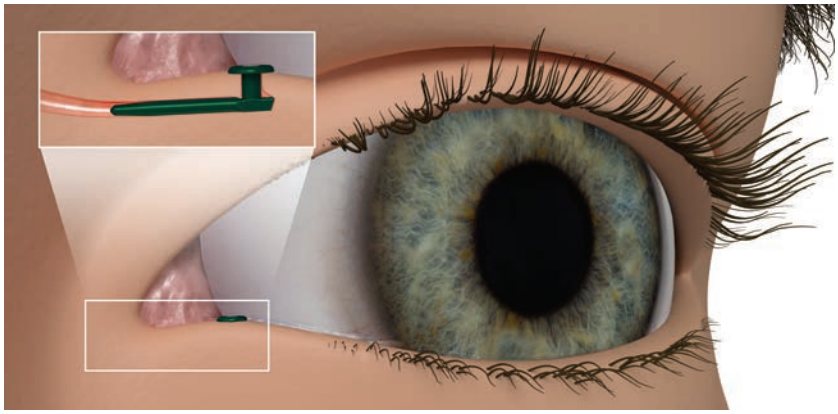

**Fig. 2. The Evolute punctal plug delivery system from Mati Therapeutics has been tested using latanoprost in patients diagnosed with OAG and ocular hypertension.**

lipophilic balance is essential. High ocular surface contact time is also an important aspect in topical formulations. Prodrugs, additives, pluronics, cyclodextrins and colloidal dosage forms have all been used to balance these factors in the drugs available today.[7] Although many effective glaucoma drugs are readily available, their ability to lower IOP diminishes as compliance suffers.

## Current Glaucoma Management

Today's mainstream glaucoma therapies include topical medications, laser treatments and surgical procedures. The Ocular Hypertension Treatment Trial Study, Early Manifest Glaucoma Trial, Advanced Glaucoma Intervention Study and Glaucoma Laser Trial have all been influential in determining when and how we treat our patients to adequately lower IOP for the longest duration possible in the most effective sequence.[3]

Topical medications, the primary approach for glaucoma management, have both advantages and disadvantages. They are minimally invasive, provide low absorption into systemic circulation, avoid first pass metabolism, are easy to administer and need a relatively small dose. However, they also have rapid drainage, interact with tear protein, are affected by tear turnover rate (1µL/min), and are rapidly removed due to reflex blinking and rapid metabolism.[7]

Selective laser trabeculoplasty (SLT) was a remarkable advancement that added an alternative for the treatment of certain glaucomas. FDA approved in 2001, SLT improved upon argon laser trabeculoplasty (ALT) and made laser treatments less damaging to ocular tissue.[8] SLT has proven effective as a primary or adjunct treatment in OAG with minimal side effects and complications.[9,10] However, SLT has some disadvantages. Many patients still need to remain on drops after treatment, the procedure often needs to be repeated and data on its long-term effects is lacking.

Surgical options have vastly improved with the introduction of minimally invasive glaucoma surgery (MIGS) into clinical practice. The iStent (Glaukos), CyPass (Alcon), Xen gel stent (Allergan) and Trabectome (NeoMedix) have provided glaucoma specialists with several surgical options that possess excellent safety profiles.[11,12] The biggest benefit of surgery is that it often is very effective in lowering IOP.[11,12]

However, any ocular surgery has significant associated risks, and patients remain fearful to undergo these procedures.[11,12]

## Rise of Sustained-Release

Although the concept of a sustained-release device has been around in eye care since the 70s, none have become part of mainstream ophthalmic treatments thus far.[7] Sustained-release medications are unique in that they would, in theory, target almost all non-compliance barriers.

Several companies are working on new mechanisms for sustained-release medications for many ocular diseases, including glaucoma—some of which show strong promise of implementation in the near future. Devices that deliver medication from outside the eye include punctal plugs, gel-forming drops and drug-eluting devices. Those that deliver medication from inside the eye include injectables and implants.[13] Here's a look at what's in the pipeline for these devices:

## Outside the Eye

***Punctal plugs.*** The OTX-TP (Ocular Therapeutix) delivers travoprost to the ocular surface via an intracanalicular punctal plug for two to three months, which then resorbs and drains through the nasolacrimal system (*Figure 1*). This plug is similar to a wine-cork design, allowing it to sit in the canaliculus without stressing the elasticity of the punctum.[14] Its advantages include being minimally invasive, containing fluorescein for retention monitoring, allowing comfort for most patients and clears the body through absorption—all while being preservative free. Studies show the therapeutic benefit has been significant for 90 days with a consistent 90% retention rate.[15,16] Phase II trials in South Africa and the United States, which suggest no serious adverse effects so far, shows a slightly less hypotensive effect when compared with timolol.[15,16] Phase III clinical trials in patients with ocular hypertension and glaucoma are underway.[15,16]

Images: Allergan

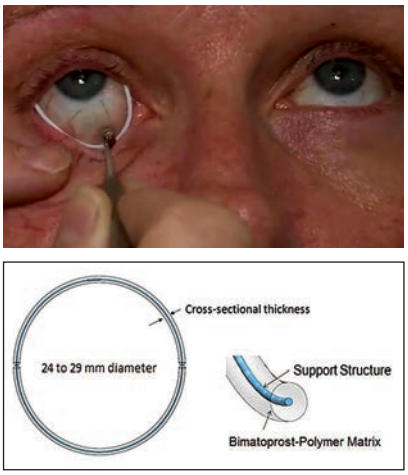


**Fig. 3. Allergan's drug-eluting periocular ring is currently in Phase II clinical trials.**

Mati Therapeutics has developed the Evolute (OR) punctal plug delivery system, which has been tested using latanoprost with OAG as well as with patients with ocular hypertension (*Figure 2*). The Evolute's drug core allows unidirectional sustained drug elution into the tear film, thereby minimizing systemic absorption.[17] The trials studied two different delivery doses and placement variations in the upper and lower puncta, both separately and simultaneously. So far, multiple multicenter US clinical trials indicate the lower punctum has retention rates up to 96% over a 12-week period.[17,18]

However, punctal plugs are foreign objects and have the potential to move, which may affect drug release and cause irritation or infection. Additionally, both companies are working to prove the intended drug is released at a constant rate for months at a time.

***Contact lenses.*** A recent study shows latanoprost-eluting contact lenses are at least as effective as daily administration of latanoprost ophthalmic solution eye drops when tested in glaucomatous monkeys.[19] This preclinical study was conducted with both low-dose and high-dose contact lenses via a thin latanoprost-polymer film inside a methafilcon hydrogel periphery lathed into a contact lens.[19]

***Other drug-eluting devices.*** Allergan recently acquired a promising drug-eluting periocular product that rests on the surface of the eye just underneath the eyelids (*Figure 3*). The results of a controlled Phase II study show that a single administration of the ring using bimatroprost provided sustained reduction of IOP of 4mm Hg to 6mm Hg for six months at the study's primary endpoint of 12 weeks. The retention rate was approximately 90% of subjects after six months.[20]

Although the company has plans for a six-month Phase III non-inferiority trial, this product is currently in its second multicenter, randomized, controlled Phase II clinical study to assess the insert over six months. A larger Phase II randomized, controlled trial has been completed.[16,21]

Amorphex Therapeutics' topical ophthalmic drug delivery device (TODDD) is made of a biocompatible soft elastomeric material that rests on the sclera underneath the eyelid (*Figure 4*). It can incorporate several different drops separately or simultaneously, including timolol maleate, prostaglandins, pilocarpine, brimonidine and even some anti-inflammatories and antibiotics.[22] The TODDD contains a large capacity for the delivery of medication for several months.[22] Obvious advantages include the improved efficiency and easy insertion and removal. The placement and replacement is fairly simple, takes less than a minute and can be done by patients at home.

The TODDD's Phase IIa trials demonstrate uninterrupted therapeutic efficacy for 180 days in humans with safety, comfort and strong retention. It has also shown reduced IOP of more than 35% in a prostaglandin study using dogs. Amorphex has secured a regulatory path with the FDA to gain approval.[22,23]

Although nanoparticle crosslinked collagen shields are still in development, a new study shows a pilocarpine sustained-release delivery using a nanoparticle shield was a safe and promising option in formulating a sustained-release treatment.[24]

***Gel-forming drops.*** These use pentablock copolymers as a vehicle for topical and intraocular drug delivery. Five polymer blocks are currently FDA approved for use in the eye. In the topical version, the intended drug is added to a non-viscous polymer drop. After touching the eye, this drop reacts with body temperature and transforms into a gel, which then lies under the eyelid, forming a film that releases the drug slowly until the polymer naturally degrades. Depending on the dose, the time course can be two, four or even six months.

DuraSite ISV-215 (InSite Vision) uses pentablock copolymers to form a stable mucoadhesive matrix of bimatoprost 0.03% that provides prolonged contact with the cornea, conjunctiva and other ocular tissues. DuraSite ISV-215 significantly improved the delivery of bimatoprost to rabbit eyes compared with Lumigan (bimatoprost 0.03% ophthalmic solution, Allergan).[25]

However, pentablock copolymers are subject to a "burst effect" in which 30% to 40% of the drug may release quickly on initial placement. So far, studies indicate DuraSite ISV-215 is resilient.[26]

Image: Amorphex Therapeutics

**Fig. 4. This topical ophthalmic drug delivery device (Amorphex Therapeutics) rests on the sclera, providing sustained drug delivery for several different drops separately or simultaneously.**

## Inside the Eye

***Injectables.*** Allergan's bimatoprost sustained-release implant, which is injected into the anterior chamber, is currently undergoing Phase III clinical trials. In Phase II trials, it lowered IOP in 92% of patients at four months and 71% at six months. So far, no serious adverse ocular events have occurred during the trials.[27]

ENV515 (Envisia) is also an anterior chamber injection, for which an extended-release formulation of travoprost is used in a biodegradable polymer drug delivery system. In Phase I clinical trials, IOP was reduced an average of 35% (6.4 +/- 0.6mm Hg) over eight months and then returned to baseline at nine months.[28] In Phase IIa clinical trials, IOP was reduced 28% (6.7mm Hg) at day 25, which was comparable with once-daily Travatan Z (travoprost ophthalmic solution, Alcon) in the other eye.[28,29,30] A Phase II trial shows that a single administration can result in a clinically meaningful decrease in IOP for the entire nine-month period. The only adverse effect noted in the trial was transient hyperemia.[30]

GrayBug is in the preclinical phase of developing an implant with a microparticle controlled-release drug delivery system using technology from the Wilmer Eye Institute of

Johns Hopkins School of Medicine in Baltimore. Three agents are under investigation: a single hypotensive compound, a dual action hypotensive compound and a hypotensive compound combined with neuroprotective properties. Testing in animal models showed successful decrease in IOP with subconjunctival administration. These initial studies showed no inflammation after six months, and the implant resorbed completely after six months.[16]

Clearside Biomedical and Santen are collaborating on supraciliary drug delivery in glaucoma via Clearside's microinjector and Santen's sustained-release formulations.[31] Injection into the supraciliary space puts the drug in close proximity to the ciliary body—the targeted tissue of most glaucoma meds. Researchers tested sulprostone and brimonidine with the delivery system, and both decreased IOP at a significantly lower dosage than their topical counterparts.[32,33] Brimonidine ophthalmic solution given as a topical formulation is 75ug three times per day, while the injected dose of brimonidine to the supraciliary space is roughly 100 times less than that. In the preclinical phase, a single administration of brimonidine injected into rabbit eyes reduced IOP initially by 6mm Hg; unfortunately, the effect slowly tapered off over a month.[32,33]

Currently in the preclinical trial phase, Ohr Pharmaceuticals has developed an injectable nanoparticle device to administer latanoprost in, on or around any portion of the eye for glaucoma, specifically steroid-induced glaucoma. Researchers hope nanotechnology can offer advantages to ocular drug delivery with improved consistency without corneal penetration. Additionally, adnexal adverse effects do not occur with this device. Some disadvantages include risk of toxicity, damage to intraocular structures and the need for lifelong procedures to ensure sustainability of decreasing IOP.[16]

Photo: pSivida

**Fig. 5. Durasert, develped by pSivida, is a bioerodible subconjunctival implant roughly the size of a grain of rice.**

***Implants.*** Icon Bioscience is developing a glaucoma device, IBI-60089, which is currently in preclinical trials. The goal is for a single injection of latanoprost to have a hypotensive effect for at least six months. Researchers are combining the drug with a carrier platform called Verisome into a true liquid injection. It degrades and eventually disappears as the active agent is released over time. The most evolved product at this time is called IBI-10090, which is meant for uveitis and postsurgical inflammation and is currently in Phase III clinical trials.[34]

pSivida recently invented a device for glaucoma called Durasert (*Figure 5*). This is a bioerodible, sustained-release drug delivery subconjunctival implant roughly the size of a grain of rice (3mm to 4mm in length, 0.4mm in diameter) containing latanoprost in a tiny translucent cylindrical polymer tube. The device would not need to be surgically removed and may work for three to six months, according to the company. A Phase I/II clinical trial is underway with subjects diagnosed with ocular hypertension to determine the safety and efficacy of the implant.[35]

Replenish has developed a smart device to be implanted in the sclera. This micropump has a programmable system designed to dispense nanoliter-sized doses of drugs every hour, day or month for up to 12 months. The current studies show a good safety profile in dogs without excess inflammation or scarring at 12 months.[14,36]

## Still Looking for Answers

Even if, or when, any of these devices become FDA approved, clinicians will still question whether patients will accept this change in glaucoma therapy. A review of available sustained-release systems in Singapore sheds some light on the possible adoption of such therapies, as patients preferred sustained-release devices over topical administration, with a special preference for punctal plugs. The same review also shows that 85% of patients were even willing to pay more for the sustained-release option.[13]

Long-term comfort and use of each device will largely be responsible for whether or not sustained-release medications are integrated into mainstream glaucoma care. Researchers will also have to determine if and when additional topical medications or laser and surgical procedures will be recommended after sustained-release instillation.

Although sustained-release devices resting on the eye should already be within optometric scope of practice, we as a profession will have to come together to fight for the right to insert and remove devices that are injected or inserted into the eye.

Even with so many unanswered questions, eye care providers can look forward to the day many of these sustained-release devices enter their clinic. With the prevalence of glaucoma increasing worldwide, causing a tremendous strain on the health care system, the improved compliance they could create would be hugely beneficial. Without ques-

tion, sustained-release devices have the potential to revolutionize glaucoma care in the near future. ■

*Dr. Kresch is an instructor in Optometric Sciences at the Columbia University Medical Center and is the pioneering optometrist in the glaucoma clinic and the general ophthalmology clinic of New York Presbyterian Hospital. Dr. Kresch is also the founder of a patient education website, www.eyeinquire.com.*


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PEER • REVIEWED • PRA • ARTICLE

# Eyesight to the Blind: The Promise of Retinal Prosthetics

The team that developed the Argus II implant explains the state of the art.

By Brian K. Do, MD, Mark S. Humayun, MD, PhD, and Hossein Ameri, MD, PhD

Devices that can restore vision lost to disease or injury have long been a staple of science fiction. The idea that technology might allow us to transcend our physical limitations captivates the imagination of scientists, doctors and the public at large. Despite many exciting breakthroughs over the last two decades, in 2017 such a device remains in the realm of fiction. But today, a number of patients are using a retinal prosthetic to regain some functional vision that helps them make their way in the world. The hard work and ingenuity of researchers, coupled with the pioneer spirit of early patients willing to undergo a radically new procedure, gives hope to everyone with a stake in the enterprise that one day functional vision might truly be restored.

We present here the technology of the Argus II retinal implant (Second Sight) from the team that made it a reality—and is now hard at work on future enhancements.

## The Argus I & II

Named for the mythical Greek giant with 100 eyes, the Argus is an epiretinal prosthesis that combines internally fixated electronics, with an external camera mounted on a pair of sunglasses.

The first-generation device, the Argus I, used cochlear implant principles and some components for an FDA Phase I clinical trial in 2002 during which the safety of long-term stimulation was demonstrated, making way for the more advanced Argus II.

Argus I was implanted monocularly in six participants blinded by retinitis pigmentosa (RP).[1-4] All participants perceived light once the device was activated and were able to perform visuospatial and motion tasks after brief training. Safety was observed in all instances of implantation during the study period; one participant had the device explanted for unrelated health reasons, and no evidence of tissue damage or electrode corrosion existed post-implantation at the five-year follow-up.[1-4]

**Here, an Argus II recipient is wearing the device.**

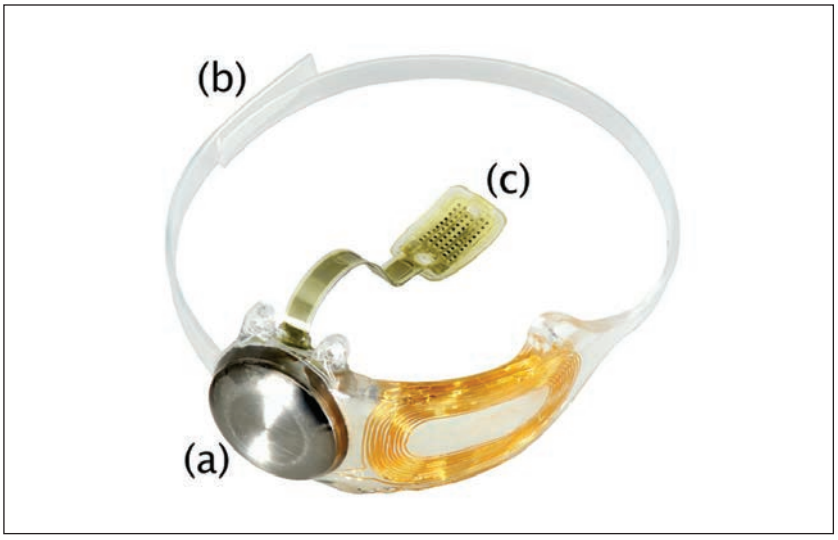
Prior to receiving FDA approval as a humanitarian device in 2013, the Argus II obtained European Union approval and since then has been implanted 150 times to date globally; this number is anticipated to increase in the coming years. The device has been used primarily in patients with profound vision loss

from RP in the United States, and, to a lesser extent, choroideremia and other retinal dystrophies.[5]

***Function.*** The Argus II retinal prosthesis system is intended to function as a surrogate for the degenerated outer retinal layers and photoreceptors, interfacing with and stimulating the intact inner retinal neurons. Studies conducted in postmortem eyes concomitant with the engineering of the first Argus device showed approximately 80% survival of the inner nuclear layer and 30% of the ganglion cell layer in the maculae of patients suffering from RP.[6] Nearly 90% preservation of these layers was seen in both the neovascular and atrophic forms of age-related macular degeneration.[7,8]

***Device components.*** The Argus II consists of both implanted and external components. The external portion of the system is comprised of a pair of glasses with a small camera mounted into the frame, which is connected via cable to a video processing unit worn on the belt or on a shoulder strap. The visual information collected by the camera (when the system is turned on) is received, processed and converted into a brightness map in real time by the video processing unit. Power and data are sent via a radio frequency link from an external coil on the glasses to a receiving coil, which is contained within a hermetically sealed enclosure secured to the eye with a scleral band and sutures.

Subsequently, an inbuilt application-specific integrated circuit (ASIC) in the electronics unit generates stimulus-appropriate electrical pulses, which are relayed via a metallized polymer connecting cable to a 60-channel microelectrode epiretinal array. The array comes into contact with the retinal surface, where it is placed over the macula and tacked to the retina with a custom-made, spring-tension, metallic tack (Second Sight), allowing transmission of the electronic signal originating from the external portion of the system.




**The implanted components of the Argus II Retinal Prosthesis System can be seen here: The hermetically sealed coil and electronics enclosure (a) is attached to the scleral band (b), as well as via a metallized cable to the electrode array (c).**

## Surgical Procedure

Here is a concise summary of the key steps of the surgical implantation of the device:

1. A 360-degree limbal conjunctival peritomy is performed.
2. The receiving coil is inserted under the lateral rectus muscle and extended into the inferotemporal quadrant; the electronics case is placed into the superotemporal quadrant.
3. The scleral band, to which the receiving coil and electronics case are attached, is passed under the inferior, medial and superior rectus muscles.
4. Suture tabs on the implant allow fixation to the sclera, and a Watzke sleeve (Labtician Ophthalmics) and mattress sutures or scleral tunneling secure the scleral band in the nasal quadrants.
5. Core and peripheral vitrectomies are performed.
6. A temporal sclerotomy of approximately 5mm is made to allow the introduction of the electrode array into the eye, after which the array is secured to the retina using a retinal tack. The sclerotomy is then sealed with sutures, and all other sclerotomies are closed.
7. An allograft consisting of either processed pericardium or aponeurosis (manufactured in France), donor cornea or autologous fascia lata is sutured over the implant to reduce the risk of irritation or erosion, and Tenon's capsule and conjunctiva are subsequently closed.[9]

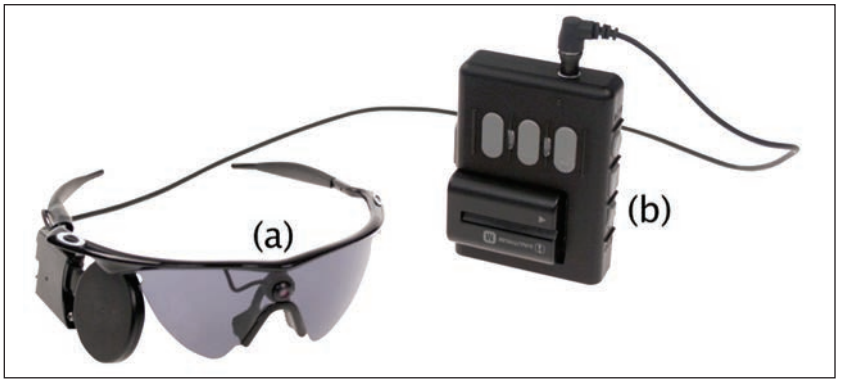
Surgical time generally falls between one and a half and four hours.

## Safety, Reliability and Complications

Between 2007 and 2009, 30 participants (29 with RP and one with choroideremia) received the Argus II implant in the United States and Europe.[10] The Argus II was

implanted in the worse-seeing eye of each patient. At five years post-implantation, two Argus II implants had failed, both because of progressive loss of the ability to maintain the radiofrequency link between the external antenna on the glasses and the receiving antenna implanted on the eye.[9] Both failed at approximately four years after implantation, but remained implanted to continue collecting long-term safety data.

Also at five years following implantation, recently published data indicate that 60% of patients—18 out of 30—had experienced no device or surgery-related serious adverse events (SAEs). Of the described SAEs (e.g., conjunctival erosion, hypotony, conjunctival dehiscence and presumed endophthalmitis), all were manageable with standard ophthalmic treatment approaches and no eyes were lost during the course of the study.




**The external components of the Argus II: (a) The camera mounted on a pair of sunglasses, connected by cable to the battery pack (b) and video processing unit.**

Between years three and five post-implantation, only one additional SAE was reported: One patient with a rhegmatogenous retinal detachment who remained stable for one year eventually underwent pars plana vitrectomy, epiretinal membrane peel, fluid-air exchange and injection of silicone oil once neovascular glaucoma was noted. The intraocular pressure returned to normal shortly after surgery and the retinal detachment resolved. One patient died at six years after implantation due to causes unrelated to the Argus II.[9]

During the first five years of follow up, a total of three explantations were performed. One implant was removed at 14 months due to

## Luminaries in Artificial Vision

While significant progress in treating degenerative outer retinal disease has been made in the past several decades, the scientific exploration that ultimately led us to the present day began as early as the mid 18th century. French physician Charles LeRoy created the sensation of light in the blind by passing electrical currents around the head in 1755.[25] Foerster discovered in 1929 that it was possible to elicit transient and reproducible visual percepts—phosphenes—with direct electrical stimulation of the visual pathway. In the 1960s, Button and Puttnam implanted four occipital lobe electrodes with percutaneous connections in three blind patients.[26] Using handheld photocells, which transmitted light-dependent signals to the occipital lobe electrodes, participants were able to roughly determine the location of illuminated objects.[26]

Other investigators have conducted important work in visual prosthetics, further showing that electrical stimulation of the visual cortex can create spatial visual percepts.[27,28] However, due to advances in alternative approaches—namely, retinal artificial vision prostheses as well as the significant associated morbidity—the cortical visual prosthesis has not yet reached medical implant approval status.

Twenty-five years of focused work on the epiretinal implant around the globe has led us to the Argus II. The original work leading to the advent of the Argus II implant's current iteration began in 1987. Dr. Humayun was a Duke medical student beginning his career in ophthalmology and struggling with patients' loss of vision, including his own grandmother, who suffered from diabetic retinopathy. Eugene de Juan, Jr., MD, a member of the medical staff and vitreoretinal surgeon at the Duke University Eye Center at the time, and Howard Phillips, a nuclear and electrical engineer, along with Dr. Humayun, comprised the three-person team that launched the research that eventually led to the Argus implant.

In 1991, Dr. Humayun's group reported the results of its retinal stimulation experiments in rabbits in which photoreceptor lesions had been chemically induced.[29] Shortly thereafter, groups at Harvard, MIT and in Cologne, Germany also began to use implanted electrodes to stimulate the visual pathway.[30,31] From 1992 to 1994, Dr. Humayun's group conducted the first human tests to evaluate epiretinal electrical stimulation. Studies began at the Duke Eye Center and continued later at the Wilmer Eye Institute.[32,33]

So began an intensive period of animal experiments, development of surgical technique and instruments, intraoperative patient testing and simulation studies of prosthetic vision. While at Wilmer, the intraocular retinal prosthesis laboratory obtained three important new collaborators: Gislin Dagnelie, PhD, for his knowledge of psychophysics and simulation studies, MD-PhD student Robert Greenberg and post-doctoral researcher James Weiland, PhD, from the University of Michigan for his expertise in electrode materials and neural stimulation.[34]

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recurrent conjunctival erosion.[11] An additional two patients requested explantation at three-and-a-half and four-and-one-third years post-implantation, respectively. The first request was due to recurrent conjunctival erosion, despite the absence of other device-related issues. The second patient experienced chronic hypotony and ptosis in the operated eye and chose explantation for cosmetic reasons. During each of the partial explantations, the cable was cut mid-vitreous, the sclerotomy sutured closed, and the extraocular portion of the device and attached portion of the cable were removed, leaving the electrode array tacked to the retina. No adverse events were noted following explanation.

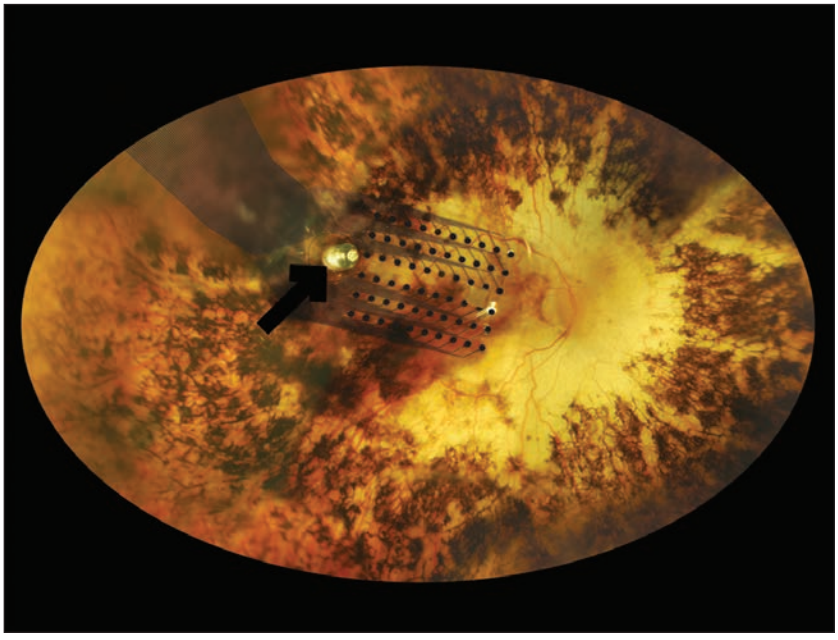
Another recently published study reported the outcome of six patients who received the implant in a single center, by a single surgeon. At 12 months follow up, no SAEs requiring further surgery, such as wound dehiscence, endophthalmitis or retinal detachment, were noted.[12]

More complete data on adverse events experienced by the 100+ patients implanted with the Argus II device since approvals in Europe and the United States are awaiting publication.

## Functional and Psychophysical Assessment

The vision afforded by today's retinal prostheses is quite limited. While some individuals are capable of significant feats of object and shape recognition, Argus II recipients experience visual percepts in a manner described most accurately as seeing "moving shadows."

The difficulty in quantifying visual improvement in these patients has presented issues with market approval by regulatory bodies. For instance, FDA guidelines have typically equated efficacy with measurable benefits, at least according to standardized outcomes (e.g., letter visual acuity, contrast sensitivity, visual field testing). While Argus II users have not shown these kinds of measurable improvements, the visual benefits become clearer when compared with patients with ultra-low vision—defined as hand movements, light projection or light perception—in whose cases a list of 760 distinct activities that can benefit has previously been elicited.[13]



**This fundus photograph shows the electrode array component of the Argus II, which has been fastened to the recipient's retina via the metallic tack (black arrow).**

The testing modalities used in both the Argus II feasibility study and the post-approval study include: (1) target localization, (2) motion direction discrimination and (3) grating visual acuity.[14-15] Further, researchers have shown a significantly improved ability to identify high-contrast shapes in 11 participants with the implant.[16]

Target localization was evaluated by assessing the ability of participants to scan a visual scene and point to a white square that appeared in random locations on a black screen. At three years post-implantation, 89% of the 28 participants were able to localize the target more accurately with the system turned on than with the system off.[10]

In testing motion direction discrimination, participants were asked to identify the direction of movement of a white bar across a black screen at a random angle, and 56% of the 27 participants were better able to do so with the system on than with the system off.[10]

Grating visual acuity, originally developed for a clinical trial of a subretinal photodiode array, measures the ability of an individual to differentiate the orientation of black and white gratings of varying spatial frequencies.[17] And while no participant was able to perform this test in any measurable capacity with the system turned off, 48% at one year and 33% at three years post-implantation scored 2.9 logMAR or more with the system on.

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## Future Developments

In the last decade, we have seen several different treatment modalities for degenerative retinal disease emerging in the form of clinical trials. Trials with gene therapy, stem cell transplantation and electronic neural prostheses implanted within different locations in the eye are all underway.[18-22] However, retinal prostheses are currently the only market-ready modality to date.

Only two retinal prostheses—the Argus II and the Alpha AMS (Retina Implant AG)—are approved in Europe, the US or both. However, modifications to currently available image-processing software will likely help existing prostheses garner greater benefits for patients. As it stands, image-processing software modifications have already been applied to improve the vision obtainable with the Argus II device. Various modifications have allowed improved edge-detection and image enhancement, which took place during early studies on shape and object recognition. In 2013, researchers presented new software that uses image magnification and minimization, as well as image enhancement features, to achieve resolution that seems to exceed the electrodes' limit. It allowed one Argus II patient to achieve visual acuity equivalent to 20/200 (logMAR 1.0) on grating acuity testing.[23]

Future devices are likely to have more and smaller electrodes. However, the potential additional benefit is still uncertain. The makers of Argus II have alluded to a next-generation device with 240 electrodes, with the possibility of the addition of peripheral electrodes.[24]

The ultimate goal: design devices with electrodes similar in size to the cell bodies of individual retinal ganglion cells, allowing individual cell activation.

Although more progress is needed, the ability to offer functional vision in those who otherwise would have had none is a remarkable feat in itself. The Argus II retinal prosthesis is the end result of years of hard work on the part of innumerable investigators who contributed their time, ideas and efforts. Further, were it not for the altruism of dozens of volunteers during the trials of both the Argus I and Argus II, this work would not have been possible. ■

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*Dr. Ameri is the director of the USC Retinal Degeneration Center and an assistant professor of Clinical Ophthalmology at the USC Roski Eye Institute.*

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Smart contact lenses might appear in your office sooner than you think. Here's what's ahead and how it might impact your practice.

**By Brian Chou, OD, and Jerome Legerton, OD, MS, MBA**

With today's exponential advancement of technology, contact lenses are evolving in much the same way telephones have over the past few years. The smartphone's role of making and receiving calls is now overshadowed by its ability to access the internet, provide real-time driving directions, take photos and video and summon transportation, just to name a few. In analogous fashion, contact lenses are now moving beyond simply correcting refractive error into the category of "smart contact lenses." For example, the Vistakon division of Johnson & Johnson Vision Care's product pipeline and patent activity reveals contact lenses designed to release an anti-allergy drug (with plans to file a New Drug Application in 2017) and a photochromic contact lens that adapts to the ambient light level, offering "glare and eyestrain relief."[1,2]

Perhaps the most profound applications will come from contact lenses embedded with microelectronic sensors, actuators and components to transfer energy and data. The miniaturization of electronics and advances in consumer electronics bring forth possibilities that once seemed like a moonshot. In vision correction, contact lenses may soon deliver adaptations that will transform the perceived world of both wearer and prescriber. Contact lenses will allow health monitoring and augmented reality, not to mention photo and video recording. In short, contact lenses will join the increasing web of interconnectivity, where devices collect data with sensors, allowing cloud-based analysis and real-time action.

Let's review what's in the works with smart contact lenses and discuss the implications for consumers and eye care professionals.

## Accommodating Optics

Presbyopia remains the basis for many consumers' need for eye exams, corrective eyewear and refractive surgery. Yet none of these modalities have managed to replicate natural accommodation with "autofocusing" optics. The common contact lens strategies for presbyopia degrade the quality of distance vision to improve near vision, through monovision or simultaneous vision multifocality. Thus, the prospect of an accommodating contact lens is quite attractive, and Google's startup, Verily, and Novartis hope to have a product to meet this need.

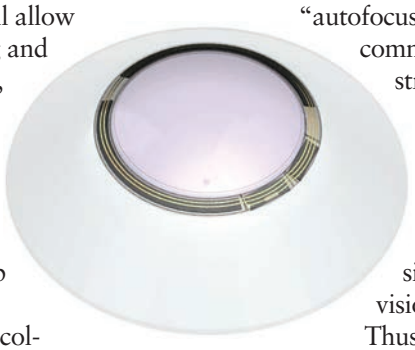


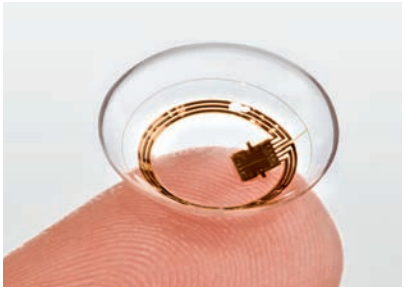
**Fig. 1. An accommodating contact lens presented by Johnson & Johnson Vision Care at its May 18, 2016 investor meeting.[1]**
Photo: Johnson & Johnson Vision Care

"We have developed a prototype of the accommodating contact lens—for complex, cutting-edge technology, the projects are progressing steadily," said Brian Otis, PhD, chief technology officer at Verily, in an exclusive e-mail interview in December 2016.

Johnson & Johnson is also making headway in this field (*Figure 1*). "In a newly created area, smart device technology, which combines contact lenses with sensors and microprocessors, we have already created a very strong portfolio: 82 US-issued patents and over 130 US pending applications," stated Peter Shen, PhD, worldwide vice president of Research and Design for Johnson & Johnson Vision Care at its May 18, 2016, investor meeting.[1] "Our smart device technology platform will enable us to explore opportunities to embed sensors, microprocessors, into contact lenses—very, very exciting opportunities." He also emphasized that the "first indication will be for the treatment of presbyopia."[1] A search of patents filed by Johnson & Johnson Vision Care suggests its smart contact lens for presbyopia will achieve variable focus using electro-active liquid crystal elements.[3,4]

eVision is developing an electrically-modulated contact lens which, according to Joel D. Zychick, president and CEO, "will be the only flexible contact lens, giving the end-user the ability to choose from a variety of prescription settings and to dynamically change focus while correcting for a variety of visual impairments, including myopia, hyperopia and higher-order aberrations." The company's first prototype was supposed to be available by late 2016.[5] Several attempts in November 2016 to obtain an update were unsuccessful.

Another accommodating contact lens concept has been developed by Hongrui Jian, PhD, and his team from University of Wisconsin, Madison, which was inspired by the eyes of the elephant nose fish. Jian's work, funded by the National Institutes of Health (NIH), involves a contact lens that uses tunable liquid powered by a solar cell that harvests and stores the energy within a network of nanostructures.[6]



Photos: Sensimed

**Fig. 2. Above, Sensimed's Triggerfish for detecting ocular dimensional changes. Top right, the lens on the eye. Right, sensor data is collected by an adhesive-backed antenna worn around the eye, connected to a portable data recorder.**

## Health Monitoring

Smart contact lenses are thought to be great tools for the detection and monitoring of biomarkers of disease since the tear film contains minerals, lipids and proteins.[7] In particular, contact lenses are emerging to monitor glaucoma risk and glucose control in a manner similar to how the Fitbit activity tracker can monitor and provide information on heart rate and steps taken.

Sensimed received FDA market clearance in March 2016 for Triggerfish, a single-use silicone contact lens embedded with a microsensor to detect tiny fluctuations in the eye's volume, capturing spontaneous circumferential changes at the corneoscleral area (*Figure 2*). Triggerfish is worn for up to 24 hours and wirelessly transmits ocular dimensional changes to an antenna placed around the eye with adhesive. A portable data recorder worn by the patient collects the information from the antenna and transfers it via Bluetooth to software installed on the practitioner's computer. The resulting data profile provides the practitioner with the time of day the eye's volume, which closely correlates with the patient's IOP, is highest and lowest.[8] Triggerfish does not, however, directly measure IOP. The product information states the technology is not intended to diagnose and is not for correcting vision.

While there doesn't seem to be immediate plans to commercialize Triggerfish in the United States, Sensimed is collaborating with a Japanese manufacturer and collecting clinical data on patients with normal tension glaucoma. Additionally, Sensimed is looking for a strategic partner in the United States to execute a planned prospective study comparing fast vs. slow-progressing glaucoma, according to an October 2016 company press release.[9]

Two separate efforts to commercialize a contact lens for continuous glucose-monitoring are underway. The target market is the 29.1 million with diabetes in the United States and the 387 million with diabetes globally.[10,11] The promise is to offer patients a noninvasive method, instead of a finger prick, to track glucose levels.[12] Although researchers still debate how well glucose levels in the tears correlate with blood glucose levels, it does not appear to dampen development attempts.[13] The first effort by Verily was announced in January 2014, and Novartis was announced as a partner in July 2014.[14]

"The smart lens projects, including the glucose sensing lens, remain active, and both Alcon and Verily are pleased with the progress of the collaboration to date," according to Dr. Otis of Verily. "Elements of the smart lens technology we are developing are in early clinical development. The tools we are developing are intended to passively monitor people's health-related behaviors so that they can more easily manage their conditions, rather than having to regularly check on themselves throughout the day. We consider these devices complementary to the patient-provider relationship, as doctors may have more data points to consider when making a diagnosis or considering treatment options."

Photo: Medella Health

**Fig. 3. Prototype of a continuous glucose-sensing contact lens.**

The second effort is by Medella Health, a Canadian start-up with $1.4 million in seed funding. In an exclusive phone interview, Harry Gandhi, CEO of Medella Health, said his team is developing a smart contact lens platform with a microchip, sensor, power and communication components (*Figure 3*). "Bio-sensing the tear film represents the low-hanging fruit of development compared to other applications of smart contact lenses such as augmented reality," Mr. Gandhi said. "We found, based on countless interviews, that consumers are willing to invest in their own preventative health."

## Augmented Reality

Last year's Pokémon Go craze, where Pokémon appeared on users' smartphone camera screens, highlighted the recent consumer drive toward augmented reality (AR). The National Football League has been using the computer-generated yellow first-down line for years now, and soon technology will be able to superimpose real-time directions on what you see while driving your car. This is the world of mixed reality (MR)—where digital content is placed over the normal vision content—and AR, where the digital content is registered and fixed to normal vision content. MR and AR are fast becoming their own form of reality, promising to deliver mobile content, including social media and streaming text and video, in your normal field of view in eyewear.

Many consumer electronics companies are chasing MR and AR, as well as virtual reality (VR), where the world around you is occluded. Smartphone accessories such as Google Cardboard, Zeiss VR and Samsung's Gear VR provide somewhat immersive VR experiences, as do the dedicated headsets such as Facebook's Oculus Rift, Sony PlayStation VR, HTC Vive and others. MR and AR offerings include Vuzix Blade 3000, Microsoft's HoloLens, Meta glasses, Osterhout Design Group's smartglasses system and Sony SmartEyeglass, to name a few. Yet the bulkiness, obtrusiveness, heat generation and small field of view with these devices are limiting. This is where smart contact lenses come into play.

Innovega recently completed a fourth round of clinical trials at the Ohio State University with the iOptik contact lens-enabled wearable display technology.[15,16] The soft disposable contact lens has a linear polarizer filter in the distance optical zone of the lens that blocks the near eye display light and a 1.0mm center microlens that focuses the display light in the lightweight display eyewear (*Figure 4*). "The eye-borne optics make a wide field of view, simultaneous viewing of the display and the real world, lightweight eyewear, low power consumption, high resolution, reduced vergence-accommodation conflict, and wide range of augmented, mixed and virtual reality eyewear configurations possible," said Steve Willey, Innovega CEO, in an interview. The company anticipates starting FDA clinical trials in 2017.

The first clinical use for the iOptik system is for patients with low vision. The NIH recently granted Innovega a Phase I Small Business Innovation Research grant to investigate the low vision application. A minimum field of view to deliver more than 4x image amplification and retain significant context is estimated at 50 degrees. Innovega has a prototype that delivers more

Images: Innovega

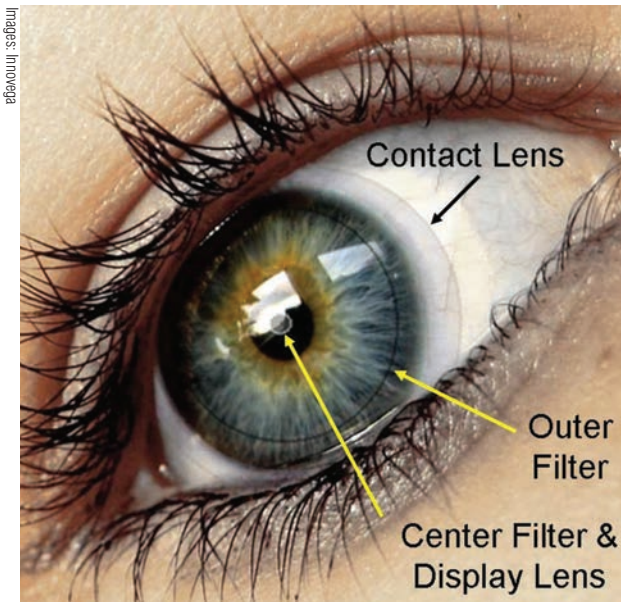



**Fig. 4. Above, Innovega iOptik's soft disposable contact lens with a linear polarizer filter in the distance optical zone for blocking the eyewear's polarized display and a 1.0mm center microlens that focuses the eyewear's microdisplay. Below, the iOptik eyewear for immersive virtual reality.**

than a 100-degree field of view using the contact lens. "The same contact lens-enabled systems can provide enhanced vision for fully sighted users," Mr. Willey said.

Samsung has been granted a patent in South Korea for a contact lens with a display that projects images into the user's eye.[17] The Gear Blink trademark, filed in both South Korea and the United States, may have to do with this smart contact lens and may involve blinking to control activation.

Night vision contact lenses, described by researchers at University of Michigan and Massachusetts Institute of Technology, are another form of MR and AR.[18,19] The concept uses graphene, a one-atom thick layer of carbon, to detect the infrared spectrum in combination with signal amplification. While the idea of night vision contact lenses is intriguing, significant research and development is needed.

## Photo and Video Recording

The rise of social media has placed increased value on the ability to capture photos and video through the user's point of view.[20] Here, contact lenses with photo and video recording could be the ultimate wearable.

Sony and Samsung each have patent applications for contact lenses that record what you see, and they both propose using the blink to control activation.[21,22] Samsung's patent application shows their lens is equipped with a tiny display, camera, antenna and several sensors to detect movement. An external device such as a smartphone is needed for processing.[17] Sony's patent shows a contact lens with a built-in camera, storage and transmission unit, as well as features including autofocus, zoom, aperture control and image stabilization. In all these cases, the patent applications describe product concepts rather than actual final products.

News reports suggest Google may also be pursuing such contact lens technology.[23]

## Implications

With so many innovations in the pipeline, smart contact lenses that enable accommodation, health monitoring, VR, MR, AR and photo and video recording may one day become the preferred platform to connect to our digital world. Until that time, many questions persist.

Will smart lenses for presbyopia finally crack the code of restoring natural accommodation? If so, we predict a resurgence in consumer and practitioner interest in contact lenses for presbyopia, significantly expanding the contact lens market.

Health monitoring wearables are ushering in a trend toward data-driven healthcare. While the initial efforts emphasize monitoring health status, health monitoring wearables may one day lead to data-driven diagnosis, which could replace the role of common physician functions in a manner that is more efficient, accurate and cost-effective to the consumer.[24] Still, it is not clear whether consumers will make bad medical decisions based on faulty interpretation.

For digital device users, much has been said about how our smartphones and tablets are distracting us to the point of sickness.[25] While most incoming messages are inconsequential, the reward of "good news" creates device addiction that can interfere with our personal relationships and true connection. The future user interface through mixed reality and augmented reality, if designed well, could minimize the unfortunate phenomena of being together yet isolated in our individual digital worlds.

The prospect of photo and video recording with contact lenses will create concern for their use in financial institutions, casinos, child-care centers, public restrooms, private social settings, test-taking centers and movie theaters, to name few. Some recording eyewear has tried to minimize this concern by implementing a recording light that turns on when it is in use. Yet, such a visual reassurance may not be possible with contact lenses.

Smart contact lens wearers might have to contend with security concerns as well, if the communication system is vulnerable to hacking or malfunction. For example, imagine the catastrophe for an accommodating contact lens wearer if full accommodation is unexpectedly activated while the user is driving at high speed. Or if video data is intercepted from a user's smart contact lens in the same manner that hackers take control of a laptop's webcam. These concerns are not insurmountable; yet, they underscore the importance of developing secure technology for smart contact lens wearers.

## Smart CLs in Your Office

Meanwhile, optometrists wonder what part they may play with smart contact lenses. With accommodating contact lenses, our integral role is obvious. For health-sensing contact lenses, Mr. Gandhi of Medella Health believes that optometrists in the United States will be pivotal in bringing patients onboard. "It's an exciting future because these smart contacts will create a closer working relationship between the optometrist and patient's primary care physician or endocrinologist," Mr. Gandhi said. Smart contact lenses with AR and photo and video functions will most likely require prescribing and progress visits by eye care practitioners. Additionally, these smart contact lenses may share a consumer-driven paradigm with cosmetic contact lenses, in which patients tend to request them from the doctor rather than doctors recommending them to patients. Dr. Otis said Verily's smart contact lens technology "has the potential to transform eye care, and we anticipate that both ECPs and consumers will play a role in its adoption."

The development efforts in smart contact lenses are significant and indicate a new era for patients and practitioners. Just as Alexander Graham Bell would be stunned by today's smartphone, Otto Wichterle, who developed the material for the first soft lens, would surely react the same way to smart contact lenses. ■

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*Dr. Legerton is an author, lecturer, inventor and consultant to the ophthalmic industry. He is a cofounder of SynergEyes and Innovega and has 52 issued US patents for contact lens technology including SynergEyes, Paragon CRT, myopia progression control, presbyopic laser refractive surgery and novel multifocal contact lenses.*


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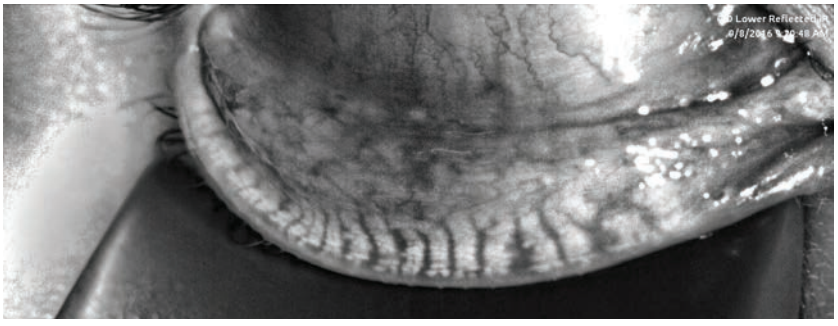
# Dry Eye: A Young Person's Disease?

The literature shows symptoms are no longer limited to older patients. Here's what that means for your practice. **By Whitney Hauser, OD**

Dry eye disease (DED) once had strict demographic considerations. Doctors could almost identify dry eye patients by simply glancing around a crowded waiting room without employing a single diagnostic test. Aging women with red eyes clutching a tissue in one hand and a bottle of tears in the other were the most likely sufferers. If they weren't picked out in the waiting room, DED patients could be easily discovered in the exam lane. Patients' medical histories told the tale. Diabetes, autoimmune disease, a laundry list of medications or prior eye surgery made them predisposed, and doctors knew it. While treating DED was never simple, patient identification was the most straightforward part.

Unfortunately, DED patients today can hide in plain sight. They can be men. They can be healthy. And they can be young. Though many would argue ocular surface disease has always been complicated because of its multifactorial nature, the demographic shift only adds to its complexity.[1] A survey conducted in July 2015 polled 1,000 eye care providers (ECPs) and 1,200 dry eye symptomatic adults.[1] According to ECPs surveyed, 92% suspected that modern technology contributes to dry eye symptoms and the incidence of DED is increasing due to "today's multiscreen lifestyle."[1] Perhaps the survey's most telling statistic: 76% of ECPs reported an increase in dry eye symptoms among patients 18 to 34 years old, relative to a decade ago.[1] That may not prove a definitive connection between digital device use and DED, but whatever the cause, an increase in DED is a call to action for optometrists.

This article looks at several cases of dry eye disease in younger patients, the likely instigators and how they can be successfully managed.



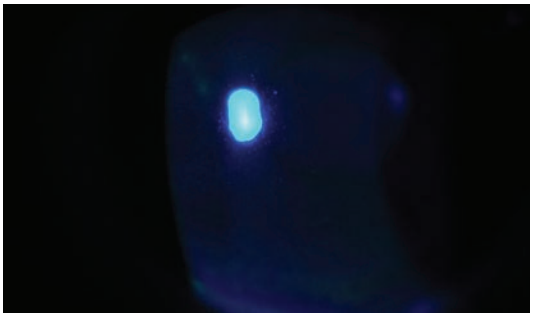


**Fig. 1. At top, a patient's meibography displays third degree gland atrophy.**
**Fig. 2. At left, a slit lamp examination of the same patient revealed diffuse superficial punctate keratitis throughout the cornea.**

## An Indigenous Disease of Digital Natives

Whether it's a computer or ever-present hand-held electronics, such as smartphones and tablets, patients of the Millennial generation (Americans born approximately between 1980 and 2000) are glued to digital devices for both work and play.[2] In our clinic, fewer patients these days are attributing their dry eye symptoms to traditional offenders such as contact lenses, environmental factors and aging. Anecdotal evidence suggests the culprit could be digital devices.

To wit, teens consume approximately nine hours of screen time every day on average.[2-4] They check social media outlets as many as 100 times per day.[4] With prolonged digital device use, blink performance is impacted.[5] When performing a visual task, research shows basal blink rate decreases by approximately 40%.[5] Furthermore, studies show that blinking while at rest is initiated by desiccation of the cornea and conjunctiva, but blink phenomenon affected by computer use appears to originate from a central neural mechanism.[5] A decrease in total blink rate with near activity has long been established. However, the importance of total blink number may be less significant than the completeness of each individual blink.[6] A 2013 study found that the tear film continues to be stable with decreased blink frequency as long as the blinks remain complete.[6] Incomplete blinks affected tear film stability and was variable with digital device exposure.[6]

The Tear Film & Ocular Surface Society (TFOS) offers a handy way to encourage blinking with its "Think Blink" campaign.[7] The organization suggests "every 20 minutes, close your eyelids and squeeze lightly for a count of two. Open and then look 20 feet across the room for 20 seconds."[7]

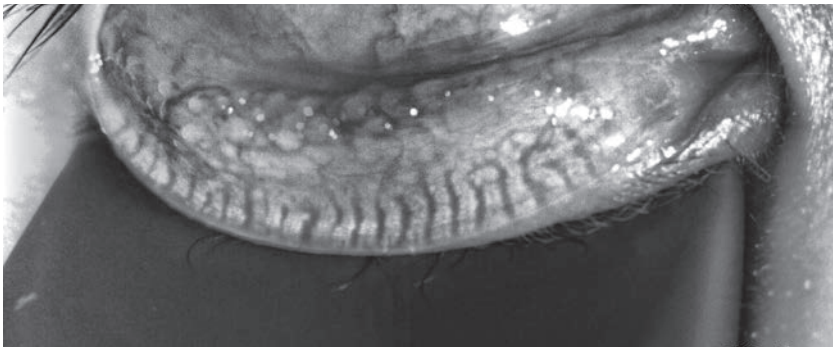


**Fig. 3. This patient's meibography shows a patient with first-degree gland atrophy indicating less than 25% gland loss via the Pult scale.**

In a busy practice, screening all young patients may be impossible and reap few benefits for patients or a practice. Identifying at-risk patients will streamline the diagnostic process. Consider evaluating for: presence of complaint (burn, irritation, blur increasing throughout the day), digital device time (prompting the patient to consider not only time on computers, but also tablets and smartphones), make-up application (liquid foundation, concealer, eyeliner and mascara applied inside the lash margin may pose increased obstructive risk) and medications.

## Testing

The ubiquitous nature of DED only complicates the lives of practitioners and necessitates additional diagnostic and point-of-care testing to identify this beneath-the-surface condition. If practitioners can no longer rely on demographics or symptomatology to point them in the right direction, additional objective testing is required. Point-of-care tests have the ability to add another piece to the diagnostic puzzle. For example, research found osmolarity accurately identified 81.3% of normal eyes and 90.7% of severe dry eye patients.[2] Perhaps the more critical statistic is that osmolarity correctly identified 73.2% of mild-to-moderate patients.[2] While the young, mild patient may be fairly complaint-free, the chronicity and, probably, progressive nature of DED necessitates education and, likely, treatment.

While awareness of functionality in the form of blinking exercises can be coached and behavior modified, the anatomy of young patients may also show vulnerability. Meibomian gland atrophy has long been noted in aging populations.[8] However, recent reports are identifying changes in a much younger demographic.[9,10] Papers presented at the 2016 Academy of Optometry found gland loss in a younger subset of patients within clinical practice.[9,10] Investigators found that early loss was equal between genders, unlike what we've seen in patients of previous generations.[9] Additionally, researchers recognized that contact lens wear (orthokeratology lenses) negatively impacted gland structure in the study.[9] A retrospective analysis found statistically significant differences among gender through average gland atrophy measured by the Pult score, which was 1.29 ± 0.81 for female subjects and 0.59 ± 0.58 for males.[10]

While additional study is required to identify risk factors for early propensity of gland atrophy,

initial clinical evidence suggests that young patient populations are not immune to gland loss and tortuosity.[10]

## Case #1

A 20-year-old biracial college sophomore presented with the complaint of chronic dry eye. Her symptoms included pain ranging from moderate irritation to sharp, intolerable discomfort and redness. She classified her symptoms as severe and they exacerbated with soft contact lens wear. The patient could only comfortably wear her lenses for four hours per day and often alternated the days to increase tolerability. Her ECP switched her from a bimonthly modality to daily lenses approximately six months ago. However, she did not experience significant improvement from the modification. She took no systemic medications and had no history of isotretinoin use.

Prior failed treatments included artificial tears, warm compresses, lid hygiene products, antibiotic drops and steroid drops. Her entrance acuity was 20/20- OD and 20/25 OS with spectacle prescription. Her SPEED and OSDI scores were 14 and 44, respectively. Tear osmolarity was 289mOsm/L OD and 301mOsm/L OS. The Inflammadry (Rapid Pathogen Screening) testing results were negative. She had 8/8 partial blinks upon analysis and a lipid layer thickness (LLT) of 61nm OD and 58nm OS. Her tear volume, collected by phenol red thread testing, was 30mm OD and 28mm OS. Her noninvasive tear break-up time was 6.31 sec OD and 5.74 sec OS, and tear prism height was 0.35mm and 0.33mm as measured by Oculus Keratograph. Meibography revealed third-degree gland atrophy indicating between 51% and 75% gland loss via the Pult scale (*Figure 1*).[11]

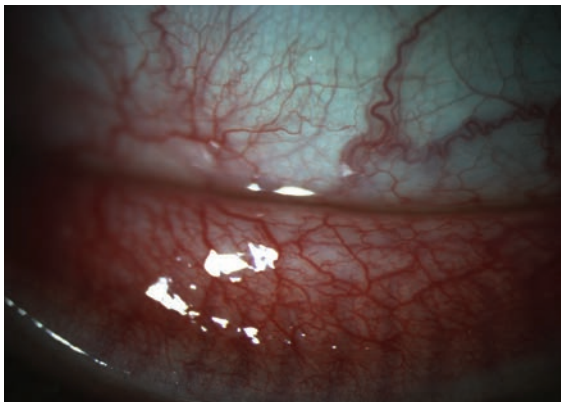


**Fig. 4. This patient's 3+ papillae at the inferior fornix could be visualized at both the slit lamp as well as on meibography.**

Slit lamp examination revealed diffuse superficial punctate keratitis (SPK) throughout the cornea (*Figure 2*). While the tear prism was ample, the line of Marx, or mucocutaneous junction, had modest shift anteriorly to a point of almost transecting the meibomian gland orifices. Mild conjunctival hyperemia was noted in both eyes. Upon expression, scant meibum was visualized though the quality of the secretions was clear.

Both pharmaceutical and in-office treatment options were discussed with the patient. She elected to first pursue medical therapy with consideration for future thermal pulsation. The patient was placed on Xiidra (lifitegrast 5.0%, Shire) twice daily, a lipid-based artificial tear and provided blinking exercises with instructions for use. She was advised to remove contact lenses before using the medication and to reinsert 15 minutes after administration if needed.

Upon returning four weeks later, she noted compliance with all recommended therapies and significant resolution of symptoms. Both the SPEED and OSDI surveys improved. However, though the cornea had improved, some diffuse SPK remained in both eyes.

## Case #2

A 23-year-old Asian male optometry student presented with a complaint of intermittent dryness with recent improvement in symptoms. His ECP prescribed Lotemax (loteprednol etabonate ophthalmic suspension, 0.5%, Bausch + Lomb) four times daily and artificial tears. He has worn orthokeratology lenses every evening for the past three years to improve daytime acuity secondary to moderate myopia. He felt the lenses slip some at night and reported days when the quality of vision was poor. He also had a history of hypothyroidism and daily use of levothyroxine.

Prior failed treatments only include artificial tear use. Entrance acuity was 20/20 OD, OS without correction. His SPEED and OSDI scores were 9 and 31, respectively. Tear osmolarity was 296mOsm/L OD and 292mOsm/L OS. Inflammadry was faintly positive. He had 7/7 partial blinks and a lipid layer thickness of 53nm OD and 46nm OS. The patient's tear volume was 30mm and 23mm. His noninvasive tear break-up time was 2.29 sec OD and 3.19 sec OS, and tear prism height was 0.24mm and 0.25mm. Meibography showed first-degree gland atrophy, indicating less than 25% gland loss via the Pult scale (*Figure 3*).

Slit lamp examination showed fine, diffuse SPK centrally OD and a mechanical keratitis OS due to the ortho-K lens because of the linear nature of the defects. We noted 3+ papillae at the inferior fornix

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in each eye that could be visualized at the slit lamp as well as on meibography (*Figure 4*). Expression of the glands was difficult and the quality of the secretion was slightly turbid in both eyes. The patient also exhibited lid wiper epitheliopathy nasally in both eyes (*Figure 5*).

The clinical presentation led to the diagnoses of both meibomian gland dysfunction (MGD) and acute ocular allergy. The patient began a beaded warming mask twice daily with digital massage, lipid-based artificial tears and blinking exercises. Lotemax was discontinued in favor of Bepreve (bepotastine ophthalmic solution, Bausch + Lomb) twice daily. Referral to patient's ECP was made to reassess contact lens fit. Due to the chronic, and, likely, progressive nature of MGD, more aggressive therapies were discussed with the patient in an effort to have a greater impact on the condition and mitigate advancement.

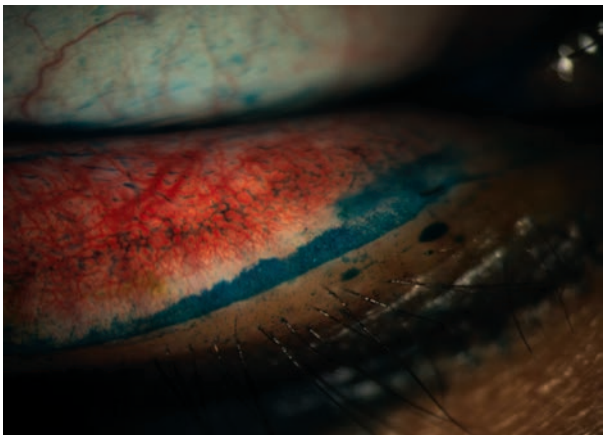

**Fig. 5. Staining revealed lid wiper epitheliopathy in a patient with dry eye symptoms.**

## Approach to Treatment

As cataract patients in their 90s may not get the same surgical recommendations as those in their 50s, so too will age play a role in the selection of dry eye recommendations. Doctors must consider the risk vs. the benefit. Equally, young patients likely face a long, winding path of DED. It is the doctor's responsibility to both identify the disease and best communicate its relevance to patients. Education is key to both a fruitful and long-term relationship. Frightening the patient and taking away their contact lenses will likely drive them to another practice. On the other hand, telling the patient "everyone has dry eye" and "it's not a big deal" will not properly convey the gravity of the condition. A balanced approach will arm the patient with the information they need to understand the disease and be compliant with recommendations. Because many of these patients will likely be in the early stages of the condition, a gradual treatment process from basic to more advanced is warranted. Consider a change in contact lens material, modality or care system, lid hygiene recommendations, blinking exercises or the appropriate artificial tear for supplementation. However, don't rule out more advanced in-office treatment options such as thermal pulsation or intense pulsed light. Earlier, more aggressive treatment may serve the patient better in the long run.

Dry eye disease was once a malady of aging women and often relegated to the lowest rung on the patient's assessment and plan.[1] Now, unfortunately, demographics of DED patients are trending younger, and doctors must broaden their minds to include this new subset.[1] While new sufferers will likely be emerging, practitioners are better armed than ever before with diagnostic options and therapeutic solutions. Perhaps awareness is the greatest challenge in initiating the dialogue between doctor and patient that ultimately kick starts the journey to improvement. ■

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# Are You Missing These Optic Nerve Disorders?

Understanding the pathophysiology of the anomaly is key to proper diagnosis.

**By Justin Cole, OD, and Jarett Mazzarella, OD**

When clinicians encounter an abnormal optic nerve, they must consider multiple differentials before reaching the correct diagnosis. Optic neuropathies can be caused by demyelination, inflammation, ischemia, infiltration, compression, toxic and nutritional causes, or can be hereditary. *Figure 1* depicts the wide variation in presentations of optic neuropathies.[1] Depending on the etiology, patients with optic neuropathy may present with loss of central visual acuity (VA) or central scotoma, changes in contrast sensitivity, a relative afferent pupillary defect (RAPD) (in unilateral or asymmetric cases), visual field (VF) defects, dyschromatopsia or eye pain. This article provides an overview of many common optic neuropathies and the diagnostic testing necessary to hone in on the proper diagnosis so clinicians are ready to tackle the challenge of identifying an optic nerve (ON) pathology.

## Patient History

Because many optic neuropathies present with similar findings, the patient's systemic health, demographics and ethnicity are key to the proper diagnosis. History of associated headaches, changes in mental state, reduced or altered hearing, muscle fatigue, temple pain, history of cancer or malignancy or presence of active infection can all be symptoms of a coexisting systemic condition(s). Knowing which conditions are associated with specific neuropathies can aid the clinician when faced with an abnormal ON. Finally, recognizing whether the anomaly is acquired or congenital and understanding the pathophysiology of the anomaly are integral to determining an accurate diagnosis (*Tables 1 and 2*).

## Diagnostics

Clinicians can rely on several diagnostic strategies to help differentiate acquired vs. congenital optic neuropathies:

***Visual acuity.*** The VA of a patient with ON pathology or ON anomalies can vary from normal vision to severely impaired. Assessing a patient's current VA, as well as looking at past history, can help in

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**Goal Statement:** When clinicians see an abnormal optic nerve, they must wade through a whole host of differentials before reaching the correct diagnosis. This article provides an overview of many common optic neuropathies and the diagnostic testing necessary to hone in on the proper condition so clinicians are ready to tackle the challenge of an unusual optic nerve.
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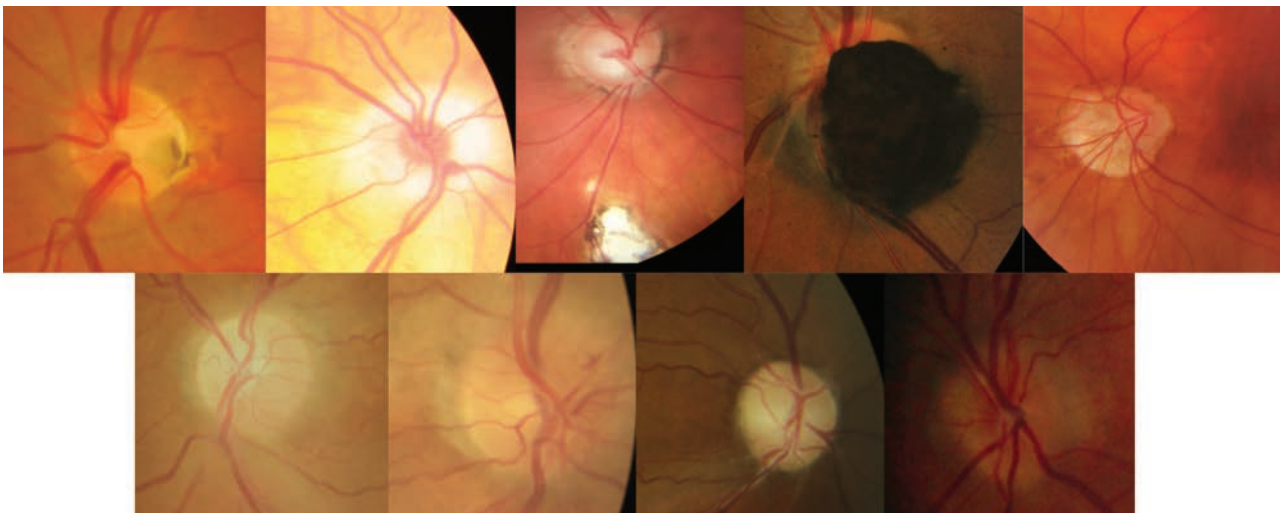


**Fig. 1. Collage of non-glaucomatous optic neuropathies. Top (left to right): ONH pit, hypoplastic nerve, ONH/retinal coloboma, melanocytoma, malinsertion. Bottom (left to right): Sectoral pallor, papillitis, optic atrophy, ONH drusen.**

the differential diagnosis, as long-term stability or normal VA may point to congenital conditions rather than acquired pathology, which are usually accompanied by acute or sudden vision changes. Clinicians must understand which ON conditions affect VA and to what degree to better educate the patient on potential visual recovery and long-term visual consequences of their condition (*Tables 3 and 4*).

***Pupil testing.*** This is crucial in determining functional vs. organic etiologies of VA loss, as well as distinguishing the exact cause of the optic neuropathy.[2] Research shows a positive association between VF asymmetry from automated perimetry and RAPD measurements.[3] However, this is not inclusive of all optic neuropathies, as some diseases of the ON are associated with relative sparing of either pupil function or visual function, as in Leber's optic neuropathy.[2]

Historically, the swinging flashlight test has been an accurate method of detecting a pupil abnormality, and clinical examination can typically elicit an RAPD sensitivity of three decibels.[2] Further accuracy can be obtained with the use of neutral density filters in front of the eye without the RAPD until neutralization of the pupillary response between the eyes is obtained.[2] When checking for an RAPD, clinicians must use a very bright light in a dark room to assess the full amplitude of the pupillary response.[1] In certain circumstances, anisocoria, reduced contrast due to dark iris color, iris anomalies or injury can lead to erroneous results.[4] The lack of light intensity standardization in successive tests can limit the clinician's ability to identify progression, stability or resolution of pupil findings over time. Also, in the presence of bilateral and symmetric optic neuropathy, an RAPD may be absent.

Recent studies demonstrate that pupillography may be as sensitive in detecting ON disease as other clinical findings such as VA, ganglion cell layer thickness or peripapillary RNFL thickness.[5] Pupillography provides more consistent duration and intensity of light compared with manual methods of pupil testing, thereby allowing clinicians to measure a stable and consistent light reflex.[4] Studies demonstrate RAPD amplitude scores can detect pupil defects with a high degree of accuracy at any stage of ON disease.[6]

***Color vision testing.*** Congenital color vision defects are often associated with a cone deficiency.[7] In contrast, acquired color vision deficiency occurs after birth and may fluctuate in severity depending on the etiology. When evaluating these patients with color plates, clinicians must assess whether the patient is correct in plate recognition as well as the speed and ease of recognition between eyes in cases where acquired color deficiency is suspected.[1] Defects can be difficult to define and may be different from right to left eyes. Acquired color deficiencies can occur from neuropathological events such as stroke or traumatic brain injury, neurodegenerative processes such as Alzheimer's and multiple sclerosis (MS), certain medications (ethambutol, amiodarone, methotrexate and cyclosporine), or chemicals (carbon monoxide and carbon disulfide).[1,7,8]

While congenital forms have a prominent male inheritance pattern and predominantly are red-green

**Table 1. Clinical Overview of Acquired/Pathological Optic Neuropathies[27-36]**

| Pathology | Demographics | Ethnicity | Systemic Health Association(s) | Systemic Symptoms | Ocular Symptoms | Ocular Exam Findings |
|---|---|---|---|---|---|---|
| **Non-arteritic ischemic optic neuropathy (ION)** | •Male=Female<br>•Average range 40 to 60, but can occur at any age | •Caucasian, African American, Asian >Latino | •Diabetes<br>•Hypertension<br>•Sleep apnea<br>•Hypercoagulable states | •Headache<br>•Rule out elevated blood pressure | •Sudden painless loss of vision or sudden loss of peripheral vision<br>•Dyschromatopsia | •Typically unilateral hyperemic ON edema (if acute) vs. pallor when chronic<br>•Small cup and potential rapid involvement of fellow eye<br>•Typically asymmetric ON edema (chalky) vs. pallor if neuropathy (longstanding) |
| **Arteritic ION** | •Median age ~75<br>•Female>Male | •Caucasian, African American, Asian >Latino | •Polymyalgia rheumatica | •Muscle pain<br>•Headache<br>•Jaw claudication<br>•Scalp tenderness<br>•~20% have no systemic symptoms | •Profound sudden vision loss<br>•Eye pain<br>•Amaurosis Fugax<br>•Dyschromatopsia | |
| **Optic neuritis secondary to multiple sclerosis** | •Female>male (3:1)<br>•Typically mid 30s | •Caucasian>all other ethnicities | •History of living in higher latitudes | •Headache, muscle aches or tingling in extremities | •Decreased VA<br>•Decreased contrast<br>•Possible double vision, pain with eye movements, and Uhthoff sign<br>•Dyschromatopsia | •ON edema, usually unilateral<br>•2/3rds of time normal appearing ON (retrobulbar) |
| **Optic neuritis secondary to *neuromyelitis optica*** | •2/3rds female>male<br>•Onset typically 40s (~10 years later than optic neuritis due to MS) | •Caucasian, African American, Japanese, South Asian | •Transverse Myelitis<br>•Myasthenia Gravis<br>•Systemic lupus erythematosus<br>•Sjogren's<br>•Celiac disease<br>•Sarcoidosis | •Headache<br>•Weakness/paralysis of arms/legs | •Decreased VA<br>•Decreased contrast<br>•Rule out double vision, pain with eye movements<br>•Dyschromatopsia | •Bilateral or unilateral ON swelling<br>•More frequently bilateral than with MS |
| **Papilledema**<br>**Secondary to idiopathic intracranial hypertension (IIH)** | •Any age<br>•Typically ages 20 to 45 in females; can occur in males and children | •No racial predilection | •Headache<br>•Nausea<br>•Alterations in mental state<br>•Obesity (IIH) | •Same as health associations<br>•Emesis<br>•Pulsatile tinnitus | •Can be asymptomatic<br>•Diplopia<br>•Cranial VI palsy<br>•Dyschromatopsia | •Bilateral ON edema, with/without parapapillary hemorrhages |
| **Nutritional/ toxic optic neuropathy** | •Nutritional: more often in adults due to alcohol and smoking<br>•Toxic: more common in children taking medications due to systemic conditions | •No racial predilection | •History of alcoholism<br>•Smoking<br>•Diet deficient in vitamins B and A<br>•Ethambutol, chloramphenicol, isoniazid, digitalis, amiodarone, disulfiram | •Possible nutritional or polypharmacy | •Decreased VA | •ON pallor/atrophy, usually bilateral, +/-APD |
| **Compressive optic neuropathy** | •Neurofibromatosis Type 2 (NF-2) children<br>•Neoplasm at any age<br>•Thyroid eye disease: female >male | •Depends on etiology; Thyroid eye disease: Caucasian> African American > Asian | •NF-2<br>•History of neoplasm of orbit, pituitary or anterior visual pathway<br>•Thyroid eye disease | •None | •Decreased VA<br>•Dyschromatopsia<br>•Variable insidious VF defects depending on location<br>•Paracentral, central, hemianopic, bitemporal scotomas | •Asymmetric ON pallor<br>•Sectoral pallor |
| **Infiltrative optic neuropathy** | •Any age | •Depends on etiology: sarcoidosis more common in African Americans | •History of non-Hodgkin's lymphoma<br>•Sarcoidosis<br>•History of metastatic cancer | •Dependent on etiology, recent infection vs. history of malignancy | •Decreased VA<br>•Dyschromatopsia<br>•Sudden cecocentral, central, hemianopic, arcuate scotomas | •ON edema<br>•Possible retinopathy |
| **Primary open-angle glaucoma** | •Adult population older than 40<br>•Male>female | •African American > Hispanic, Caucasian, Asian<br>•Hispanics: highest prevalence after 80 | •No consistent agreement in systemic associations | •None | •No ocular symptoms in early disease state<br>•Possible decreased VA in severe stage | •Increased ON cupping vs. pallor with intact neuroretinal rim |
| **Traumatic optic neuropathy** | •Male=female | •No racial predilection | •History of head or facial trauma (motor vehicle accidents, falls, assault) | •Likely related to head or facial trauma | •Decreased VA<br>•Dyschromatopsia | •Evidence of ON swelling, avulsion, but may appear normal or atrophic in chronic cases |

defects, acquired color vision defects have an equal propensity in males and females and are predominantly blue-yellow defects when VA is not affected, as in early glaucoma.[7] In acquired cases when the papillomacular bundle is affected, as seen in toxic neuropathies, patients often develop a red-green deficiency.[9]

Current research suggests that mechanisms of acquired color vision deficiency due to ON or retinal diseases may be ill-defined. The patterns of acquired dyschromatopsias appear to be heterogeneously associated with the distribution of color vision in the central VF and the particular disease state. For example, patients with ION may have normal color vision if their central field remains intact. Interocular asymmetry and interocular differences in disease manifestations can lead to variances in the color vision defects within and between the two eyes in acquired dyschromotopsia.[10]

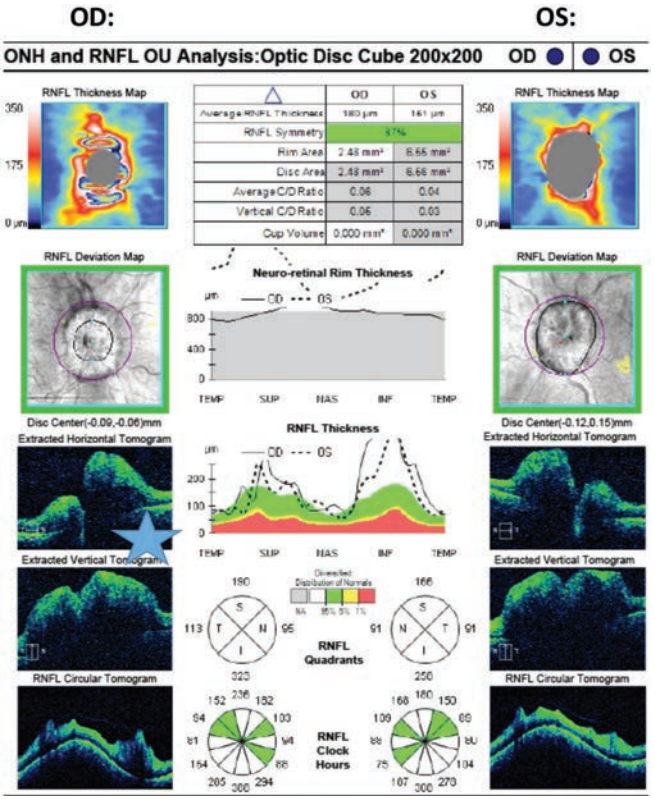
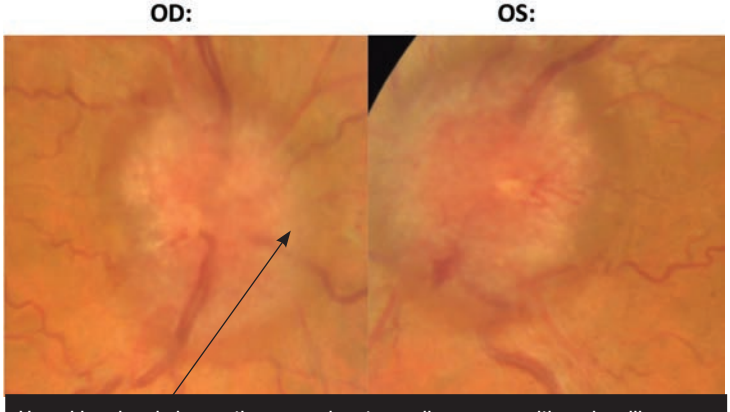



**Fig. 2. Cirrus SD-OCT pRNFL scan illustrating significant ONH elevation on B-scans and pRNFL thickening in all quadrants OU. Just superior to the blue star is the retinal nerve interface illustrated in the B-scan, OD, with subretinal fluid causing separation of the RPE layer from the neurosensory retina.**

***Optical coherence tomography (OCT).*** This technology is integral in the primary eye care setting for its ability to detect structural damage in the ON and macula. The clinician is able to detect and quantify the retinal and peripapillary B-scans with a resolution of three to five microns.[11] SD-OCT can be applied to identify an optic neuropathy but may be non-specific, not always providing the clinician the exact etiology of the disease state. This technology is also valuable in monitoring disease progression over time and in cases where intervention is prudent, the ability to evaluate the response to treatment.

For example, studies demonstrate that OCT RFNL analysis and ganglion cell-inner plexiform (GC) analysis can detect patients with glaucoma compared with the machine's normative database (which varies between OCT manufacturers) with a high degree of sensitivity and specificity.[12,13] Research on non-glaucomatous neuropathies, such as papilledema, has found a proportion of these patients develop subretinal fluid at the edge of the ON (lazy V sign), which can easily be defined by an OCT line scan (*Figures 2 and 3*).[14] In contrast, *Figures 4a* and *4b* illustrate the lumpy internal contour of the ON and the sharp demarcation at the ON's edge consistent with ON drusen. These tools can also be used to differentiate papilledema from more benign findings such as vitreopapillary traction, small crowded nerves, ON malinsertion and ON head drusen (*Figures 5 and 6*).[15]




Vessel bend and obscuration secondary to swollen nerves with peripapillary subretinal fluid. Indistinct nerve margins and vessel congestion noted OU.

**Fig. 3. These fundus photos demonstrate papilledema OD and OS.**

Neurodegenerative disorders such as MS, Parkinson's disease

**Table 2. Clinical Overview of Congenital Optic Neuropathies[37-40]**

| Condition | Demographics | Ethnicity | Systemic Health Association(s) | Systemic Symptoms | Ocular Symptoms | Ocular Exam Findings |
|---|---|---|---|---|---|---|
| **High myopia/ posterior staphyloma** | •Identified in early childhood | •Asian, Japanese > Caucasian | •Marfan's syndrome<br>•Stickler's syndrome<br>•Weill-Marchesani syndrome<br>•Down syndrome (Trisomy 21)<br>•Homocystinuria | •No direct systemic symptoms (high myopia can exist in Marfan's syndrome) | •May have decreased VA, but may be normal | •Tilted discs<br>•Circumpapillary myopic crescents<br>•Fuchs' spots<br>•Potential for dislocated lens (Marfan's) |
| **Dominant optic disc atrophy** | •1st to 2nd decade<br>•Autosomal dominant due to mitochondrial DNA mutation<br>•No sex predilection | •No racial predilection | •Associated deafness<br>•Cognitive impairment | •Possible neurological symptoms<br>•Auditory symptoms | •Slow decrease in VA<br>•Dyschromatopsia | •ON pallor |
| **Leber's hereditary optic neuropathy** | •Onset ages 15 to 35<br>•Due to mitochondrial DNA mutation<br>•Predominantly males | •No racial predilection | •Heart conduction defects: Wolff-Parkinson-White syndrome<br>•Lown-Ganong-Levine Syndrome<br>•Long QT syndrome | If plus disease:<br>•Neurological defects<br>•Movement disorders<br>•Brainstem syndromes | •Slow progressive decrease in VA<br>•Dyschromatopsia | •ON hyperemia, telangiectasia, mild swelling (early)<br>•ON pallor (late) |
| **Morning glory optic disc** | •Female>male (2:1) | •No racial predilection | •Transsphenoidal encephalocele<br>•Hypopituitarism<br>•Hypertelorism<br>•Basal encephalocele | •May have facial midline defects<br>•Mental deficits | •Normal to reduced VA<br>•Possible flashes and floaters w/ retinal detachment | •Unilateral, ON pallor with radial vessels extending from disc margin 360 degrees |
| **Congenital optic nerve pit** | •None | •No racial predilection | •High myopia | •May have neurological developmental defects | •Normal to reduced VA<br>•Central scotoma<br>•Dyschromatopsia<br>•Metamorphopsia (those w/ serous retinal detachment) | •Often unilateral<br>•Gray-white pit within optic disc, usually located temporally |
| **Optic nerve hypoplasia** | •None | •No racial predilection | •Preterm birth<br>•Smoking or alcohol consumption during pregnancy | •Endocrine dysfunction<br>•Maternal drug use<br>•Midline abnormalities in bilateral cases | •Normal to reduced VA<br>•Longstanding central scotoma<br>•Dyschromatopsia | •Unilateral (asymptomatic) small disc; with "double ring" sign<br>•Can be bilateral |
| **Optic nerve coloboma** | •None | •No racial predilection | •CHARGE syndrome<br>•Walker-Warburg syndrome<br>•Goltz focal dermal hypoplasia<br>•Aicardi syndrome<br>•Goldenhar syndrome<br>•Dandy Walker malformation | •Heart conduction defects | •Normal to reduced VA<br>•Longstanding central scotoma<br>•Dyschromatopsia | •Usually inferior optic nerve<br>•May have iris coloboma |

and Alzheimer's can also manifest structural changes in the eye. Current OCT software can segment specific retinal layers, and future technology aims to define individual cell types—possibly enabling OCT to define biomarkers for early detection prior to the development of debilitating symptoms.[16]

While this technology allows early visualization of structural changes, there are limitations. A scan's reliability can be affected by artifacts, such as media opacities and excessive movement, aberrations due to

pupil size, or even the failure of the software to properly define structural boundaries.[17] Also, patients are not limited to one disease state, so individuals with concurrent ocular pathologies may have less accurate OCT measurements, specifically in GC analysis.[18]

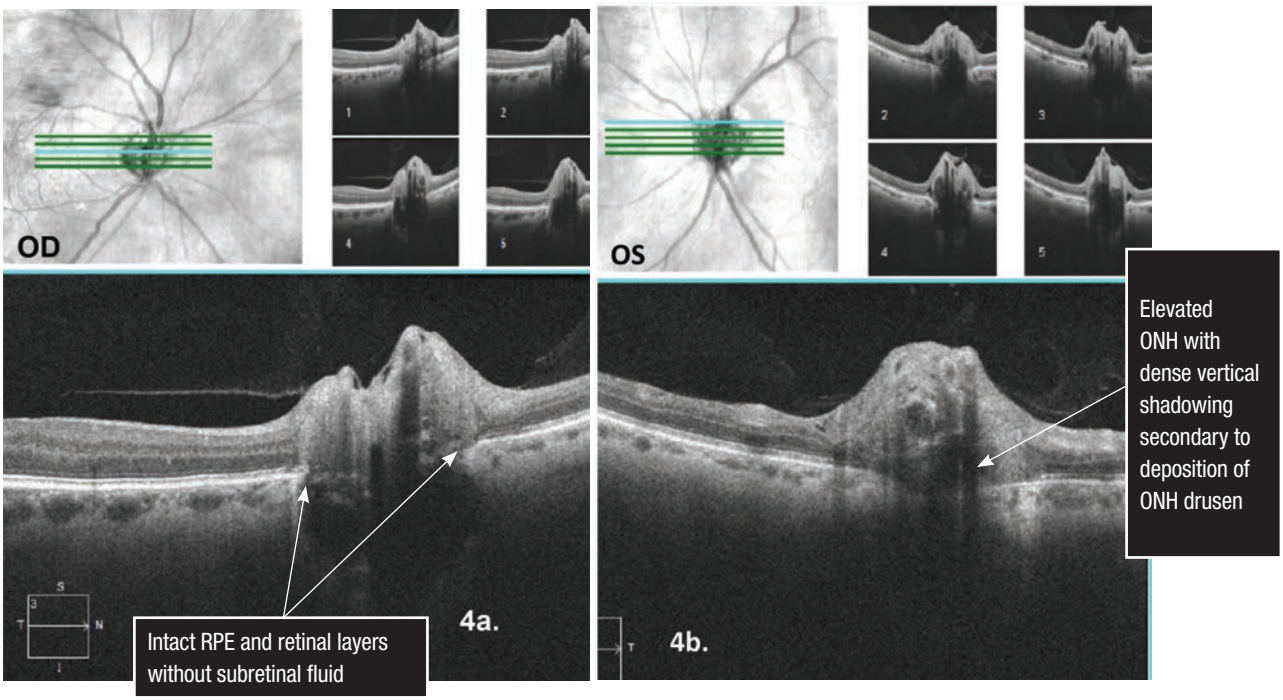
***Visual field testing.*** The time of onset of the VF defect and its location can aid in determining the etiology of an optic neuropathy (*Table 5*). If the onset of the VF loss is sudden, a clinician is more apt to think of inflammatory, ischemic, traumatic or demyelinating etiologies. If the VF loss occurs more gradually over time, the differential should include a compressive lesion, nutritional or toxic neuropathy, inherited neuropathy and glaucoma.[1] Although VF defects are not specific for a particular optic neuropathy, patterns emerge that can aid in the proper diagnosis. When the location of the defect is cecocentral, toxic, nutritional and hereditary etiologies must be considered. If a clear altitudinal defect exists, ischemic causes should be investigated. If an arcuate or Bjerrum scotoma is found, a glaucomatous etiology should be explored. A defect respecting the vertical meridian should indicate possible lesions at or behind the optic chiasm, and a central defect in one eye with a corresponding "pie in the sky" defect of the opposite eye should elicit prompt workup for a junctional lesion at the chiasm.[1]

***Fundus photography/clinical exam.*** Retinal fundus photography is useful for documenting normal, swollen, pale or anomalous optic discs, as well as to monitor the ON for structural changes over time.[1] Optic disc cupping is often associated with glaucoma, but less commonly may be associated with non-glaucomatous neuropathies.[19] Studies have found evaluating the ON rim color, inspecting for rim ablation and correlating these structural clues with the degree of VF loss can differentiate if the optic neuropathy is glaucomatous or non-glaucomatous.[20] In glaucoma, cupping is greater than pallor, and pallor typically only occurs in very advanced stages of the disease. When functional field loss is much greater than the structural cupping, the clinician must look at other neurological etiologies.

Finally, in early glaucomatous neuropathy there is a loss of neural

**Table 3. Pathological ON Conditions and Their Effect on VA[41-45]**

| Pathology | Visual Acuity Range |
|---|---|
| **Non-arteritic ION** | •Typically unilateral<br>•~50% patient will have VA in the range of 20/15 to 20/30<br>•Those with VA >/= 20/70:<br>* ~40% will improve<br>* 20% get worse<br>* 40% remain same<br>•Those with VA 20/70 or worse:<br>* 25% improvement in VA<br>* 15% worsen<br>* 60% remain the same |
| **Arteritic ION** | •Typically unilateral, but can become bilateral rapidly if untreated<br>•Entering VA loss can be more profound: counting fingers to light perception<br>•VA outcome can vary |
| **Optic neuritis secondary to MS** | •Usually unilateral<br>•VA: 20/20 to 20/30 initially<br>* can drop to less than light perception over days<br>•Vision worsens over two weeks, then gradually improves, but with sustained contrast sensitivity deficits |
| **Optic neuritis secondary to neuromyelitis optica** | •Usually unilateral, but more likely bilateral than with MS<br>•Acute severe VA loss >20/200<br>•Poor recovery<br>•~50% (>20/200) in one or both eyes five years after onset |
| **Papilledema** | •Bilateral<br>•VA: 20/20 to 20/400 depending on severity of swelling and etiology |
| **Toxic optic neuropathy** | •Bilateral, can be asymmetric<br>•Progressive<br>•Painless loss of vision |
| **Compressive optic neuropathy** | •Unilateral<br>•Slow, insidious onset, depending on etiology<br>•Depending on location and size, can present with range of VA |
| **Infiltrative optic neuropathy** | •Unilateral or bilateral with asymmetry; depending on etiology<br>•Typically poor VA; 20/400 to light perception with guarded visual recovery |
| **Primary open-angle glaucoma** | •Typically bilateral<br>•Can be asymmetric<br>•Normal VA in early stages of disease; however, in severe/end-stage disease, can have total loss of VA |




**Figs. 4a and 4b. 5 line raster Cirrus SD-OCT scans over the optic nerves illustrating optic nerve head drusen.**

rim tissue. In case presentations where pallor is present without rim notching or loss, clinicians should suspect non-glaucomatous neuropathies. In manifestations of optic neuropathy where disc hemorrhages or exudative retinopathy are present, the clinician should consider other etiologies such as ischemic, inflammatory, infectious or infiltrative disease. If the ON looks pale with a chalky appearance, arteritic ION must be considered. In clinical presentations where the ON demonstrates temporal pallor, toxic or nutritional neuropathy needs to be ruled out.[1]

In evaluating the ON for glaucoma, clinicians should follow the ISNT rule—normal ONs typically show a larger rim width Inferior, Superior, Nasal and Temporal. Although this rule is sensitive for glaucoma, it is not specific for a glaucomatous neuropathy.[21] Another finding to consider when evaluating the ON for a glaucomatous process is assessing for focal enlargement of the cup, typically greater in the vertical dimension. Excavation of the cup of the ON with the presence of laminar dots, RNFL defects, Drance hemorrhages or beta peripapillary atrophy also may point to a glaucomatous process.[22] In non-glaucomatous neuropathy, the ON is typically pale, may have more diffuse rim loss, the field defect does not necessarily correlate with the structural appearance of the ON and peripapillary atrophy is less common.[23]

In younger patients with normal eye pressure, reduced VA or VF defects respecting the vertical meridian, clinicians should investigate for

**Table 4. Congenital ON Conditions and Their Effect on VA[37,39,46-48]**

| Condition | Visual Acuity Range |
|---|---|
| **High myopia/posterior staphyloma** | •Normal to slightly reduced VA (best corrected) |
| **Dominant optic disc atrophy** | •Bilateral<br>•Range from 20/20 to light perception; 80% better than 20/80 |
| **Leber's hereditary optic neuropathy** | •Bilateral<br>•VA usually worse than 20/200 |
| **Morning glory optic disc** | •Unilateral<br>•Rare, but can be bilateral<br>•VA can range from 20/20 to counting fingers |
| **Congenital optic nerve pit** | •Unilateral; depending on presence of concurrent macular serous detachment; VA can range from 20/20 to 20/400 |
| **Optic nerve hypoplasia** | •Bilateral ~80% of the time<br>•Can be unilateral or asymmetric<br>•1/3 can have nystagmus<br>•VA can range from normal to NLP |
| **Optic nerve coloboma** | •Bilateral or unilateral VA can vary; depends on involvement of the fovea |

neurological associations.[23]

When evaluating any condition that relates to laterality, clinicians should look for comparisons and symmetry. Looking at the non-involved eye to distinguish an anomalous from normal anatomical variation is important. When evaluating the ON, this principle also applies, as tilted discs, ON hypoplasia, past ischemic events or ocular trauma can all lead to an asymmetry between the right and left ON, which can help to further define the etiology of the ocular pathology. Optic nerve size and RNFL thickness should be fairly symmetrical between the two eyes. In cases of larger or smaller appearing ONs, a measurement of the overall disc diameter is prudent.

***Lab testing.*** With unilateral optic disc swelling, a clinician must know when to consider blood work. In an adult population, ION is one of the more common optic neuropathies, and arteritic ION can be indicative of giant cell arteritis (GCA), which can be fatal. Therefore, clinicians should obtain a complete blood count (CBC), c-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) in cases of suspected arteritic ION based on clinical assessment. Knowing the expected normative values of these labs is also important. For ESR, the expected range is equal to twice the age of a male patient divided by two and twice the age of a female patient +10 divided by two. Elevated ESR, CRP and thrombocytosis (elevated platelet count) are positive predictors of GCA. Studies show elevated levels of CRP and the presence of thrombocytosis may be indicative of arteritic ION, even when the ESR is not elevated. When all three lab values are elevated, the likelihood of a positive temporal artery biopsy due to occult GCA is eight times more likely.[24]

**Table 5. Non-Glaucomatous Optic Neuropathy VF Defects[21]**

| Etiology | Location of Typical Visual Field Defect | Onset |
|---|---|---|
| **Demyelinating** | Central<br>Cecocentral<br>Arcuate | Acute |
| **Ischemic** | Arcuate<br>Altitudinal | Acute |
| **Inflammatory** | Central<br>Cecocentral<br>Arcuate | Acute/sub-acute |
| **Infiltrative** | Arcuate<br>Hemianopic | Acute/sub-acute |
| **Compressive** | Arcuate<br>Hemianopic<br>Bitemporal | Chronic |
| **Toxic/nutritional** | Central<br>Cecocentral | Acute/sub-acute/chronic |
| **Hereditary** | Central<br>Cecocentral | Dependent on inheritance |
| **Trauma** | Arcuate<br>Central<br>Hemianopic | Acute |
| **Radiation** | Arcuate<br>Hemianopic | Acute |
| **Neoplastic** | Central | Sub-acute/chronic |

Unilateral morning glory syndrome, ON hypoplasia, ON pits, ON coloboma, myopic/tilted discs and ON drusen generally don't require laboratory testing, as they are a clinical diagnosis.

In cases of unilateral ON pallor, and especially those with contralateral optic nerve swelling, and the presence of VF loss, neuroimaging of the head and orbits is indicated to rule out compressive or intracranial etiologies.

Bilateral ON edema, symmetrical or asymmetrical, is an ocular emergency. Differential diagnoses must be explored based on history, and neuroimaging, specifically an MRI of the head with contrast, is warranted. In suspected cases, magnetic resonance venography may also be completed to rule out sinus thrombosis. In cases where intracranial etiologies are ruled out by negative neuroimaging, a lumbar puncture can confirm the diagnosis of idiopathic intracranial hypertension when suspected.[25]

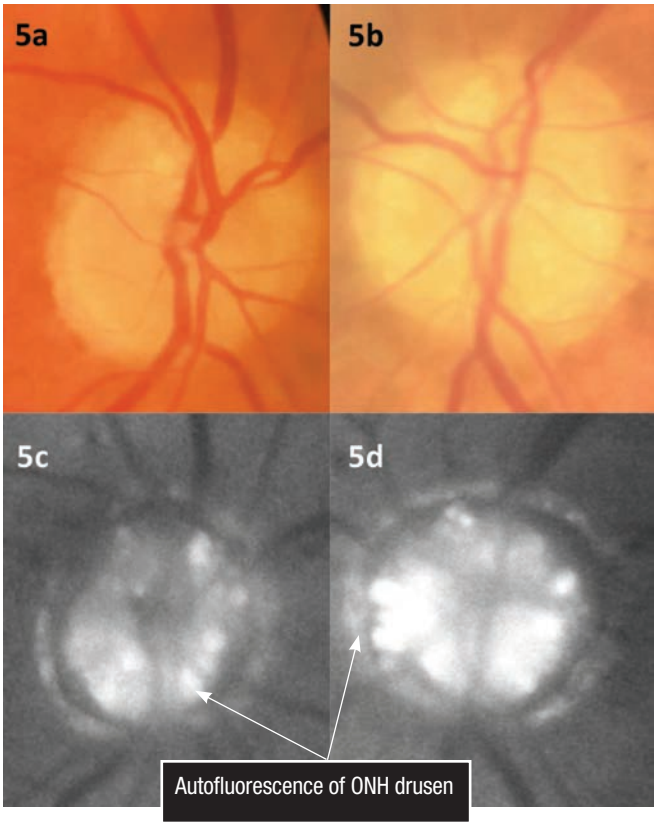
In other optic neuropathies, the clinical presentation can vary; in toxic, infiltrative or infectious etiologies it is usually bilateral; however, it can assume an asymmetric appearance. Patients with suspected malignant hypertensive retinopathy or diabetic papillitis should have their blood pressure evaluated and their hemoglobin A1c checked. Clinicians should order a CBC with differential in cases with a history or suspicion of blood dyscrasias or leukemia. During the case history, any radiation to the face, orbit or adnexa within the last five years should be addressed, as there can be delayed ocular sequelae. Referral to the primary care provider is necessary in suspected cases of ocular lymphoma or metastatic disease.

Angiotensin converting enzyme (ACE) and a chest x-ray are indicated in cases of suspected infiltration to rule out sarcoidosis. Clinicians should also consider tuberculosis, Lyme disease and syphilis testing for individuals who have potential risk

or history of exposure, especially those whose clinical profiles do not seem to fit the ocular signs and symptoms. It is imperative to review any past and current systemic medications that can contribute to optic neuropathy, and it is also prudent to obtain vitamins B1, B12 and folate levels in suspected toxic or nutritional optic neuropathies.

Bilateral optic neuropathies tend to have the worst visual consequence. Leber's hereditary optic neuropathy and dominant optic atrophy tend to occur early in life, and in some cases a cardiac workup is advised secondary to associated heart block. Lab testing for the presence or absence of the main mitochondrial mutations in Leber's accounting for most cases can be performed to confirm the diagnosis.[26] In all cases of hereditary optic neuropathy, the visual prognosis is guarded, and genetic counseling may be considered for the patient and their family.[27]

Optic neuropathies may manifest in various presentations, and as primary eye care providers, it is crucial to identify ocular conditions that can have long-standing health and visual consequences. By possessing the knowledge and the tools to identify the ON abnormality, clinicians can arrive at the correct diagnosis and provide timely and appropriate management. ■




**Figs. 5a and 5b. Color fundus photos illustrating ONH drusen. Figs. 5c and 5d. Fundus autofluorescence of the optic nerves.**

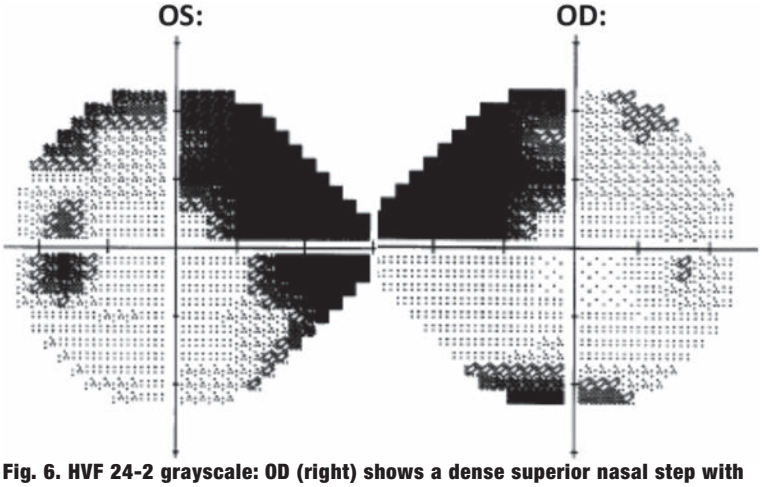



**Fig. 6. HVF 24-2 grayscale: OD (right) shows a dense superior nasal step with inferior defect. OS (left) shows a dense superior nasal step with associated incomplete arcuate formation as well as a dense inferior nasal step. Also noted OS is an enlarged blind spot.**

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1. Systemic health associations for non-arteritic ION include all of these, except:
a. Diabetes.
b. Polymyalgia rheumatica.
c. Hypertension.
d. Sleep apnea.

2. An RAPD sensitivity of ____ can be elicited by clinical examination.
a. 10dB.
b. 5dB.
c. 50dB.
d. 3dB.

3. In cases of optic neuropathy when the location of a VF defect is cecocentral, what etiology should most likely be in your differential?
a. Infectious.
b. Infiltrative.
c. Idiopathic.
d. Toxic/nutritional.

4. When VA is intact in cases of early glaucoma, what acquired color vision defect is most likely to occur?
a. Red-blue.
b. Red-green.
c. Blue-green.
d. Blue-yellow.

5. A central VF defect in one eye with a corresponding "pie in the sky" defect of the opposite eye should prompt the clinician to consider this etiology.
a. Toxic neuropathy.
b. Arteritic ION.
c. Junctional lesion at the chiasm.
d. Optic nerve head drusen.

6. The following medication(s) can be associated with acquired color vision defects:
a. Cyclosporine.
b. Methotrexate.
c. Ethambutol.
d. All of the above.

7. All of the following lab tests should be considered in suspected ischemic optic neuropathy, except:
a. ESR.
b. CRP.
c. ACE.
d. CBC.

8. Which lab test(s) have shown to be stronger positive predictors of arteritic ischemic optic neuropathy related to a positive temporal artery biopsy due to GCA?
a. Elevated ESR.
b. Elevated CRP.
c. Elevated platelets.
d. Elevated CRP and platelets.

9. What is the correct formula to calculate the expected level of ESR for a male patient?
a. Twice the patient's age divided by two.
b. Four times the patient's age divided by two.
c. Twice the patient's age +10 divided by two.
d. Four times the patient's age +10 divided by two.

10. What is the potential for visual recovery for a patient diagnosed with NAION with an entering visual acuity of 20/60?
a. Vision will return to 20/20 over next several months.
b. 40% chance vision will improve, 20% chance it gets worse, 40% chance it remains the same.
c. Vision will continue to decline over time.
d. Vision will return, but visual field will continue to decline over time.

11. In optic neuritis associated with MS, the presentation of optic neuritis is "retrobulbar" in presentation what percentage of the time?
a. 25%
b. 33%
c. 50%
d. 67%

12. When differentiating between optic neuritis from MS vs. neuromyelitis optica, what manifestation is most often associated with neuromyelitis optica?
a. Female preponderance one-third of the time.
b. Age of onset generally 10 years later than MS.
c. Associated headache.
d. Double vision and pain with eye movements.

13. What retinal condition can be associated with a congenital optic pit?

## OSC QUIZ

a. Intraretinal hemorrhages.
b. Serous macular detachment.
c. Fuchs' spot.
d. Angioid streaks.

14. What optic neuropathy is due to a mitochondrial DNA mutation?
a. Optic nerve coloboma.
b. Leber's hereditary optic neuropathy.
c. Dominant optic atrophy.
d. b and c.

15. What ocular finding is commonly associated with an optic nerve head coloboma?
a. Lens coloboma.
b. Iris coloboma.
c. Posterior embryotoxin.
d. Corneal guttatae.

16. What type of VF defect is most often pathognomonic for an ischemic optic neuropathy?
a. Cecocentral defect.
b. Altitudinal defect.
c. Nasal step defect.
d. Bitemporal hemianopsia.

17. When performing the swinging flashlight test, what can lead to erroneous results?
a. Anisocoria.
b. Iris anomalies.
c. Injury.
d. All the above.

18. Congenital color vision defects are often associated with:
a. Rod deficiency.
b. Cone deficiency.
c. Müller cell deficiency.
d. None of the above.

19. The following chemicals have been shown to cause acquired color vision deficiency, except:
a. Carbon monoxide.
b. Carbon disulfide.
c. Sodium chloride.
d. None of the above.

20. The clinician is typically able to detect and quantify the retinal and peripapillary B-scans using SD-OCT with what resolution?
a. Less than 1µm.
b. 2µm.
c. 3µm to 5µm.
d. 1mm.

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# Read the Retinal Vasculature Like a Pro

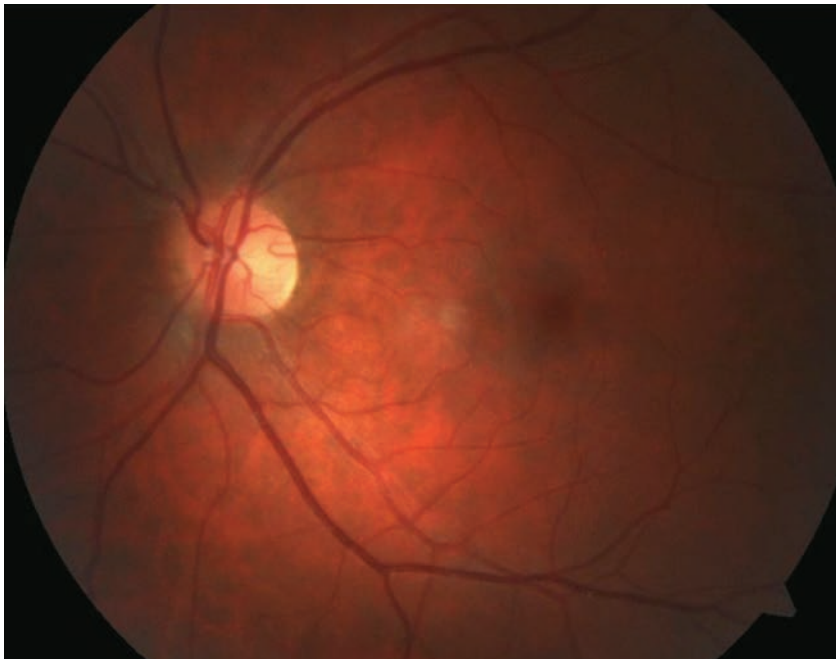
Experts describe the causes and contours of congenital arteriovenous anomalies.

By Bisant A. Labib, OD, Andrew S. Gurwood, OD, and Andrew L. Meagher, OD

The retina relies on a well-functioning blood supply to do its precious work without interruption or incident. When investigating suspected retinal disease, it can sometimes be difficult to discern the role played by the vasculature, in part because of how unique it is to each patient. The patterns of our retinal vessels are as distinctive and idiosyncratic as fingerprints, which is why retina scans can be used as a high-tech identification tool.

To help you better prepare to recognize vascular-based abnormalities, we provide here a guided tour of the blood vessels and structures surrounding the retina, as well as an explanation of the congenital anomalies associated with each structure. Though these aberrations are rare, they can have a significant impact on vision and treatment of visual complications. They can also serve as a marker for underlying systemic illnesses and dictate the clinician's work-up and treatment plan.

This feature is the second in a four-part series that offers an in-depth look at the intricacies of retinal vasculature to highlight the importance of the vascular supply in maintaining retinal integrity and function. Part one can be found online at www.reviewofoptometry.com/article/recognizing-abnormal-vasculature.



**Pictured here is a cilioretinal artery coming off the temporal rim.**

## Retinal Blood Supply and Drainage

The ophthalmic artery (OA), the first major subdivision of the internal carotid artery on each side, is the first in a series of several arteries responsible for nourishing various ocular structures and adnexa.[1-3] The retina is supplied with blood through two branches of the OA: the central retinal artery (CRA) and the posterior ciliary arteries.[1-3] The first branch, the central retinal artery, runs along the optic nerve, passing through the lamina cribrosa and entering the optic disc nasal to the postocular center.[1] It then branches superiorly and inferiorly, dividing further into nasal and

temporal arcades (branches). These branches continue to bifurcate and supply blood to the inner retinal layers.[1,2] The CRA is a terminal branch of the OA and serves as the primary source of blood to the retina.[2,3]

The outer and middle retina is nourished by the choroid, which gets its blood supply from a different branch of the OA called the posterior ciliary arteries.[3,4] These arteries vary in number per individual, ranging from one to five, and divide further into several short arteries that supply the proximal choroid and optic nerve head. They pierce the sclera and continue as long ciliary arteries to supply the distal choroid.[3]

Venous drainage of the inner retina occurs first through the branch retinal veins into the central retinal vein, which emerges from the meningeal sheath of the optic nerve. The vortex veins of the choroid are responsible for draining the outer retina and choroid. They drain into the superior and inferior ophthalmic veins. These, and the central retinal vein, flow into the cavernous sinuses in the middle cranial fossae.[1] Blood supplied by this route enters the systemic circulatory system via the petrosal venous and sigmoid sinuses, which terminate in the internal jugular veins in the posterior cranial fossae.

## Cilioretinal Arteries

There are anatomic variations in which an arterial branch originating from the posterior ciliary arteries or choroid, or both, assists the CRA in supplying the inner retinal layers.[3,5] Studies show it is the most common congenital vascular anomaly of the retina, found in 6% to 32% of individuals.[3,6,7] A study using fundus photography and fluorescein angiography (FA) to aid in observation of cilioretinal arteries estimated the prevalence at 32.1% of eyes, suggesting these entities may have been underdiagnosed without the use of ancillary testing in earlier studies.[8] Uncommonly, multiple cilioretinal arteries can be present in one eye.[8] The incidence of bilateral presentations is approximately 14% to 18%.[3] Studies suggest genetics play a strong role in cilioretinal artery development.[5]

Cilioretinal arteries appear clinically as vessels that originate at the disc margin and arch outwards. The sharp loop caused by the arch is an important diagnostic feature, as is the absence of a direct connection to CRA.[8] They may be singular, multiple and vary in size from minor to substantial. On FA, cilioretinal arteries fill seconds before the rest of the retinal circulation because they gain their blood supply from the choroid.[9]

These vessels commonly supply the fovea, followed by the lower temporal region of the retina; occasionally they supply regions directly temporal to the disc.[8] Only a few cases have been reported in a nasal location.[5,8] Even more rare are cases in which a cilioretinal artery supplies the entire retina, suggesting that they may create an anastomotic network with normal CRA branches.[3]

A cilioretinal artery may be beneficial in cases of a central retinal artery occlusion. Here, the presence of this congenital anomaly can prevent catastrophic vision loss by providing an alternate blood supply to the fovea and maintaining circulation in the event of a CRA occlusion.[3,5,9] In a similar manner, it can also aid in retaining central vision in cases of advanced primary open angle glaucoma (POAG), due to the increased level of circulation to the temporal rim of the optic nerve head.[10] However, it has not been shown to markedly influence the pattern of neuroretinal rim loss or parapillary atrophy progression in POAG.[11]

While the presence of a cilioretinal artery can be of great clinical benefit, it may also pose potential risk, such as in instances of cilioretinal artery occlusion. However, unlike in cases of central or branch artery obstructions, visual prognosis is often good, with 20/40 visual acuity or better in more than 90% of cases.[9,12]

In addition to the risk of occlusion, the cilioretinal artery is also associated with an increase in blood flow velocity. This can boost the incidence of diabetic macular edema.[13] One case in the literature reported reduced visual acuity and mild amblyopia due to an aberrant branching cilioretinal artery and macular thickening secondary to its compromise.[6] Finally, patients with this anomaly can fall victim to pathological processes such as macroaneurysm, although this is rare.[14]

## The Collateral Vessel

Retinal collateral vessels (CVs) originate from the existing retinal capillary network and become evident when one vessel is occluded, leaving the adjacent vascular channels operative.[15,16] Collateral vessels arise from processes that try to correct or compensate for vessel obstruction. Here, the obstructed vessel joins with an unobstructed one.[16] Collateral vessels have similar characteristics as normal retinal vessels; the channel they form connects inner retinal layers.[16] Although there is potential for CVs to be preexisting, they have no clinical significance and typically remain stationary throughout life in healthy patients.[17]

CVs are generally classified into three categories: arterioarteriola, arteriovenous collaterals and venovenular collateral.

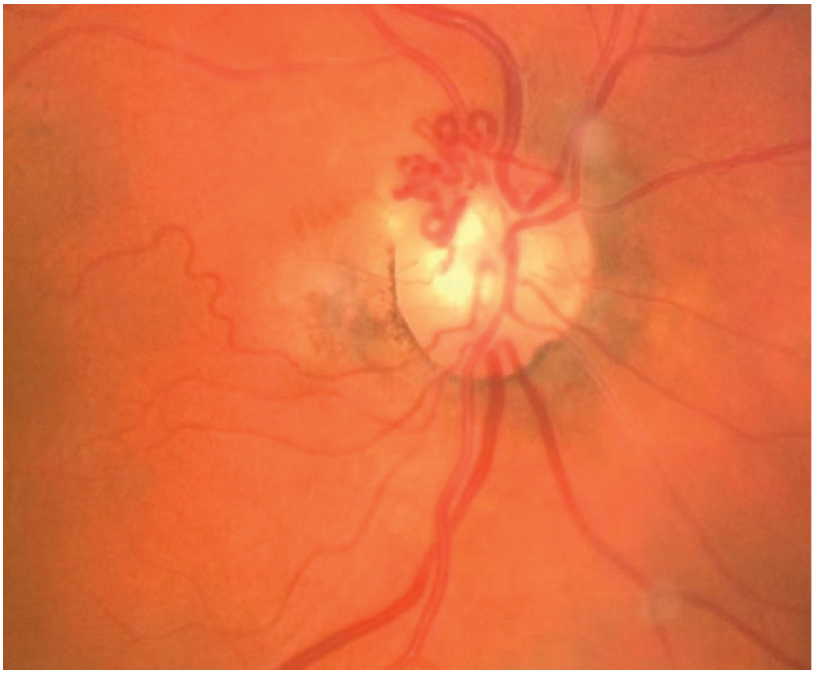
Arterioarteriola collaterals

become evident following a branch retinal artery obstruction and, rarely, in central retinal artery occlusion.[15,16] This subtype is seen infrequently and appears within weeks of arterial occlusion. The timing of their manifestation is not early enough to forestall the damage from the occlusive event, but serves instead as a marker of previous arterial occlusive disease on funduscopic examination.[15]

Arteriovenous collaterals may arise and fill in cases of capillary bed obstruction to allow blood to cross from the arterial side to the venous side of retinal circulation. Examples of retinal pathology that promote this include conditions such as diabetic retinopathy, long-standing glaucoma, sickle cell retinopathy and Leber's multiple miliary aneurysms.[15,16]

A venovenular collateral, the most frequently encountered subtype of collateral vessels, manifests following venous obstruction to provide an alternative means of blood flow from congested retinal areas relieving the "log jam" at the site of the thrombosis and mitigating the secondary ischemia produced by the poor perfusion induced by the venous stasis, preserving retinal function.[16,18] They become patent as a result of the pressure alteration produced by a vein occlusion and resultant increase of the intraluminal blood flow within the retinal vessels. This mechanism leads to an increase in the volume of blood that flows through the CVs.[17]

Investigators presume that any capillary channel can be formed into a larger-caliber vessel, such as an arteriole or vein, if circumstances initiate the process.[15] In the case of a branch vein occlusion (BRVO), CVs arise in approximately 22% of cases, allowing venous drainage into alternative, adjacent areas of the retina; these manifestations are associated with superior functional improvements and better visual prognosis.[17,18] Less is known regarding the benefit of CVs in central vein occlusion (CRVO). Some studies report an association with improved visual prognosis, while others hypothesize that their presence may delay the resolution of macular edema, hence worsening the prognosis.[16,19]



**Pictured here are collateral vessels.**

CVs are observed as early as weeks after venous obstruction.[15] They initially appear as small, tortuous vessels crossing the horizontal raphe to bypass the acquired venous blockage.[15,16] They take on a similar tortuous appearance in a CRVO, presiding over the optic disc.[16,19] Over a course of approximately six to 24 months, CVs mature and stabilize, appearing less tortuous.[18]

It is sometimes difficult to distinguish CVs from neovascularization on ophthalmoscopic evaluation. The predominant difference between these two vessel types lies in their appearance: CVs appear as normal, tortuous vessels that can be traced from vein to vein, artery to artery, artery to vein or vein to artery. Neovascular vessels are small and resemble "angel hair pasta." They are extremely tortuous and are nowhere close to normal-caliber vessels. Furthermore, their behavior on fluorescein angiography (FA) demonstrates leakage, where neovascular membranes leak but CVs typically do not.[18]

## Congenital Retinal Macrovessel

An aberrant blood vessel (either an artery or vein) that courses over the macula with its tributaries crossing the horizontal raphe, to either supply or drain the macular region, is known as a congenital retinal macrovessel (CRM).[20-23] These are rare, reported to only occur in approximately one in 200,000 cases.[21,24] CRMs are typically unilateral and are thought to develop from anomalous embryonic tissues during the 15th and 16th weeks of gestation, at which time the differentiation of

the mesenchymal cells invade the retinal nerve fiber layer.[20,21,25] It is often categorized under group one of the retinal arteriovenous anastomoses classification system, which describes single or multiple vessels with direct arteriovenous communication and interposition of a capillary network.[22]

CRMs are diagnosed on fundus examination and can be confirmed with FA. Typical FA findings show early filling of the vein, followed by a slight delay in emptying. An atypical capillary network is also revealed through FA.[24] Optical coherence tomography (OCT) can be a useful tool in the diagnosis of CRM, as it may exhibit a disruption within the architecture of the foveal avascular zone.[26]

The presence of these isolated vascular malformations is benign and often an incidental finding on routine retinal examination.[25] Rarely, CRMs can cause a decrease in vision precipitated by the development of macular hemorrhage, foveal cysts, serous macular detachment, or simply the presence of the vessel coursing over the macula.[21,22] Arterial CRM, in conjunction with leaking retinal arterial macroaneurysm, can cause a decrease in vision due to macular impingement, according to investigators.[23] Some evidence suggests an association of CRMs with systemic vascular formations, as is seen in arteriovenous malformation (Wyburn-Mason syndrome), but this has only been recorded in one case revealing a coincident ipsilateral venous malformation within the left frontal lobe, suggesting that not all of these patients would need emergent neuro-imaging.[25]

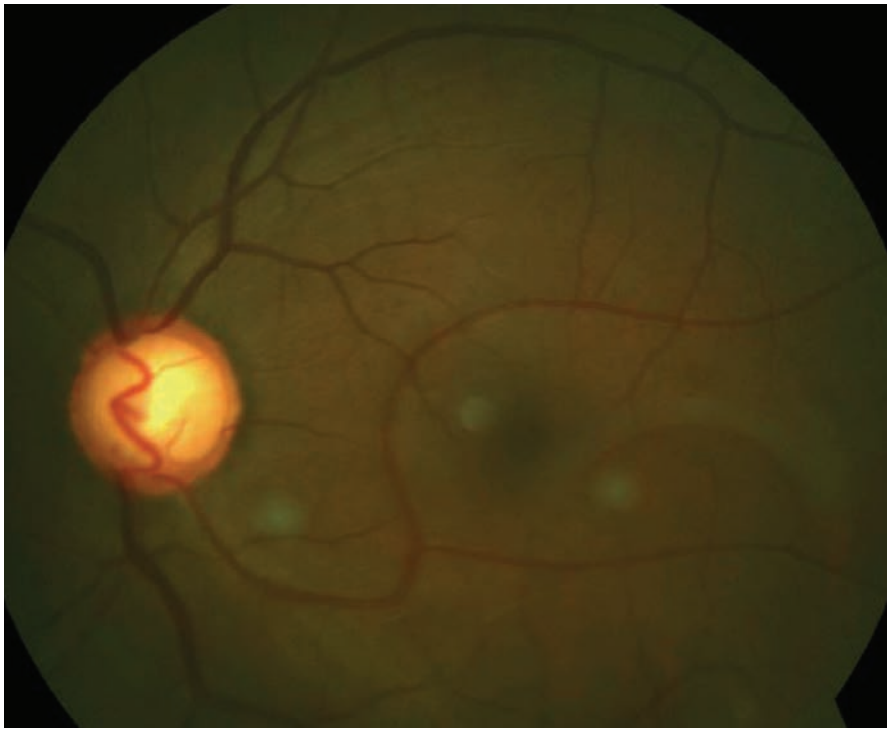
## Retinal Arteriovenous Malformation

Also known as racemose hemangioma and retinal arteriovenous communication (angiomatosis retinae), retinal arteriovenous malformation (AVM) is a rare, congenital but nonhereditary sporadic phacomatosis arising from the same tissue normal to the area.[27,28] It is characterized by marked dilation of venous and arterial tissue.[29]

The condition presents unilaterally and may either be confined to a specific area of the retina or diffusely widespread.[29,30] Most commonly, retinal AVMs are found in the superior temporal quadrant, followed by the papillomacular bundle and least commonly nasal to the optic disc.[29] On FA, retinal AVM fill rapidly and do not leak.[29] On OCT, retinal AVMs appear as prominent, intraretinal vessels that correspond in area to the abnormal retinal vasculature on fundus examination. Though they vary in depth, these vessels typically do not penetrate deeper than the outer nuclear layer of the

retina.[30]

Retinal AVMs are classified into three subtypes. Type 1 involves a capillary plexus or network connecting artery and vein. Type 2 retinal AVMs do not involve this adjoining capillary network, and are classified based upon their direct arterial and venous communication. Finally, the third type is characterized by intertwined and convoluted connections that it make it difficult to differentiate between the arterial and venous components.[29,30] Type 3 is also often associated with concurrent lesions of the optic nerve, chiasm and the cerebral cortex, as in Wyburn-Mason syndrome.[29]



**Here is an inferior congenital retinal macrovessel extending over the horizontal raphe.**

Typically, retinal AVMs are nonprogressive and not visually threatening.[29,31] Occasional, rare ocular complications that lead to visual decline can arise from these high-flow developmental vascular anomalies.[27,29] The most common cause of visual disruption is venous occlusion, which is thought to arise from the high, turbulent venous blood flow and decreased arterial pressure leading to thrombus formation and surrounding areas of induced retinal ischemia.[32]

Vitreous hemorrhage may occur as a consequence of the same process. Increases in hydrostatic pressure on the venous side of the malformation can also produce retinal and vitreous leakage.[30,33] Neovascular glaucoma has been documented in cases of retinal AVMs occurring secondary to retinal ischemia.[27,28] Macular edema is also possible.[34] Associated ocular conditions include morning glory syndrome, macular holes, Sturge-Weber disease, Vogt-Koyanagi-Harada disease, Duane's retraction type 1 and retinal macroaneurysms.[27]

Wyburn-Mason syndrome, also known as Bonnet-Dechaume-Blanc syndrome, occurs in approximately 30% of retinal AVMs and is accompanied by angiomas of the brain, most commonly the midbrain, or within the face.[27,35] Wyburn-Mason syndrome results from a disruption in embryological development within the vascular mesoderm.[28] Associated intracranial AVMs can result in neurological morbidity and even death, making retinal AVMs of great clinical importance when diagnosed.[28] The presence of these intracranial or orbitocranial lesions can also result in optic atrophy and resultant vision loss.[27,28]

Though the risk of ocular complication and vision loss is rare, retinal AVMs require prompt referral to neurology or neuro-ophthalmology departments for neuroimaging to rule out the possibility of associated intracranial or facial AVMs. Prompt magnetic resonance imaging and cerebral angiography can identify these conditions, at which point referral for a neurosurgical consult is recommended.[28] Due to advancements in surgical and radiation techniques, the intracranial AVMs can sometimes be fully eliminated without compromising cerebral blood flow.[28]

Although these congenital anomalies are not often encountered, they carry great clinical significance. Some of these conditions can appear funduscopically in response to a vascular event, while others may point to underlying systemic abnormalities. Despite the rarity of ocular complications in these cases, prompt identification and appropriate referral or management can aid in the prevention of visual decline. ■

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# See the Future at SECO 2017

Optometrists can stay ahead of the curve with more than 200 CE courses at this year's conference. **By Jane Cole, Contributing Editor**

Want to get your practice's future in better focus? Then head to Atlanta March 1-5 for SECO 2017, where more than 300 continuing education (CE) credit hours will be offered. Optometrists will have the opportunity to earn up to a maximum of 41 CE hours.

SECO 2017 will feature more than 200 CE courses for optometrists and ophthalmic professionals, including five special sessions, nine learning labs and the new MedPro360 program.

"Our theme this year, 'Your Future in Focus,' says it all," says Paul C. Ajamian, OD, SECO CE Committee chair. "SECO's CE committee works year-round to provide a slate of select speakers who will discuss not only what is relevant today, but how we prepare for tomorrow. From custom-crafted special sessions to hourly breakout courses to high-level staff training, there is no other meeting that raises the bar like SECO does, year after year."

In addition to a wealth of hourly courses that will take a deep dive into topics such as contact lenses, myopia control and glaucoma, you won't want to miss these special sessions and other clinical educational offerings:

• ***MedPro360:*** This year, SECO will launch MedPro360, a one-day business management program.

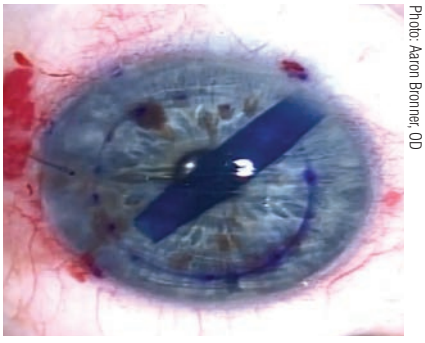

Photo: Aaron Bronner, OD

**In this DMEK procedure, a scroll of donor Descemet's membrane tissue within the anterior chamber is stained with trypan blue for visualization. Clinicians can learn about such procedures during the "Skinning Cats: Corneal Transplantation From Front to Back" session.**

MedPro360 will bring together health care professionals from all specialties and areas of practice to learn ways to improve the profitability of their business. The program will teach proven strategies in marketing, management, analytics and human resources. Keynote speakers will include Daniel Kraft, MD, presenting "The Future of Health and Medicine: Where Can Technology Take Us?" and Dennis Snow, a former Disney executive, presenting "The Employer Factor." SECO will be targeting 27 different medical specialties for attendance.

• ***Retina Roundup:*** Over the last decade, the paradigm has shifted in diagnosis and treatment of posterior segment disease. As such, retinal specialists Mohammad Rafieetary, OD, John Randolph, MD, and Eric Sigler, MD, will discuss the latest in imaging technologies, genetic testing, chemotherapeutic modalities and surgical vitrectomy. They will discuss the most commonly seen retinal diseases and disorders, along with practical advice for the comanaging optometrist.

• ***Cornea Cornucopia:*** This special session will feature a panel of two of ophthalmology's brightest young women, Elizabeth Yeu, MD, and Preeya Gupta, MD, and will be moderated by one of optometry's brightest young men, Walter Whitley, OD, MBA. Attendees will hear updates on what's hot in corneal and external disease. From clinical cases to current trends in eye care, the panelists will provide take-home pearls that will keep attendees on the cutting edge.

• ***Skinning Cats: Corneal Transplantation From Front to Back:*** This special session, taught by one of the nation's leading corneal pioneers, Peter Veldman, MD, will cover the techniques that have revolutionized, and in some cases replaced, corneal transplantation, such as Descemet's stripping automated endothelial keratoplasty, Descemet's membrane endothelial keratoplasty and deep anterior lamellar keratoplasty. He will show videos to demonstrate the proce-

dures, and will review postoperative management and expected outcomes.

• ***Lessons Learned From Larry:*** Honoring a Giant: Larry Alexander, OD, was a mentor to many. This special session will highlight many of his "Eye Lessons" that have been a source of instruction and inspiration to the profession that he loved. Leo Semes, OD, Joseph Pizzimenti, OD, and Blair Lonsberry, OD, MS, MEd, will present.

• ***Learning Labs:*** Choose from nine learning labs, including the Laser Learning Lab, which will demystify the various laser procedures in eye care, and the MGD Learning Lab, which will provide a hands-on look at the diagnostic and therapeutic modalities for dry eye.

• ***What's My Beef?:*** This series is a fun way to end the day. Topics will include frustrations with pharmacists, vision plans, referrals, glaucoma meds and speakers.

• ***CEE at SECO:*** Choose from 22 courses for 43 hours of continuing education with examination during SECO 2017. The University of Alabama at Birmingham School of Optometry will be the examination provider.

• ***MACRA and MIPS:*** The New MU: Learn about the OD's expanded role with these federal health policies.

• ***The Optic Neuropathies:*** This course, presented by Tina Porzukowiak, OD, will review the clinical presentations of optic neuropathies, including advances in treatment and management with reference to current medical literature.

• ***Maximizing Profits in a Competitive Market:*** The traditional optometric practice mode is facing the ever-increasing challenge of maintaining profitability. This course will identify key metrics a practice owner can use to more accurately measure financial indicators. Speaker David Mills, OD, MBA, will discuss strategies on how to improve the financial health of a practice. On completion of the course, providers will be able to immediately implement changes in their business that will increase the profitability of the practice.

• ***Free Continuing Education in the Presentation Theaters:*** Visit SECO's expanded slate of Presentation Theater courses with 25 additional opportunities to earn free continuing education inside the exhibit hall.

For more information or to register, go to www.seco2017.com. ■

## No "I" in Team

For 2017, SECO will offer 30 team-centered courses designed to benefit ophthalmic professionals as well as optometrists. These courses will offer a unique opportunity for doctors and staff members to learn side-by-side.

Here is a snapshot of some of the team-centered courses:

- ***OSD is Not a Four-Letter Word:*** According to a recent report, the dry eye products market is expected to generate $4.5 billion by the year 2020.[1] New technologies and new treatment modalities are emerging at an explosive rate. This course will help you make sense of the many options available to detect and manage ocular surface disease. The speakers, Alan Kabat, OD, and Whitney Hauser, OD, will discuss the latest information on appropriate diagnostic testing, therapeutic interventions and practice patterns to help ensure success.
- ***The Future of Cataract Surgery:*** Presented by Dr. Ajamian, this course will explore modern-day cataract surgery, including advances that are available now but not universally embraced, along with future technology.
- ***In-Office Emergencies:*** It is crucial for every health care provider, including optometrists, to be prepared for potential in-office emergencies and have an appropriate response plan in place. Caroline Pate, OD, and Elizabeth Steele, OD, will discuss potential non-ophthalmic emergencies, along with training and supplies for the office and staff.
- ***Making Sense Out of Health Care Reform:*** This topic has been extremely dynamic in recent years and still remains a confusing topic. This course, which will be presented by Bryan Rogoff, OD, MBA, will provide an overview of how to transform your practice to remain compliant. Dr. Rogoff will share information on the stress this can cause on practice operations, revenue growth, marketing, and patient flow and retention.

For a full list of team-centered courses, go to:
www.seco2017.com/education/team-centered-course-list.html.

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**SECO 2017 honors Larry Alexander, OD, with a special session in his name.**

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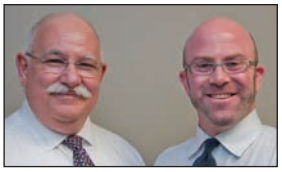


# Binocularity, How Sacred Art Thou?

Easily obtained single binocular vision is certainly a priority. But its achievement isn't always the best end goal for the patient. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

As optometrists, we all strive to help our patients attain clear, single binocular vision (CSBV) and to do so with ease. Now that we see more and more patients who have sustained a traumatic brain injury (TBI) or an acquired brain injury (ABI), we also see patients who have had CSBV for years only to have it altered abruptly and, in some cases, permanently. The case we discuss here highlights the unique challenges to finding a path back to CSBV.

## The Case

Dennis was diagnosed with a brain tumor in his fourth ventricle, which was removed in April 2012. After the removal, he had been seen at a local rehab hospital but suffered a fall and had meningitis. During his stay at the facility, he had his right eye patched by rehab staff because of double vision; prior to this, he simply closed his left eye all the time.

The patient was first seen in our clinic on June 19th, 2012, prior to beginning chemotherapy. He had no lens prescription and his unaided distance visual acuities were 20/30 OD, 20/40 OS and 20/30 OU. At near, unaided acuities were 20/60 OD, 20/40 OS and 20/30 OU. Cover testing at distance in his primary gaze showed a 35 prism diopter (PD) alternating esotropia with 10 PD of left hypertropia. At near, the horizontal component of the turn decreased to 30 PD. His eye movements were full, but he had trouble looking directly at the target and saw double in all fields of gaze. No mention of concomitancy vs. non-concomitancy was recorded. The retinoscopy exam yielded the following data:

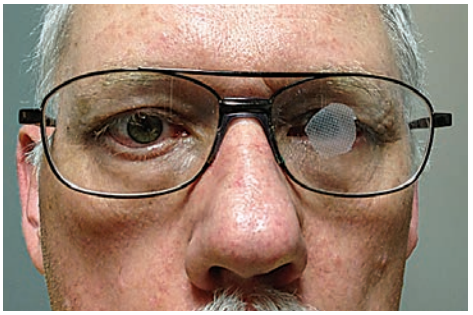


**Fig. 1. For patients who have lost CSBV due to TBI or ABI, sometimes the best outcome doesn't revolve around getting it back.**

- OD Plano -0.50 x 90 20/30.
- OS -0.25 -0.25 x 90 20/40.

We wondered why he wasn't seeing better without lenses, given the retinoscopy findings. Often, the fixation skills of these patients have been so affected by brain problems that they achieve lower-than-normal visual acuities, but show improvement over time.

## Attempting for CSBV

We measured the patient's broken habitual Rx, which was:

- OD -1.00 -0.75 163 +2.00 add, 20/60 distance VA.
- OS -0.75 -0.75 14 +2.00 add, 20/40 distance VA.

First, we tried to help Dennis establish CSBV using a compensating prism. This approach allows the person to continue using their eyes in the turned position. We like to think of a prism used this way as shifting the images to where the eyes are. We often find that during periods of constant double vision, the patient is often quite agitated and cannot concentrate on looking or seeing. As a result, they fail to engage in the visual world around them. We first tried Fresnel prisms with 10 PD base up over the right eye and 35 PD base out over the left eye, over plano lenses in a distance-only frame. He returned on June 2nd and, based on his initial report that the prisms were indeed helping, the same prisms were put on +2.50 lenses for near. He reported that day seeing single vision with them.

Dennis returned on October 9th, after completing chemotherapy. He was still wearing his lenses, but reported blurred vision. Fresnel prisms do degrade the image seen through them—the higher the power, the worse the acuities. Since he had been closing his left eye prior to receiving prism, the higher amount was put over the left eye, degrading its acuity more than the right. The magnitude of the horizontal and vertical prism amounts was significant enough, and of high enough powers, that using one prism and rotating it to the right position, based on vector addition, was not a practical option. When it is practical, it allows us to rotate with the least amount of disruption.

We hoped that, as we helped him establish fusion, the binocular sys-

tem would, on its own, become more robust and we could begin decreasing the prism amounts slowly over time. At the October 9th visit, Dennis was able to concentrate well enough that we obtained a better refraction, which gave him 20/25 acuity. Some myopia—approximately -1.00 OU—with a small amount of against-the-rule astigmatism was noted. The amount of prism needed had not changed, so we decided to begin vision therapy and prescribe two pairs of glasses, one for distance and one for near with the Fresnel prism.

## A Change in Priority/Direction

Following 11 sessions of vision therapy, Dennis was reevaluated. We saw that he had a degree of non-concomitancy and, when he tracked from side to side, his two eyes were not moving smoothly together. The left eye was actually moving more smoothly than the right over most of the horizontal range. The left eye could not move far to the left without significant effort, and his right eye could not move far to the right. This led to him having to move his head to keep objects inside a narrow cone of vision.

Since he continued to complain about his clarity of sight, and it appeared that non-concomitancy was the underlying issue keeping him from progressing, we undertook a radical change: The prisms were removed and a spot patch was placed over his left eye.

He immediately loved how he saw. He remarked at the clarity of his vision—which resulted primarily from removing the prisms. The right eye was designated as the uncovered eye, because he still tended to close the left eye in the absence of prism. A spot patch, in this case a section of transpore tape, was placed on the front surface of the patient's lens *(Figure 1)*. This was done quickly and crudely as an experiment, and we checked him at his next scheduled vision therapy session. He loved the results of the transpore tape.

He could move his eyes and head freely now and, when he saw double, instead of shutting his left eye, he simply rotated slightly until the second image ducked under the spot patch. Over time, we moved the patch to the inside surface of the lens and, instead of transpore tape, we used Bangerter filters, which are more inconspicuous to the non-trained observer.

## CSBV—Not Always the Goal

Patients like Dennis help us recognize that CSBV may not always be the ultimate end point in care. In this case, the degree of non-concomitancy and the lack of change following 11 sessions of vision therapy suggested we should take a different tack, and Dennis was sure glad we did. ■

# A Real Dog of a Diagnosis

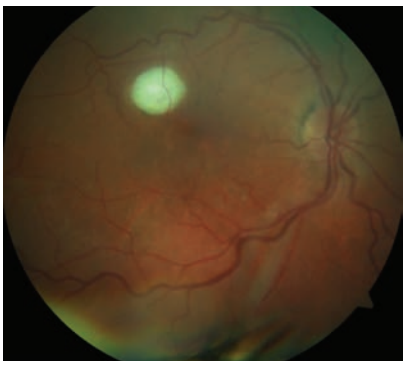
When a patient's vision is blurry even after cataract surgery, check the retina.

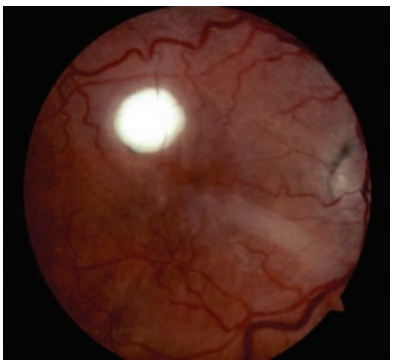
By Tania Patel, OD, and Mark T. Dunbar, OD

A 66-year-old Caucasian male presented with complaints of blurry vision in the left eye more than the right and difficulty reading labels on medication bottles. Ocular history was remarkable for cataract surgery in the right eye in 2012, which he felt did not result in any improvement in his vision. The patient was lost to follow-up until 2016. His medical history was significant for chronic obstructive pulmonary disease, hypertension, coronary artery disease and depression.

On ocular examination, his best-corrected visual acuity was 20/150 OD and 20/200 OS. Extraocular motilities were full, and confrontation visual fields were full to careful finger count in each eye. Pupils showed physiological anisocoria but no afferent pupillary defect. Intraocular pressures (IOP) were 14mm Hg OD and 12mm Hg OS.

Anterior segment evaluation revealed corneal arcus 360 OU. The anterior chamber in each eye was deep and quiet. In the right eye there was a posterior chamber intraocular lens, and the left eye had 3+ cortical, nuclear and posterior subcapsular cataracts. Posterior segment showed clear vitreous and healthy optic nerves in both eyes. In the right eye adjacent to the macula, an elevated, well-circumscribed lesion could be seen (*Figures 1 and 2*). Spectral domain ocular coherence tomography (SD-OCT) through the lesion (*Figure 3*) and a macular cube were also obtained (*Figure 4*). Posterior segment findings in the left eye were unremarkable.





**Figs. 1 and 2. Both these fundus photos show our patient's right eye. What does the lesion represent?**

## Take the Quiz

1. How would you characterize the lesion?
a. Choroidal neovascular membrane.
b. Old macroaneurysm.
c. Posterior pole granuloma.
d. Chorioretinal coloboma.

2. What is the likely diagnosis?
a. Toxoplasmosis.
b. Choroidal osteoma.
c. Toxocariasis.
d. Ocular tuberculosis.

3. What is the OCT interpretation?
a. Hyper-reflective mass with RPE disruption.
b. Choroidal neovascular membrane.
c. Macular edema.
d. Both a and c.

4. How should this patient be managed?
a. Monitor the condition without treatment.
b. Steroid drops and order laboratory testing.
c. Steroid injection.
d. Anti-VEGF injection.

5. Which is the most likely etiology?
a. Roundworm.
b. Protozoan parasite.
c. Mycobacterium tuberculosis.
d. Bony mass.

*For answers, see page 114.*

## Diagnosis

This patient was seen in 2012 with no retinal abnormalities noted. At the 2016 visit, he presented with a yellowish-white, round, raised lesion with intraretinal fluid and macular edema in the right eye. No cells or flare were observed in the anterior chamber and vitreous

was clear. SD-OCT of the macula confirmed intraretinal fluid. SD-OCT 5-line raster of the lesion showed a hyper-reflective mass with retinal pigment epithelium disruption. The differential diagnosis included toxocariasis, toxoplasmosis, tuberculosis, choroidal osteoma and sarcoidosis.

Choroidal osteoma has a predilection for women and usually presents in the teenage years to the twenties. It presents as a minimally elevated bone-like mass of the choroid. We felt the clinical presentation, OCT findings and epidemiology were inconsistent with choroidal osteoma.

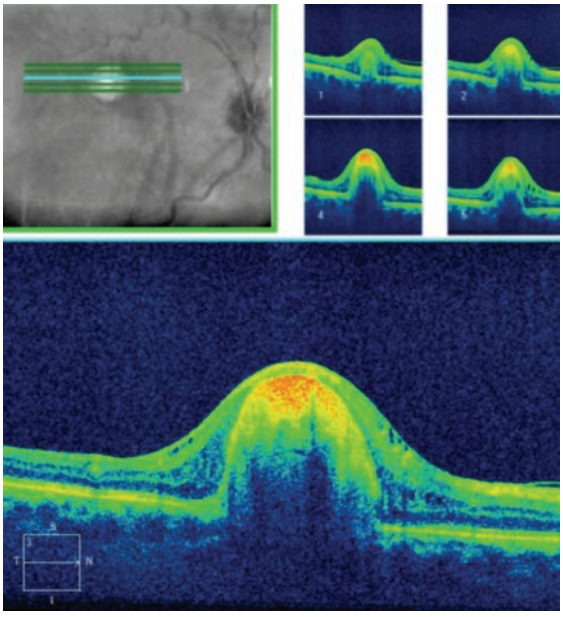


**Fig. 3. OCT 5-line raster of the lesion. How does this correlate to the fundus image?**

Active toxoplasmosis commonly presents with a significant amount of vitritis ("headlights in a fog"). There may also be spillover into the anterior chamber. The anterior chamber and vitreous were clear in our patient. It is possible that the lesion may now be inactive. Inactive toxoplasmosis is generally flat and has pigmentation at the edge of the chorioretinal scar. The lesion in our patient was elevated and there was no hyperpigmentation present.

Both sarcoidosis and ocular tuberculosis may present with a granulomatous anterior or posterior uveitis or both; however, sarcoidosis has a predilection in the African-American population. Our patient did not present with anterior or posterior segment inflammation. A complete blood work and chest x-ray were performed on the patient a few weeks prior to his presentation at the eye clinic and revealed no abnormalities.

Toxocariasis presents unilaterally as a posterior pole or peripheral granuloma. Fibrous bands may be present from the disc to the granuloma.[1] Depending on the location, intraretinal edema, optic nerve edema and endophthalmitis can be noted.[1] However, there are generally no chorioretinal scars adjacent to the granuloma, as is seen with toxoplasmosis scars.

## How the Worm Turns

As indicated, our patient had undergone a medical evaluation prior to his eye exam that included complete blood work, which was unremarkable, and a negative chest x-ray. Upon further questioning he confirmed that he obtained a dog three or four years prior to his exam, after which he noted poor vision in the right eye. Through a careful ocular history and clinical findings, including SD-OCT, we felt our patient had presumed ocular toxocariasis.

Toxocariasis is an infection caused by a nematode—most commonly the roundworms *Toxocara canis* and *Toxocara cati*. Dogs and cats are carriers of the roundworm. It exists as visceral larva migrans and ocular larva migrans (OLM).[2]

*Toxocara canis* can be transmitted by ingesting contaminated soil (geophagia) or water. Children and young adults are commonly affected, but it can also be seen in adults. Larvae enter the human host and can travel to the liver, lungs, central nervous system and eyes.[2] Ocular larvae enter the eye via choroidal, ciliary or retinal arteries where they are encapsulated in an eosinophilic granuloma due to inflammatory process.[3]

Aqueous and vitreous fluids may reveal increased eosinophils and toxocara antigens/antibodies. However, such tests are reserved for severe cases involving opaque vitreous and are usually deemed unnecessary for diagnostic and therapeutic reasons.[1] Peripheral immunological tests such as the toxocara-specific ELISA are unreliable for detecting OLM. Ocular toxocariasis is primarily diagnosed on the basis of clinical criteria during an ophthalmologic examination.[2]

## Treatment

Treatment for toxocariasis includes topical steroids for active inflammation, cycloplegic drops for ocular discomfort and even pars plana vitrectomy in the presence of significant inflammation or retinal detachment. Anthelmintics such as albendazole or thiabendazole is also used for active disease. Albendazole is the medication of choice due to its increased permeability

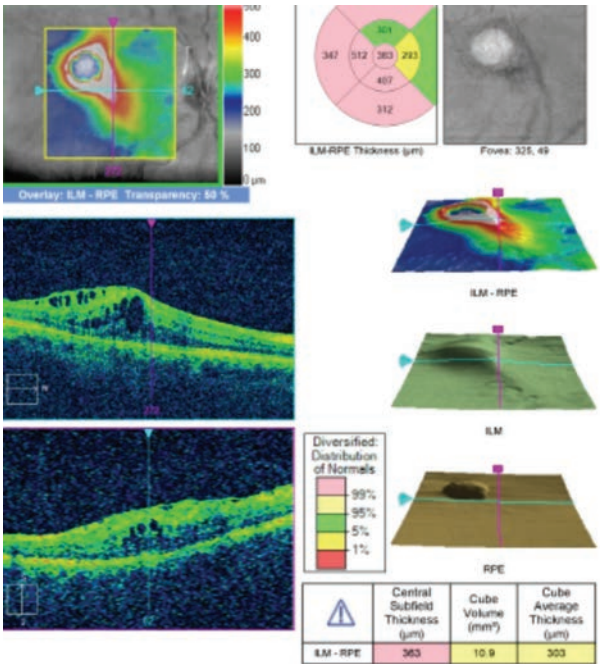



**Fig. 4. Macular cube OCT of the macula. What does it show?**

to blood-brain barrier and lower toxicity than other antihelmetics.[4]

Our patient was treated with Durezol (difluprednate, Alcon) QID and Nevanac (nepafenac, Alcon) QID OD to control the macular edema. Cataract surgery was recommended for the left eye post treatment of macular edema in the right eye. QuantiFERON gold and ACE testing were ordered to further rule out tuberculosis and sarcoidosis. Toxocara titers were also ordered.

Unfortunately, the patient was lost to follow-up and has not completed further testing. However, during a subsequent telephone conversation, he reported subjective improvement in vision in the right eye with the prescribed drops. ■

*Dr. Patel is a resident at the VA Medical Center in Lexington, KY.*

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# Welcome to the Office

When new patients give you a try, use all the tools at your disposal to display confidence. **By James L. Fanelli, OD**

A 69-year-old Caucasian male presented to our office as a new patient in December 2016 with a chief visual complaint of slightly decreased vision with computer tasks. Otherwise, he had no significant complaints. He mentioned undergoing "some laser procedures" to both eyes about seven years earlier and had glaucoma surgery in his right eye approximately five years earlier.

He was last seen by his glaucoma doctor approximately nine months earlier and reported that he had been due visit again this past September, but was just getting around to following through with that task.

Current systemic medications included daily lisinopril for hypertension and alprazolam and loratadine as needed. He reported no allergies to medications. Ophthalmic medications included Simbrinza (brinzolamide/brimonidine tartrate ophthalmic suspension, Alcon) twice a day in both eyes and generic latanaprost at bedtime in both eyes. He reported that he has been on the same glaucoma medications for the past two to three years and, furthermore, that his glaucoma doctor was pleased with his tolerability of the medications and their efficacy.

## Presentation

Entering visual acuities were 20/40 OD and 20/30- OS. Best-corrected visual acuities were 20/30+ OD and 20/20- OS. Pupils were reactive to light and demonstrated no afferent pupil defect by inverse Marcus Gunn testing. The right pupil in the right was surgically distorted and larger than the left, with evidence of iris sphincter damage. He subjectively reported that the vision in his right eye has been bad for some time due to the glaucoma. A quick view of the posterior pole demonstrated advanced glaucomatous optic neuropathy.

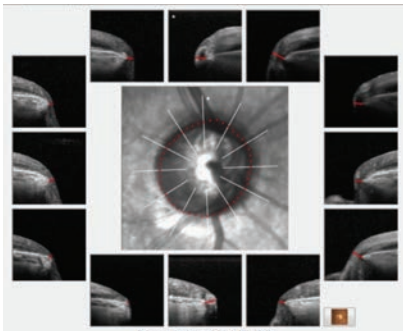

**Fig. 1. These images show the Bruch's membrane opening overview of the patient. Note the thinned neuroretinal rim tissue in all sectors, especially inferotemporally.**

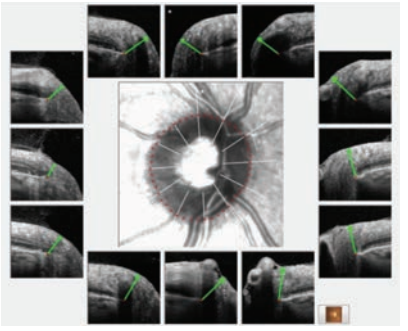

**Fig. 2. A non-glaucomatous eye in the context of BMO minimum rim width and nine representative radial scans of the optic nerve. Note the robust neuroretinal rim in each scan as evidenced by the green marker, as compared with Figure 1 that shows significantly eroded margins.**

A slit lamp examination of the anterior segments was remarkable for several items, including a patent trabeculectomy bleb, a surgical PI, sectoral iris atrophy and the aforementioned pupillary irregularity—all in the right eye. He was pseudophakic OU with clear posterior chamber intraocular lenses (IOLs) and clear intact posterior capsules. Applanation tensions were 14mm Hg OD and 15mm Hg OS at 11am. Pachymetry readings were 531µm OD and 544µm OS. Prior to dilation, threshold visual fields were performed with reasonable reliability indices. The fields in the right eye demonstrated a dense, superior arcuate scotoma involving fixation, with a pronounced nasal step; the left visual fields demonstrated above and below arcuate defects not involving fixation.

Through dilated pupils, the IOLs were well centered in the capsular bags. There were bilateral posterior vitreous separations. His cup-to-disc ratio in the right eye was estimated to be 0.95 x 0.95; in the left, it was 0.80 x 0.85.

The macula in the right eye was characterized by a centrally located epiretinal membrane (ERM) with epiretinal wrinkling along with fine retinal pigment epithelium (RPE) mottling present in both eyes. The retinal vascular examination was remarkable for mild arteriolarsclerotic retinopathy. The peripheral

retinal examinations were remarkable only for cystoid degeneration in both eyes.

HRT 3 images were obtained, as were optical coherence tomography (OCT) optic nerve and macular scans as per our office protocol (*Figures 1 and 2*).

## Discussion

This patient is new to the office, new to me, presenting with no previous information insofar as records, with advanced glaucoma in both eyes and decreased vision in the right that apparently is not of recent onset. One of the most important things to do at visits such as these, in my opinion, is to establish a rapport with the patient to foster a comfort zone so that they will continue their care with us and, equally important, heed my advice moving forward. Just as he is a new patient to me, I am a new doctor to him. He apparently was comfortable enough with his previous glaucoma doctor to undergo bilateral SLT procedures, as well as a trabeculectomy in his right eye and maintain that care for several years. Now that he has moved to this area, someone needs to pick up his care, and he has selected me for this job.

In an initial visit, we must also establish whether the patient is stable and, if not, make the appropriate changes in therapy. Fortunately, in this case, he appeared relatively stable and I did not need to alter his therapy.

I also took the opportunity to establish several important baseline measurements of this patient, including optic nerve imaging and visual field testing. His HRT 3 images were consistent with the clinical picture of advanced disease in the right eye (*Figure 3*) and the left.

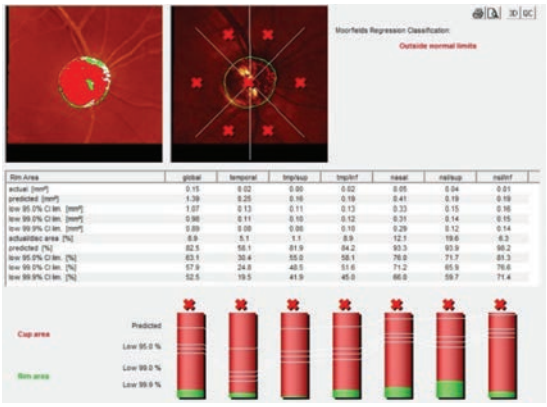

**Fig. 3. The HRT 3 image of the right optic nerve shows advanced disease and a thin neuroretinal rim.**

## Can You Picture This?

Imaging technology is rapidly evolving, and it is incumbent upon optometrists to be cognizant of these technological advances and their clinical implications. Clinicians must also be aware of the implication of using their instruments' reference databases.

This case is a good example of some imaging capabilities available to monitor optic nerve damage in glaucoma.

*Figure 4* shows a radial scan through the right optic nerve, looking specifically at Bruch's membrane opening and the minimum rim width of the ganglion cells as they pass posteriorly into the optic nerve. The reference database in the same image is that of the ethnic mix representative of the overall US population and is represented on the bottom right of the image. Clinically, we can readily see the patient has advanced disease, but interestingly enough, on the radial scan selected, the minimum rim width inferotemporally is reduced to a meager 6µm of remaining tissue, and all Garway-Heath sectors are clearly outside normal limits. For clinicians like myself who believe that damage can be detected by changes to the neuroretinal rim, such imaging technologies are appreciated.

When employing OCT scanning technologies, many use the retinal nerve fiber layer (RNFL) perioptic circle scan. In this particular patient, again, with clearly advanced disease in the right eye, the RNFL circle scans can be somewhat deceptive. *Figure 5* shows one of three circle scans taken around this patient's optic nerve; namely, a scan 4.1mm in diameter. Note that typical RNFL circle scans are 3.5mm in diameter, and with newer software, scans can be obtained with diameter of 4.1mm and 4.7mm. The key point here is that the further from the optic nerve the circle scan is obtained, the thinner, naturally, is the neuroretinal rim. Reference databases were established for each of these circle diameters.

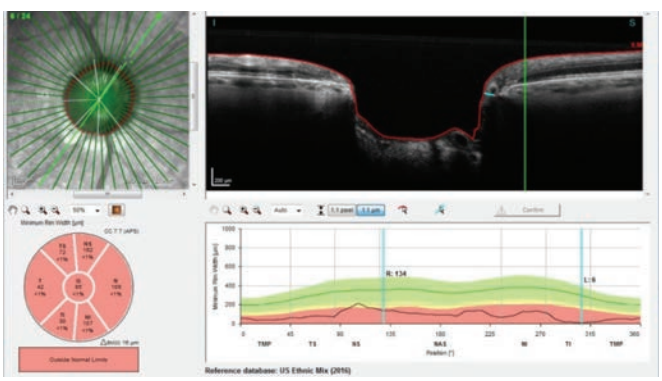

**Fig. 4. An overview of the Bruch's membrane opening in a patient with advanced disease.**

So even though advanced disease is readily seen on the physical slit lamp evaluation of the optic nerves, sometimes imaging data can give us a false sense of security.

In *Figure 5*, only one Garway-Heath sector is flagged as aberrant, implying that, at least for this particular patient, damage is more readily visible in the neuroretinal rim than the RNFL. Thus, it is critical that we are aware of precisely what our technology is telling us.

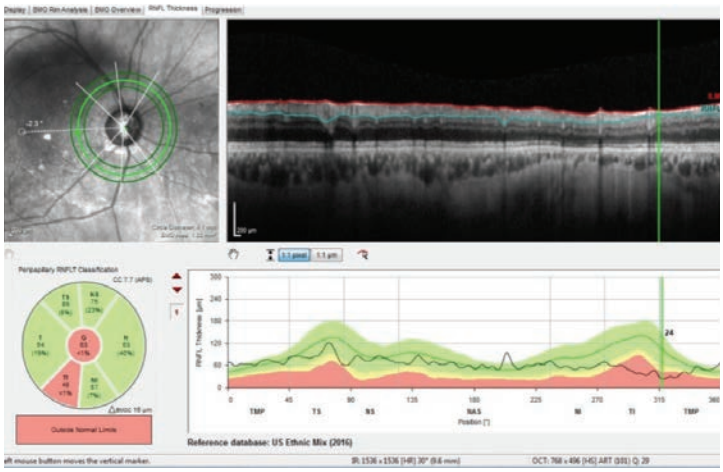

**Fig. 5. These circle scans measure the perioptic retinal nerve fiber layer. Highlighted in green in the upper left portion of the image is the 4.1mm scan, which shows statistical thinning only in the inferotemporal sector.**

## Building a Relationship

So, at the completion of the first visit with the patient, I was able to gather significant baseline data for future comparison and, just as importantly, build a strong doctor-patient relationship. I changed no medication regimens and I wrote him a new prescription for glasses. The patient is scheduled to visit again in four months for a follow-up visit, which will include the standard intraocular pressure reading and fundus examination, as well as gonioscopy and anterior segment OCT raster scans in the area of the trabeculectomy in the right eye. I think that I'm comfortable with my assessment of him being a compliant, reasonable individual. And I think he feels welcomed and comfortable with me being a reasonable and caring physician. ■



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# Is it Time to Drop the Drops?

Postoperative cataract care is becoming more user-friendly, but the jury remains undecided regarding "dropless" surgery. **By Alan G. Kabat, OD, and Joseph W. Sowka, OD**

Over the quarter-century that we've been practicing optometry, cataract surgery has undergone countless advances and improvements. From extracaps to phaco, sutures to sutureless and diamond blades to lasers, technology has helped to make cataract extraction a faster, safer and more accurate procedure in 2017 than was even imaginable in the 1990s.

Additionally, the use of perioperative medications, including antibiotics, corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs), has improved surgical outcomes and lowered the risk of postoperative complications. Studies reveal that the rate of endophthalmitis associated with cataract surgery in 2016 was 0.023%, compared with 0.12% (more than five times greater) in 1994.[1,2] Enhanced visual recuperation, amelioration of pain, diminution of corneal edema, mitigation of uveitis and attenuation of cystoid macular edema in the present era also may be directly linked to the use of postoperative corticosteroids and NSAIDs. Without question, these medications are critical to the safety and efficacy of cataract surgery. But can even these aspects of surgery be improved?

At present, our typical postoperative regimen for uncomplicated procedures consists of a fluoroquinolone antibiotic (TID to QID), a corticosteroid (typically QID) and an NSAID (either QD or QID, depending on the formulation) for 14 days after surgery; at this point, if recovery is acceptable, we discontinue the antibiotic and NSAID and begin tapering the corticosteroid for another two weeks. While such a routine may seem straightforward and easy for physicians who understand the role of these drops as well as their required frequencies, it can be monumentally difficult for patients to execute our orders with 100% compliance. The use of written instructions with pictures of the drops or descriptions of cap colors is helpful, but far from foolproof. The most frequent and time-consuming issue in our postoperative encounters is not managing surgical complications, but rather clarifying and re-educating patients on the appropriate use of their drops.

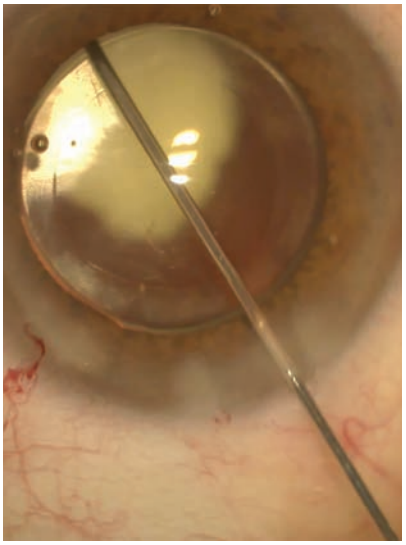

Photo: Lawrence Woodard, MD

**Dropless surgeries such as this one are changing both how cataract extractions are performed and how ODs manage patients postoperatively.**

## Drops, Out

One of the more recent and exciting innovations within the realm of cataract surgery has been the practice of "going dropless." This concept involves an intracameral injection of antibiotic and anti-inflammatory medications at the conclusion of surgery (prior to wound closure) by use of a transzonular approach into the anterior vitreous space.[3-7] Tri-Moxi (triamcinolone/moxifloxacin 15mg/1mg/mL, Imprimis Pharmaceuticals), Tri-Moxi-Vanc (triamcinolone/moxifloxacin/vancomycin 15mg/1mg/10mg/mL, Imprimis Pharmaceuticals) and Dex-Moxi (dexamethasone/moxifloxacin 1mg/5mg/mL, Imprimis Pharmaceuticals) became commercially available in mid-2014 and have gained acceptance by ophthalmologists ever since.

The advantages of a dropless strategy are numerous. As the name implies, the use of intracameral medications at the time of surgery all but eliminates the necessity of instilling drops during the postoperative period. Patients are the primary beneficiaries of this modality, saving not only in terms of pharmacy visits but also the cost of multiple medications. Additionally, eliminating the need for self-instillation reduces the risk of potential injury or contamination from dropper bottles.[8] For the doctor and staff, eliminating postoperative drops means less time is needed to educate and verify compliance with the prescribed regimen. Moreover,

it means fewer potential complications from improper drug instillation.[8] Perhaps most importantly, the dropless strategy virtually nullifies the dreaded pharmacy "call backs" regarding off-formulary medications or generic alternatives.

## The Down Side

The dropless approach is not without its disadvantages, however. The additional cost of obtaining these drugs cannot legitimately be passed along to the patient, and hence the surgeon or surgical center must bear this expense. The intracameral preparations cost $20 to $25 per single-use vial. And while this may add up for a busy cataract surgeon, many are beginning to offer the dropless approach as a "value added" proposition to patients electing to have premium cataract procedures, such as laser-assisted surgery, toric or multifocal IOLs or accommodating IOLs.

Another negative attribute of dropless surgery is the frequent issue of postoperative haze or floaters due to accumulation of drug particulate in the vitreous.[3,6] While this effect is transient and generally lasts no more than a week, some surgeons fear that it negates the "wow factor" of dramatically increased vision in the immediate postoperative period. Newer approaches aim to inject the drug into the inferior vitreous to decrease obstruction of the visual axis. Additionally, having the patient maintain an elevated head position while sleeping ensures that the drug reservoir settles and remains inferiorly.

An array of potential adverse events is also associated with dropless cataract surgery, especially in cases of poorly executed technique. Recognized complications may include: zonular rupture with subsequent dislocation of the IOL; hyphema or isolated vitreous hemorrhage; iatrogenic retinal tears or detachments; iris prolapse; and ciliary body hemorrhage.[4] But perhaps the most significant risk associated with dropless surgery is acute postoperative elevation of intraocular pressure (IOP). The association between IOP elevation and the use of intraocular corticosteroids is well-documented.[9-12] Some consider it wise to avoid the dropless approach altogether in known steroid responders and proceed cautiously in those patients with glaucoma or ocular hypertension. Patients must also realize that having dropless surgery is in no way a guarantee that eye drops can be completely avoided postoperatively. In cases of uncontrolled inflammation, associated IOP spike or other unanticipated events, adjunctive treatment with topical medications may still be necessary.

## In the Literature

Although the dropless approach has been employed for years, only a small amount of information regarding its safety and efficacy exists in peer-reviewed literature. A retrospective analysis of 1,541 eyes undergoing the dropless procedure with compounded triamcinolone/moxifloxacin/vancomycin revealed no major intraoperative complications and no cases of postoperative endophthalmitis.[4] Nearly 92% of cases required no supplemental medication after surgery. The rate of visually significant postoperative cystoid macular edema was just 2%, and the rate of clinically significant postoperative IOP elevation was <1%.[4] These values are similar to those seen in patients undergoing traditional cataract surgery with the use of topical medications postoperatively.[4] Another recently published report described a prospective, randomized, subject-masked contralateral eye study in which 25 individuals undergoing uncomplicated cataract surgery received a transzonular injection of triamcinolone/moxifloxacin/vancomycin at the time of surgery in one eye and topical, postoperative drop treatment in the fellow eye.[5] No statistically significant difference was noted between the groups with regard to IOP elevation from baseline, macular thickness or corneal thickness after one month.[5] The difference in reported pain (one day postoperatively) was also not statistically significant between groups.[5] Satisfaction with surgery was similar for both management approaches, but significantly more subjects preferred the injection for overall experience.[5]

The cataract experience continues to evolve as new technology allows for greater accuracy, greater convenience and faster recovery times. ■

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# A Quick Snip to Confirm GCA

Be prepared to comanage patients in need of a temporal artery biopsy.

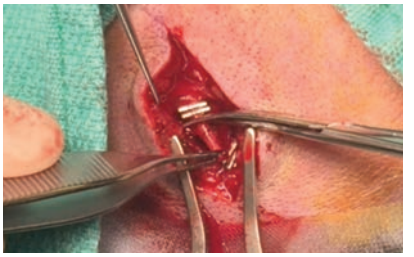
By Leonid Skorin, Jr., DO, OD, MS, and Zachary Lundgren, BA

Temporal arteritis, also known as giant cell arteritis (GCA), is a chronic autoimmune inflammatory disease that affects the major branches of the aortic arch.[1] It is a true ocular emergency due to its predilection for the branches of the carotid artery. If left untreated, a patient is at significant risk for sudden and permanent vision loss. GCA is most commonly seen between the ages of 60 and 75 and, rarely, younger than 50.[1] The disorder also presents more often in women and in Caucasians.[1]

## Presentation

Patients with GCA frequently present initially with a variety of signs and symptoms. It is crucial for clinicians to identify the disorder before ocular signs are noted, considering sudden, painless loss of vision occurs in 15% to 20% of patients with GCA.[1] Patients typically have symptoms of temporal head pain, jaw claudication, scalp tenderness and new-onset headaches. Other symptoms that may occur include fatigue, malaise and fever.

If GCA is suspected, clinicians should order testing, such as erythrocyte sedimentation rate, C-reactive protein and platelet count. If any of these three tests are elevated in a symptomatic patient, eye care providers should refer the patient for a temporal artery biopsy. If vision loss is present at examination, a consideration of intravenous methylprednisolone followed by oral corticosteroids should be started prior to biopsy to prevent further vision loss.



**Surgical clips isolate the specimen before excision.**

## Biopsy: Step-By-Step

Temporal artery biopsy is currently the gold standard for diagnosing cases of GCA. The procedure is typically performed under monitored sedation with the patient conscious.

After shaving the temple and sideburn area, the surgeon locates the course of the superficial frontal branch of the temporal artery with meticulous palpation and marks it.

The patient is given local anesthetic around the shoulders of the vessel, and the area is prepped.

After the site is tested to ensure the anesthesia has taken effect, using a #15 blade the surgeon makes a shallow incision through the skin and subcutaneous tissue directly over the temporal artery. Blunt dissection reveals the artery, and cautery is applied for hemostasis.

The artery is compressed and, if no ischemia is noted, surgical clips are placed at the proximal and distal ends of the blood vessel.[2] The specimen is excised and sent for analysis.

The subcuticular tissue is closed with dissolvable 4-0 chromic suture, and the overlying skin incision is closed with cyanoacrylate tissue adhesive and surgical tape strips.[3]

## Postoperative

Patients typically heal within two to three weeks with few complications, although minor swelling, bruising and irritation can occur. Pathology results typically return within a week, which is appropriate for follow-up. ODs should consult with the patient's primary care provider or rheumatologist to consider long-term management of any underlying systemic condition.

If the results are negative, no additional treatment is indicated. A positive biopsy confirms GCA, prompting treatment with systemic steroids, which are maintained until symptoms resolve and inflammation is under control. Typical treatment lasts six to 12 months, followed by a slow taper. Patients with GCA should be followed every three to six months to monitor ocular health. ■

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

*Mr. Lundgren is a fourth-year optometry student at Pacific University College of Optometry.*

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

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# Product **Review**

## Contact Lenses

### CooperVision Two-week Toric

Optometrists can offer their patients with astigmatism a toric lens with a new material designed for improved comfort. CooperVision's Avaira Vitality Toric is a two-week lens made with the company's fanfilcon A silicone hydrogel material designed to deliver 55% greater water content for a healthier wearing experience, the company says. The lens is also able to block 90% of UVA and 99% of UVB rays, according to CooperVision.

Avaira Vitality Toric is currently available in a power range of plano to -6.00D with cylinder options of 0.75D, 1.25D, 1.75D, in axes from 10° to 180° in 10° steps. It has a modulus of 0.6MPa, a Dk of 90, and a Dk/t of 90. The base curve is 8.5mm and the diameter is 14.5mm. Plus powers, high minus powers and a -2.25 cylinder will be available later in 2017.

Visit www.coopervision.com.

### B+L scleral Contact Lens

The Zen RC scleral contact lens, a new option from Bausch + Lomb, was designed specifically for patients with normal corneas. To simplify the fitting process, doctors may modify the parameters as needed, B+L says.

The Zen RC scleral lens is highly customizable, though the standard parameters should be adequate for most patients, according to the company. Toric peripheral curves, customized center thickness, flexure controlling profiles and front toric prescriptions can also be ordered when needed, according to B+L.

Visit www.bausch.com.

### Scleral Lens Fitting Set

BostonSight now offers a 22-fitting lens set to help practitioners more easily fit patients with scleral lenses in the large-diameter category. The BostonSight Scleral system uses patient data collected over six years from 7,000 eyes, to help eliminate the need for modifications, says BostonSight.

Scleral offers right- and left-eye anatomical designs. It is available in 18.0mm, 18.5mm and 19.0mm diameters, and it uses a single starting point regardless of a patient's condition. This is also the first fitting system to provide front-surface eccentricity options, including a quadrant-specific toric peripheral haptic system, according to the company.

The system requires no startup costs, extensive training or special equipment, according to BostonSight. Qualified practices receive complimentary diagnostic lens sets and online training webinars.

To sign up, register at www.bostonsightscleral.org or call 888-SCLERAL.

## Contact Lens Care

### Buffered Saline


ScleralFil, by Bausch + Lomb, is a sterile, buffered solution with no preservatives, indicated for inserting and rinsing scleral lenses. ScleralFil also avoids mercury-containing ingredients that can be sensitive to some contact lens wearers, and is buffered to maintain pH, according to B+L. It can also be used to rinse soft and gas permeable contacts to remove debris and traces of daily cleaner, prior to lens insertion.

Visit www.bausch.com.

## Dispensing Equipment

### Lens Checker

Asahi Vision's aLC-100 Lens Checker allows you to easily check contacts and spectacles for scratches and impurities, according to the company. It can also connect to a PC or tablet via USB and can quickly switch between dark- and bright-field modes. Asahi says the aLC-100 is a surface inspection tool for any kind of lens, giving practices the ability to observe progressive markings with limited effort, and record lens condition and usage.

Visit www.asahi-vision.com.

## Cosmeceuticals

### Under-eye Skin Cream

The OcuDerma eye gel from MediNiche, for patients who want to help minimize the appearance of aging, has been updated. MediNiche says the gel won't cloud contact lenses or cause irritation.

The gel formulation creates a non-oily, non-sticky vehicle, and the product contains no fragrances, artificial dyes, parabens, sensitizers or pore-clogging agents, according to the company. MediNiche says that the product contains unique extracts, locust bean and hyaluronate that make the skin around the eyes feel tighter and look firmer.

OcuDerma now also comes in an airless pump bottle and features a "sani-dose" pump dispenser.

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## March 2017

■ **1-5.** *SECO 2017.* Georgia World Congress Center, Atlanta, GA. Hosted by: SECO International. Key faculty: Mohammad Rafieetery, Peter Veldman, Walt Whitley, Lynn Lawrence, Valerie Manso, Sharon Carter. CE hours: 331 total, 41per OD. To register, email etaylor@secostaff.com or go to www.seco2017.com.

■ **2-4.** *2017 Winter Conference.* Big Sky Conference Center, Big Sky, MT. Hosted by: Montana Optometric Association. Key faculty: Andrew Morgenstern, Jay Haynie. CE hours: 13. To register, email Marti Wangen at mwangen@rmsmanagement.com, call (406) 443-1160 or go to www.mteyes.com.

■ **3-4.** *Borish Symposium.* Bloomington, IN. Hosted by: IU School of Optometry. CE hours: 16. To register, email Cheryl Oldfield at coldfiel@indiana.edu, call (812) 856-3502 or go to www.optometry.iu.edu/continuing-education.

■ **4.** *AZ-AAO Chapter Spring Meeting 2017.* Midwestern University, Arizona College of Optometry, Glendale, AZ. Hosted by: Arizona Chapter of American Academy of Optometry. CE hours: 6. To register, email arizona.aaopt@gmail.com or go to www.aaopt.org/azchapter.

■ **5.** *Illinois Optometric Association Winter CE Series.* Hyatt Regency O'Hare, Rosemont, IL. Hosted by: Illinois Optometric Association. Key faculty: Steve Ferrucci. CE hours: 6. To register, email Charlene Marsh at ioabb@ioaweb.org, call (217) 525-8012 or go to www.ioaweb.org.

■ **5-6.** *COVD at SECO.* Georgia World Congress Center, Atlanta, GA. Hostd by: College of Optometrists in Vision Development. Key faculty: Kellye Knueppel, Brenda Montecalvo. CE hours: 12. To register, email Penny at penny@covd.org or go to www.covd.org.

■ **5-6.** *Great Lakes Optometric Congress.* Northbrook, IL. Hosted by: Optometric Extension Program Foundation. CE hours: 13. To register, email jeffgetzell@sbcglobal.net or go to www.oepf.org/oepf_calendar.

■ **5-10.** *EyeSki Conference.* The Lodge of Mountain Village, Park City, UT. Hosted by: EyeSki. Key faculty: Thomas Arnold, James Fanelli, Mile Brujic, Leonard Messner, Joseph Pizzimenti. CE hours: 20. To register, email Timothy Kime at tandbkime@bex.net, call (419) 475-6181 or go to www.eyeskiutah.com. ■

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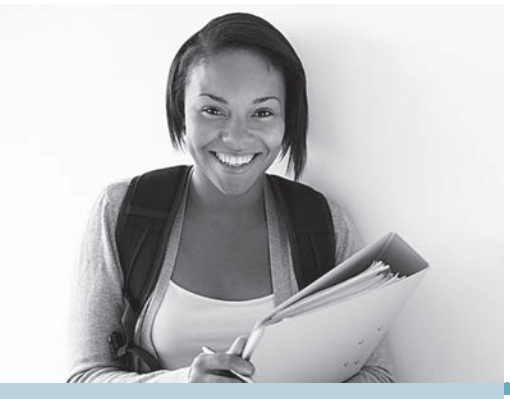

## *About Rick*

Rick Bay served as the publisher of The *Review* Group for more than 20 years.

To those who worked for him, he was a leader whose essence was based in a fierce and boundless loyalty.

To those in the industry and the professions he served, he will be remembered for his unique array of skills and for his dedication to exceeding the expectations of his customers, making many of them fast friends.

Scholarships are awarded to advance the education of students in both ***Optometry*** and ***Ophthalmology***, and are chosen by their school based on qualities that embody Rick's commitment to the profession, including integrity, compassion, partnership and dedication to the greater good.

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Diagnostic **Quiz**

# Red-eyed and Blue

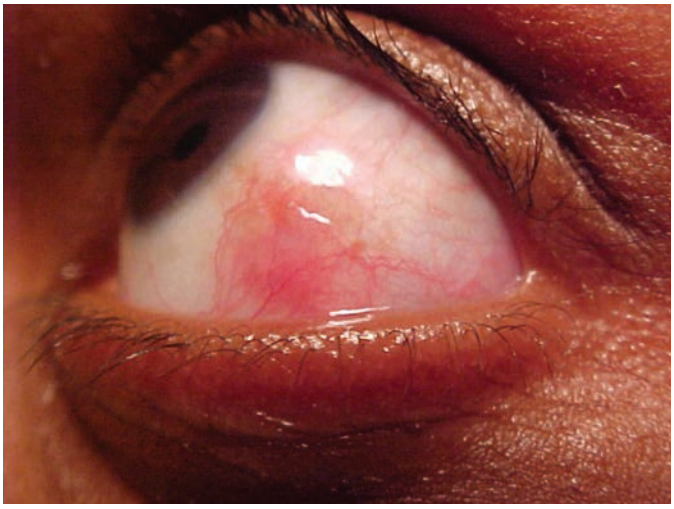
**By Andrew S. Gurwood, OD**

## History

A 22-year-old black female reported to the office with a chief complaint of pain in her left eye for one week. She explained that it started hurting after she participated in an experiment involving the installation of electrodes to her left eye. Her systemic and ocular histories were unremarkable. She denied allergies of any kind.

## Diagnostic Data

Her best-corrected entering visual acuities were 20/20 OD and 20/20 OS at distance and near. Her external examination demonstrated observable injection OS with pain upon ocular movement OS. There was no evidence of afferent pupil defect. The pertinent biomicroscopic examination of the anterior segment, OS, is demonstrated in the photograph. There was no frank cell and flare OU. Goldmann applanation tonometry measured 15mm Hg OU. The dilated examination found no peripheral pathologies OU.



**This 22-year-old patient had been experiencing eye pain for about a week. In addition to her history, can this photo help you uncover the cause of her discomfort?**

## Your Diagnosis

Does this case require additional tests? How would you manage this patient? What is the likely prognosis? To find out, please visit www.reviewofoptometry.com. ■

**Retina Quiz Answers** *(from page 94):* 1) C; 2) C; 3) D; 4) B; 5) A.

## Next Month in the Mag

In March, *Review of Optometry* will focus on a clinician's responsibilities related to diagnostic skills and techniques. Topics will include:

- *Develop the Skills to Protect Yourself from Medicolegal Risks for Failure to Diagnose*
- *Point/Counterpoint: Ultra Widefield Imaging vs. Dilated Funduscopy*
- *Condensing Lenses: Sharpen Your Skills in Choosing and Using*
- *Learning to Recognize Red Disease: False Positives in Glaucoma Diagnoses*
- *Retinal Vasculature Miniseries Part 3: The Many Ways to Apply OCT-A*

Also in this issue:

- *The Online Refraction Threat: Are You Prepared?*
- *Injectable Medication Use in Optometry* (earn 2 CE credits)

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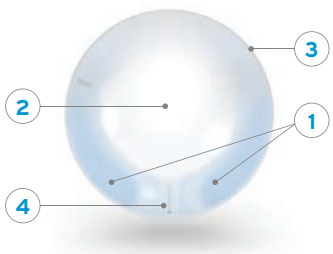


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**REFERENCES: 1.** Data on file. Bausch & Lomb Incorporated. Rochester, NY; 2012. **2.** Results from a 7-investigator, multi-site, randomized, crossover study of Biotrue® ONEday for Astigmatism and control contact lenses on 123 current non-daily disposable toric soft contact lens wearers. Each lens brand was worn on a daily wear basis for 1 week.



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**References: 1.** Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci.* 2010;87:E-abstract 105110. **2.** Nash WL, Gabriel MM. Ex vivo analysis of cholesterol deposition for commercially available silicone hydrogel contact lenses using a fluorometric enzymatic assay. *Eye Contact Lens.* 2014;40(5):277-282. **3.** *In vitro* study over 16 hours to measure wetting substantivity; Alcon data on file, 2015. **4.** *In vitro* wetting analysis: out-of-pack and wetting substantivity; Alcon data on file, 2014.


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