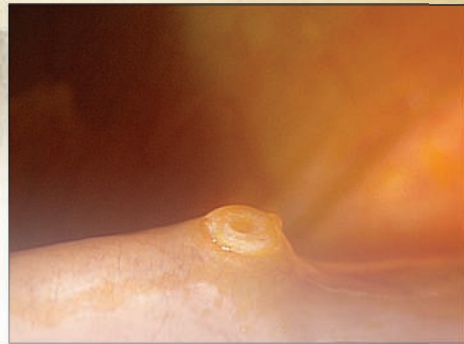


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Part 1 of 2

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IN THE NEWS

Nanoparticles called dendrimers offer a novel route into the retina for **age-related macular degeneration** and **retinitis pigmentosa** treatments, according to new research. Investigators at the Mayo Clinic, Wayne State University and Johns Hopkins Medicine found that steroids attached to the dendrimers target the damage-causing cells associated with neuroinflammation, preserving vision and leaving the rest of the eye unaffected. The results appear in the January issue of *Biomaterials*.

The FDA has launched a monitoring program to prevent outbreaks of **toxic anterior segment syndrome (TASS)**—a rare but potentially serious complication of cataract surgery. Working with the Centers for Disease Control and Prevention and the American Academy of Ophthalmology, the FDA has established a registry for devices used in cataract surgery and a program for identifying and evaluating suspected device contaminants.

Elderly patients who took the carotenoid **zeaxanthin** showed improvements in vision, according to results from the **Zeaxanthin and Visual Function Study**, reported in the November 2011 issue of *Optometry*. The study included 60 predominantly male veterans who had mild-to-moderate AMD. They were randomized to receive 8mg zeaxanthin, 8mg zeaxanthin plus 9mg lutein, or 9mg lutein for one year. Researchers concluded that, independent of lutein, zeaxanthin contributed to improved vision in night driving, fine detail and the disappearance of blind spots.

Drug Slows Metastasis Of Uveal Melanoma

A drug approved for epilepsy can also slow the spread of these deadly eye tumors. **By Michael Hoster, Senior Editor**

A type of drug that is commonly used to treat epileptic seizures may slow or prevent uveal melanoma from metastasizing to other parts of the body, according to a study in the October 28 online version of *Clinical Cancer Research*.

The researchers determined that FDA-approved anti-epileptic drugs known as histone deacetylase (HDAC) inhibitors alter the way that the aggressive form of uveal

ences and professor of biology and molecular oncology at Washington University School of Medicine in St. Louis. “When we look at aggressive melanoma cells under the microscope after treatment with HDAC inhibitors, they look more like normal cells and less like tumor cells.”

Treatment with HDAC inhibitors may allow patients with such aggressive melanomas to live for many years without any detectable metastasis, Dr. Harbour predicted.

“Melanoma in general, and uveal melanoma in particular, is notoriously difficult to treat once it has metastasized and grown in a distant organ,” he added. “I suspect that the best role for HDAC inhibitors will be to slow or prevent the growth of tumor cells that have spread out of the eye, but cannot yet be detected. This might lengthen the time between the original eye treatment and the appearance of detectable cancer in the liver and elsewhere.”

In addition, HDAC inhibitors cause only relatively mild side effects such as drowsiness, Dr. Harbour explained.

Clinical trials on patients with metastatic uveal melanoma could begin within the next six to 12 months, he noted.

Landreville S, Agapova OA, Matatall KA, et al. Histone deacetylase inhibitors induce growth arrest and differentiation in uveal melanoma. *Clin Cancer Res*. 2011 Oct 28. [Epub ahead of print]

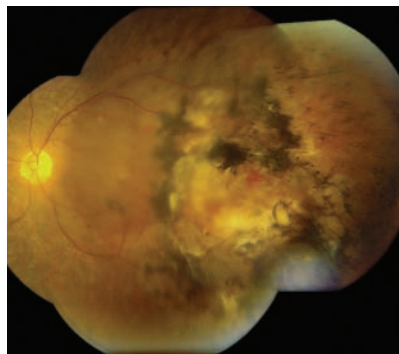


Photo: Mark T. Dunbar, O.D.

melanoma expresses its key genes. According to the researchers, uveal melanoma is very aggressive and typically spreads from the eye to other bodily organs, such as the liver.

“HDAC inhibitors appear to reverse the aggressive molecular signature that we had identified several years ago as a marker for metastatic death,” said lead researcher J. William Harbour, M.D., distinguished professor of ophthalmology and visual sci-

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Myopia Treatments for Kids Come Up Short or Cause Side Effects

Current treatments to slow the progression of myopia in children either don't work or cause problematic side effects, according to a recent review published by pediatric eye doctors and study methodologists.

The reviewers analyzed data from 23 randomized controlled trials, which included a total of 4,696 participants. They considered a number of potential myopia treatments including bifocal glasses, eye drops, intraocular pressure-lowering drugs and contact lenses:

- Two studies investigated undercorrection of myopia.
- 12 studies investigated multifocal spectacles (progressive addition lenses [PALs] or bifocal spectacles).
- One study investigated bifocal



Photo: National Eye Institute-National Institutes of Health

Of all methods of myopia inhibition, anti-muscarinic drops work the best, but their use is limited and causes side effects.

soft contact lenses (BSCLs).

- Two studies investigated rigid gas permeable contact lenses (RGPCLs).
- Six studies investigated phar-

maceutical eye drops (five of these were of anti-muscarinic medications).

- One study investigated new lenses designed to reduce peripheral hyperopic defocus (i.e., lenses that help to focus peripheral vision as well as central vision).

- One study evaluated both multifocal lenses and pharmaceutical eye drops.

The follow-up period was at least one year for all studies.

Of all the treatments, anti-muscarinic eye drops offered the largest positive effects for slowing myopia progression, the authors found, but they caused either light sensitivity or blurred near vision. Also, these drops are not yet commercially available, so their use is limited and impractical.

PALs and bifocal spectacles were found to yield a small slowing of myopia progression. RGPCLs were found to have no evidence of effect on myopic eye growth, while undercorrection of myopia was found to increase myopia progression slightly.

Lastly, "other methods of myopia control, such as the use of corneal reshaping contact lenses or bifocal soft contact lenses (BSCLs) with a distance center, are promising but currently no published randomized clinical trials exist," the authors concluded.

An overview of the report is available at: <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD004916.pub3/pdf/abstract>.

Walline JJ, Lindsley K, Vedula SS, et al. Interventions to slow progression of myopia in children. Cochrane Database Syst Rev. 2011 Dec 7;12:CD004916.

Eyes in the Back of Its Head

Biologists at Tufts University were able to make tadpoles grow eyes on their backs and tails by genetically manipulating the membrane voltage of cells in frog embryos.

This new mechanism could have great potential in the formation of complex organs for transplantation and regenerative medicine applications.

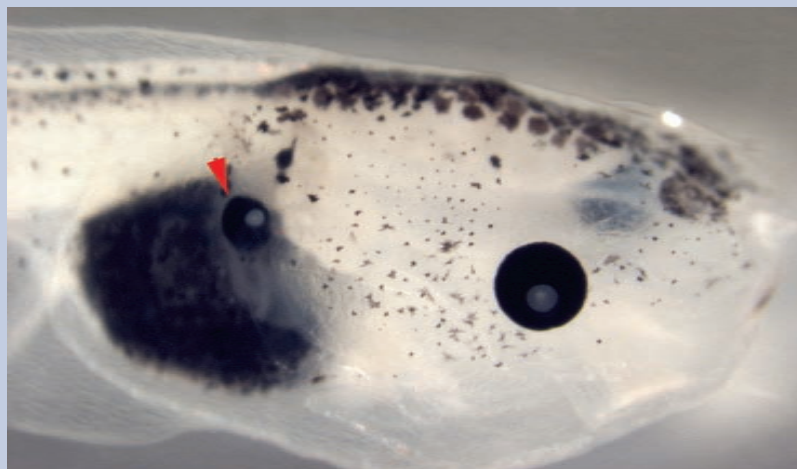


Photo: Sherry Aw, PhD, and Michael Levin, PhD

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New Online Tool Calculates Risk of AMD Progression

A new, online risk assessment model will help eye care providers more easily identify whether patients with AMD will experience disease progression and/or visual compromise.

Michael Klein, M.D., and associates at the Casey Eye Institute at the Oregon Health and Science University in Portland used phenotypic, demographic, environmental and genetic risk factors—in particular visual data from 2,846 patients enrolled in the Age-Related Eye Disease Study (AREDS)—to design this risk assessment model.¹

The researchers evaluated longitudinal data from AREDS participants who had all levels of AMD, ranging from none to unilateral advanced AMD.

With this information in hand, they performed “a Cox proportional hazards analysis with demo-

graphic, environmental, phenotypic and genetic covariates.”¹

The final model takes into account these independent variables: age; smoking history; family history of AMD (first-degree member); phenotype based on a modified AREDS simple scale score; and genetic variants CFH Y402H and ARMS2 A69S.

After testing the calculator’s validity, the authors wrote, “the model did well on performance measures, with very good discrimination and excellent calibration and overall performance. Successful external validation was performed, and a risk assessment tool was designed for use with or without the genetic component.”

They conclude, “We believe our current model is of substantial value in assessing AMD risk, and we expect that future advances will further improve its accuracy.”

Their findings appear in an article published in the December 2011 *Archives of Ophthalmology*.

In a related editorial, Ronald Klein, M.D., M.P.H., professor in the department of ophthalmology and visual sciences at the University of Wisconsin School of Medicine and Public Health, and associates commented on the clinical significance of Dr. Michael Klein’s predictive model.² “Knowing the severity of the lesions of AMD that are already present, coupled with knowledge of important lifestyle factors, gives most of the important information about risk of progression.”

The AMD Risk Assessment Calculator is available at: <http://caseyamdcalc.ohsu.edu>.

1. Klein ML, Francis PJ, Ferris FL 3rd, et al. Risk assessment model for development of advanced age-related macular degeneration. *Arch Ophthalmol*. 2011 Dec;129(12):1543-50.
2. Klein R, Klein BE, Myers CE. Risk assessment models for late age-related macular degeneration. *Arch Ophthalmol*. 2011 Dec;129(12):1605-6.

Sunshine Act Delayed—for Now

Proposed regulations from the Centers for Medicaid and Medicare Services will postpone data collection under the federal Physician Payment Sunshine Act until later this year, after the final regulations have been issued. The “physician” discussed in the ruling includes ophthalmologists and optometrists, but does not address opticians.

Part of the 2010 health care reform package, the Sunshine Act requires drug, medical device and other manufacturers to collect data on payments to physicians

and teaching hospitals and to annually report those numbers to CMS. It also requires these manufacturers, as well as drug and device supplier group purchasing organizations, to annually report physician ownership and investment interests.

The proposed rule states that CMS is considering requiring manufacturers to start tracking payments 90 days after the publication of the final rule, with a partial year report due on March 31, 2013. However, the agency is seeking comments on whether that

timeline is feasible.

The consequences of not reporting are significant, including civil fines and penalties from \$1,000 for a simple inaccuracy, up to \$1 million for a knowing failure to report.

The proposed regulations define several key terms, describe the reporting process and ask for feedback on several key issues. Comments must be submitted by February 17, 2012.

To submit a comment, go to www.regulations.gov/#!documentDetail;D=CMS-2011-0191-0004.

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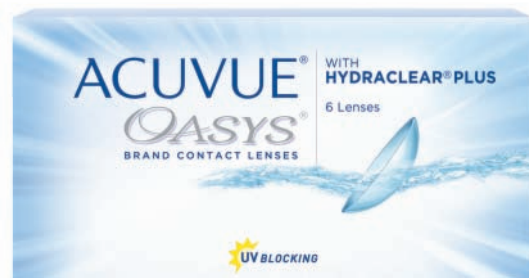
References: 1. Data on file. Johnson & Johnson Vision Care, Inc. 2009. 2. Data on file. Johnson & Johnson Vision Care, Inc. 2008.

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Location and Temperature Are Risk Factors for Exfoliation Glaucoma

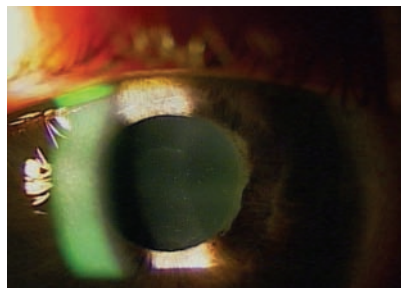
Geographic latitude and outside air temperature—not ethnicity—could be potential risk factors for the development of exfoliation syndrome (ES), according to a study published in the January issue of *Ophthalmology*.

Exfoliation syndrome is the leading cause of secondary open-angle glaucoma, and may also contribute to cataract formation.

In this study, researchers from the Massachusetts Eye and Ear Infirmary at Harvard Medical School examined the records of 78,955 females enrolled in the Nurses' Health Study and 41,191 males enrolled in the Health Professionals Follow-up Study for descriptive epidemiologic features of ES.

The researchers determined that:

- Females were at a higher risk for ES.
- Individuals with a positive



Exfoliation glaucoma.

family history of glaucoma were more than twice as likely to develop ES.

- Individuals of Scandinavian or Southern European descent are not genetically predisposed to the development of ES.
- Iris color was not a risk factor for ES.

“This large, prospective cohort study demonstrates that there is a positive association between latitude and ES risk that is robust and not related to demographic features or other systemic covariates,” said study co-author Louis

Pasquale, M.D., director of Massachusetts Eye and Ear.

Once the researchers realized that ethnicity was not a significant risk factor for ES, they suggested that the disease might have an environmental component. “Importantly, those with a lifetime residential history of living in the middle tier and south tier of the United States was associated with 47% and 75% reduced risks, respectively, compared with living in the northern tier,” the authors wrote.

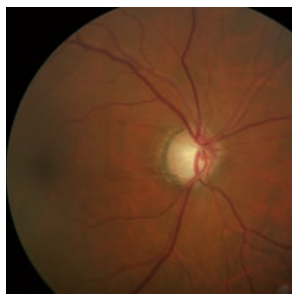
Another manuscript they recently published suggests that lower ambient temperature interacts with increased solar exposure to heighten the risk of ES.

More research needs to be done to determine how these environmental factors contribute to ES, they add.

Kang JH, Loomis S, Wiggs JL, et al. Demographic and geographic features of exfoliation glaucoma in 2 United States-based prospective cohorts. *Ophthalmology*. 2012 Jan;119(1):27-35.

It's Glaucoma Awareness Month

January is the perfect time to raise awareness in your community about the prevalence of glaucoma and the importance of making eye health a priority—particularly while those New



Year's resolutions to take better care of their health are still fresh. Encourage those at risk to get a comprehensive dilated eye exam,

especially African Americans over age 40; people over age 60, especially Mexican Americans; and those with a family history of the disease.

Stress that early detection and treatment is the best way to

prevent vision loss from this widespread disease. There are a number of educational resources about glaucoma that you can share with

your patients, including:

- Prevent Blindness America's Glaucoma Learning Center: www.preventblindness.org/glaucoma.
- The Glaucoma Research Foundation: www.glaucoma.org.
- The Take on Glaucoma website: www.takeonglaucoma.com.
- The National Eye Institute: www.nei.nih.gov/health/glaucoma.
- The Centers for Disease Control and Prevention's Vision Health Institute: www.cdc.gov/visionhealth.



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Study: Perform Visual Fields Every Six Months

Twice-a-year visual field testing, compared to once-a-year testing, leads to earlier detection of glaucoma progression for high-risk patients, especially using global trend analyses, according to researchers at the Jules Stein Eye Institute.

“This finding has significant implications for the care of patients with glaucoma,” the authors wrote in an article in the December issue of *Archives of Ophthalmology*.

Using data from the Advanced Glaucoma Intervention Study (AGIS), investigators gathered the visual field examinations of 468 eyes (381 patients) with primary open-angle glaucoma, which was no longer controlled by maximum medical treatment.

The researchers then created two data sets. The first set, which included twice-yearly AGIS field test results, was labeled as the high-frequency testing group. To create the second data set to represent low-frequency field testing, the researchers deleted every other test of the AGIS participants. The high-frequency group had a median of 20 visual field examinations and the low-frequency group had a median of 12.

“The high-frequency data set was more likely to detect progression with mean deviation or pointwise linear regression criteria,” they found.

More specifically, using mean deviation as a criterion, 43.6% (204 eyes) in the high-frequency data set progressed while 34.2% (160 eyes) in the low-frequency data set progressed. Alternatively, using pointwise linear regression as a criterion, 39.5% (185 eyes) in the high-frequency data set and 35.7% (167 eyes) in the low-frequency data set progressed.

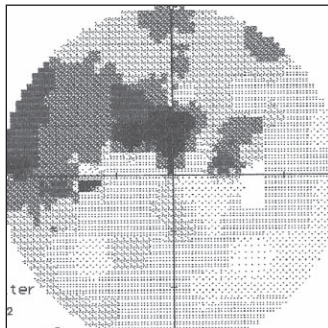
“The hazard ratios for progression are large enough, especially for [mean deviation] criteria, for the benefits to be

considered worth the extra time and expense required for earlier detection of glaucoma progression, at least in a subset of patients at higher risk of progression,” the researchers wrote.

While three examinations per year have been recommended for optimal detection of glaucoma progression, no evidence-based data supports such a recommendation, the authors wrote. And, they add, only a minority of patients have disease that progresses at a rate fast enough to justify thrice-yearly testing.

Such a “testing strategy is cumbersome and is not practical under most clinical circumstances,” they wrote. “Our data provide evidence for the advantages of a more practical six-month schedule for visual field testing.” ■

Nouri-Mahdavi K, Zarei R, Caprioli J. Influence of visual field testing frequency on detection of glaucoma progression with trend analyses. *Arch Ophthalmol*. 2011 Dec;129(12):1521-7.



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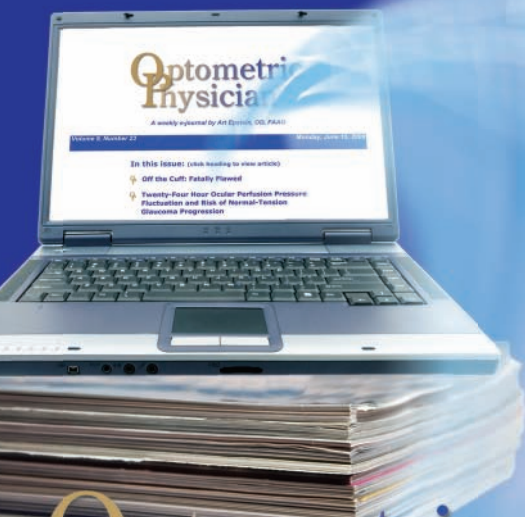
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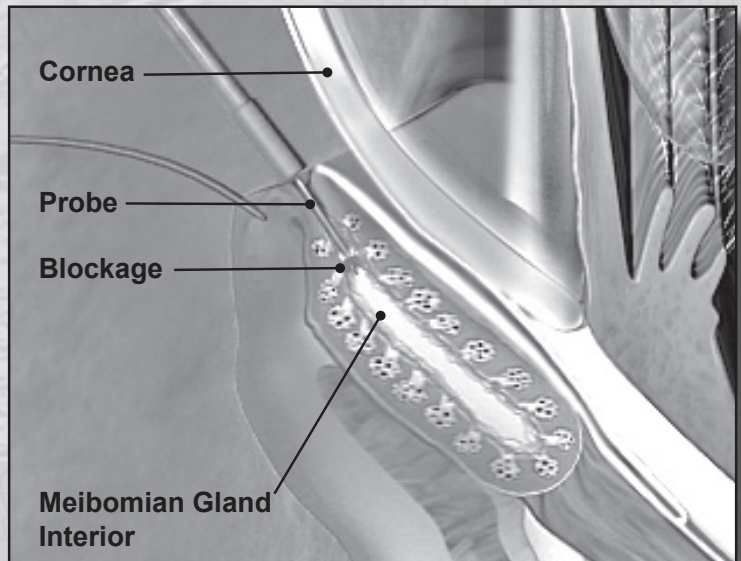


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Maskin, Steven L, Cornea. 29(10):1145-1152, October 2010.

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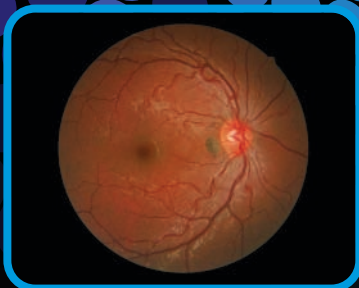
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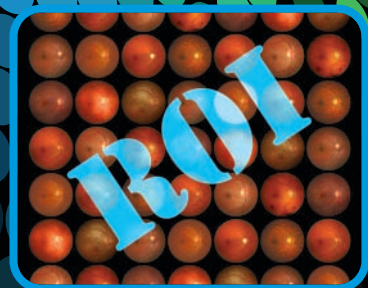
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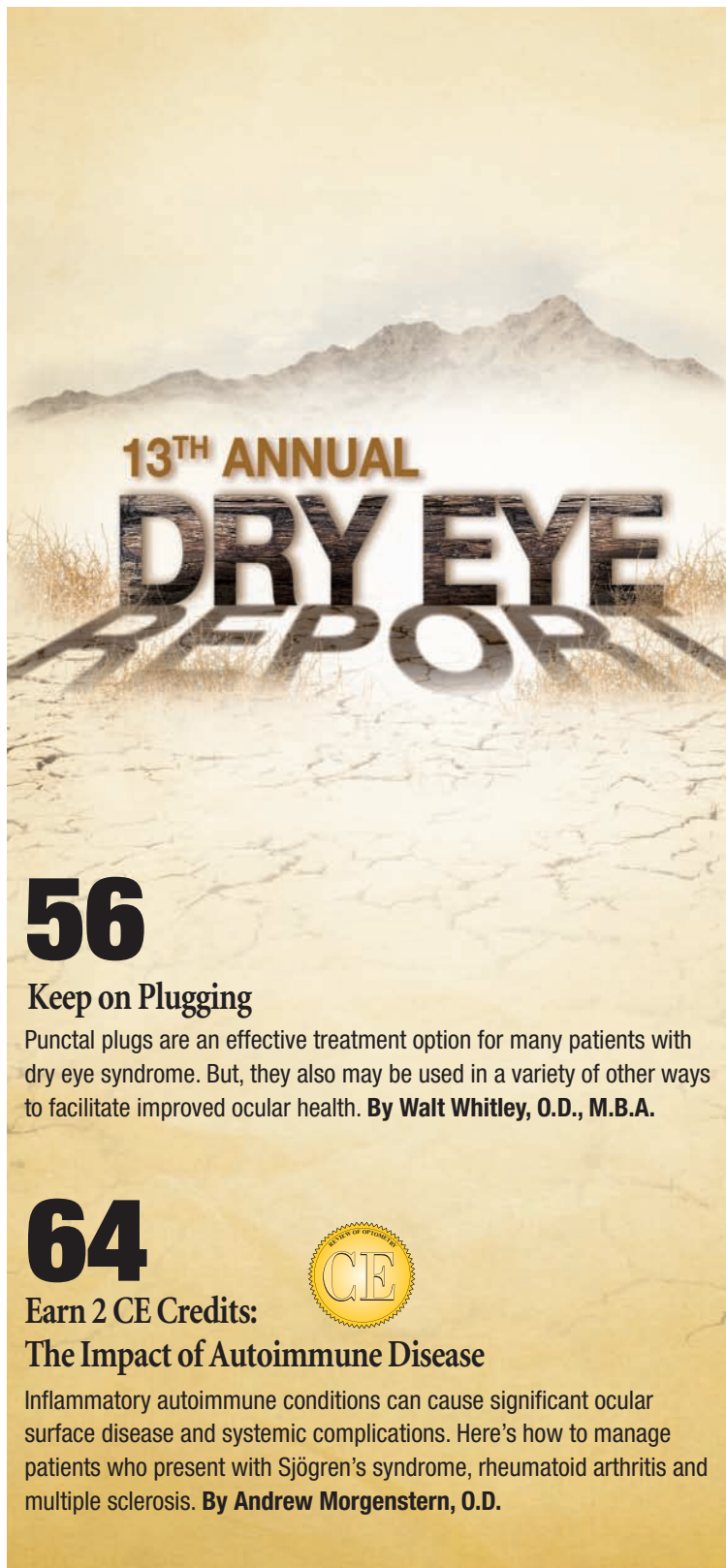
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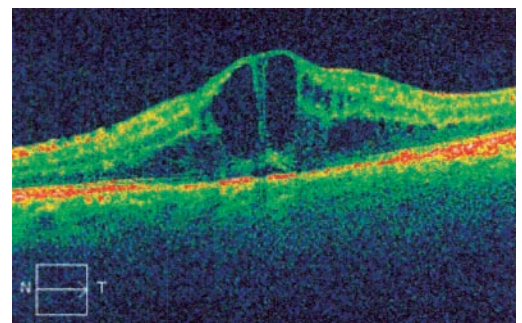
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Ocular coherence tomography has become an all-around useful tool for glaucoma, retina and even anterior seg evaluation. Not every office needs one, but we certainly couldn't do without ours.

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Choroidal Melanoma is a Life Sentence

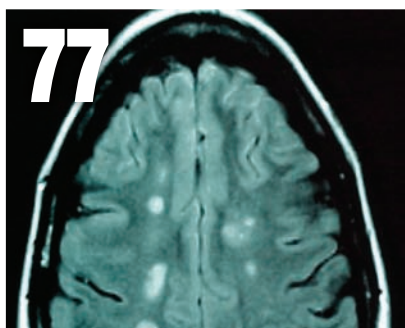
A thorough knowledge of treatment options and associated risks is crucial to ensure the best possible outcome with this dire condition.

By Sara Weidmayer, O.D.

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Brief Summary of Prescribing Information

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LACRISERT® (hydroxypropyl cellulose) OPHTHALMIC INSERT

DESCRIPTION

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

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

INDICATIONS AND USAGE

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

CONTRAINDICATIONS

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

WARNINGS

Instructions for inserting and removing LACRISERT should be carefully followed.

PRECAUTIONS

General

If improperly placed, LACRISERT may result in corneal abrasion.

Information for Patients

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

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

Drug Interactions

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

Carcinogenesis, Mutagenesis, Impairment of Fertility

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

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

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

ADVERSE REACTIONS

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

DOSAGE AND ADMINISTRATION

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

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

Issued June 2007

Distributed by:

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

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

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

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

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

References: **1.** Koffler BH, McDonald M, Nelinson D, Improved signs and symptoms and quality of life with dry eye syndrome: hydroxypropyl cellulose ophthalmic insert patient registry. *Eye Contact Lens*. 2010;3:170-176. **2.** LACRISERT [package insert] Madison, NJ: ATON Pharma, 2009. **3.** Wander A, Koffler B. Extending the duration of tear film production: review and retrospective case series study of the hydroxypropyl cellulose ophthalmic insert. *Ocul Surf*. 2009;7(3e):154-162.


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Optometry's Sharper Image

If pointing a needle at my eye requires the skill of a surgeon, why isn't there more outrage directed toward cosmetologists and tattoo artists who apply permanent makeup? **By Amy Hellem, Editor-in-Chief**

The use of injectable medications is a touchy subject and the source of ongoing dispute and deliberation.

Currently, optometrists in 35 states are permitted to administer drugs via injection; yet many well-trained, prominent O.D.s opt not to do so. Their reasons are varied and are of particular interest to many ophthalmologists who wish to retain sole ownership of injectable territory.

While I would never criticize an optometrist for steering clear of a procedure that he or she is not comfortable performing, the hushed debate got me wondering what everyone's really so afraid of—is it the needle itself, or what's inside that sparks the quarrel and hesitation?

Much of what you would potentially administer via injection is already part of your frequently-used armamentarium of pharmaceutical agents. But there are some key points to remember when administering an injection. Several years ago, James Fanelli, O.D., pointed out the following in a *Review of Optometry* article:¹

"Injectable medications have one very important and striking difference compared with both topicals and orals: The body absorbs injectables faster than other routes of administration. Faster absorption means that the desired action of the medication occurs more quickly, but it also means that untoward side effects also occur more rapidly."

While the lion's share of an

optometrist's experience is in prescribing topicals, optometrists are trained extensively on the use of a whole spectrum of pharmacologic agents—including systemic and injectable medications. And, to be fair, understanding absorption rates is not rocket science—particularly to trained doctors. So again, I ask, what is everyone so afraid of?

Perhaps the reason why ophthalmologists so fiercely attack optometry's efforts to gain injection privileges is because placing a needle anywhere near the eye is dangerous business. Certainly, states should take a close look at your training before deciding whether you are qualified to take a sharp object full of medicine or dye and inject it into a patient or consumer. That's why I was so surprised when I went shopping online for some less irritating eye makeup that wouldn't leave me rubbing my itchy lids all day. During my quest, I stumbled upon several local businesses that will tattoo permanent eyeliner along my lash line for about \$250.

Excuse me? Apparently, the great state of Pennsylvania has placed a higher educational value on its cosmetology schools than on graduates of the esteemed Salus University. That's right. Don't inject that chalcidion with a steroid, but by all means, take a needle to my lid and pierce my skin while injecting me with unregulated chemicals.

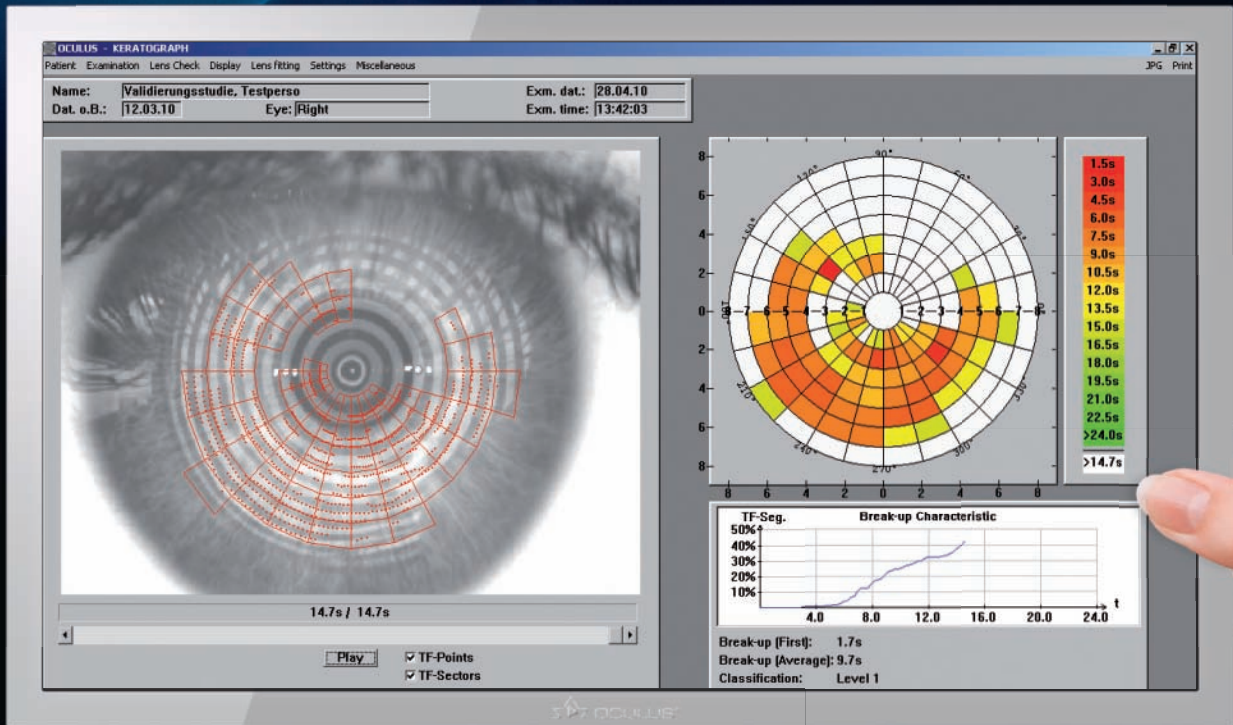
Actually, I take that back, the chemicals are—or should be—regu-

lated. According to the FDA's website: "FDA considers the inks used in intradermal tattoos, including permanent makeup, to be cosmetics and considers the pigments used in the inks to be color additives requiring premarket approval under the Federal Food, Drug, and Cosmetic Act."² They go on, however, to say that they don't traditionally exercise their authority over tattoo inks or the pigments used in them, even though they recognize the fact that "many pigments used in tattoo inks are not approved for skin contact at all" and "some are industrial grade colors that are suitable for printers' ink or automobile paint."²

I applaud the optometrist who knows his or her own limits and chooses not to go down the road of gaining injection privileges. That's not what I'm afraid of. I'm afraid of the system, which obviously ignores the differences between trained health care professionals and unlicensed tradesmen, and focuses instead on amplifying labels, rather than considering the training that's behind them.

Amy Hellem
Editor-in-Chief

1. Fanelli JL. The use of injections in primary care. *Rev Optom*. 2002 Nov;139(11):70-81.
2. The U.S. Food & Drug Administration. Tattoos & Permanent Makeup. Available at: www.fda.gov/cosmetics/producingredientssafety/productinformation/ucm108530.htm (accessed January 4, 2012).



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I'm Shootin' Blanks Here

Staring at a blank page reminds me of all the times I've come up blank. Sometimes, patients still catch me off guard without a response. **By Montgomery Vickers, O.D.**

The dreaded blank page! My editor has just reminded me that I need to send him my column for this month. I assured him that I would do so and that this event would occur just about any minute. Then, I stared at this ... blank page.

It reminds me of the many, many moments in my life when I came up blank. We've all done that. The first time I can recall "going blank" was when I was in the ninth grade and Miss Ollem said, "Just hand me your research papers at the end of class today."

Research paper? What research paper? I went blank. Perhaps I passed out. But when the room stopped spinning, my instincts kicked in and I immediately grasped the fact that one must quickly and confidently fill any blank page within reach. And—this is simple but critically important—it really matters NOT how you fill it. Just fill it!

Before long, Miss Ollem read the finest research paper ever written on one of the most important subjects of that time: The Beatles.

Another time that I went blank was in optometry school. One of the professors called me into his office, sat me down and asked me what I planned to do when I left optometry school. I proudly stated: "I will be an eye doctor!" He sighed and said, "No. Really."

OK, that left a blank I was more than a little hesitant to fill, because he knew for a fact that I had been playing darts with my buddy Big

Al instead of preparing for class. I knew he was right and I promised him that I would do all I could to improve my attitude, my study efforts and my dart game as well. My dart game did in fact improve, and I did graduate from optometry school, which proves there is a God.

Patients really find ways to make you go blank. Even after 32 years, they continue to astound me. Generally, I'm astounded in a good way, like when the 45-year-old grandmother told me she would prefer that I not dilate her eyes because she was pregnant. That deserved some combination of awe and joy and terror. I filled the blank with congratulations, grinning, giggling and breaking into a cold sweat.

Then, what do we do when the patient announces something horrible ... loss of a child ... divorce ... cancer? Is there any possible way to fill that blank? Not with words, and for a big mouth like me, that's tough. So, I fill it with a touch. Just a touch.

Those of you who have seen me perform my optometric stand-up comedy routine (complete with optometric musical interludes) would

not believe this—but I get stage fright. So, I've simply convinced myself that sheer terror is actually physiologically identical to extreme pleasure. This little tip may come in handy when you get tongue-tied taking an oral or practical board examination as optometry evolves into its future. Here's how to fill up that blank page that is your brain.

Examiner: "Describe the differential diagnosis of Posner-Schlossman syndrome and its relationship to Behçet's disease."

You: "I just peed myself, so I must be thrilled."

You may not pass the test, but you'll think that you are filled with joy. That's nice, right?

Oh, I just noticed—I've filled this blank page! So here is my column that I've been working on diligently since last month's column, Mr. Editor. Gotta go now. It's darts night. ■



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Happy(?) New Year!

Well, here we go again. A new year is just beginning, but we're still dealing with problems from previous years. **By John Rumpakis, O.D., M.B.A., Clinical Coding Editor**

I was hoping that by the time I wrote this, there would be some long-term clarity on Medicare's Physician Payment Schedule for 2012 and beyond.

Unfortunately, there isn't. Here is what we know so far...

On December 23, all physicians—including optometrists—got a brief reprieve from a 27% Medicare pay cut scheduled to go into effect on January 1, because the U.S. House of Representatives reached an agreement with the Senate on a two-month extension of important policies. The Sustainable Growth Rate (SGR) cut to physicians servicing Medicare payments is not going to happen, at least for another month or so, because the current fee structure will remain in place until the end of February 2012.

A House-Senate conference committee convenes this month to work on a longer-term agreement. However, this will not happen quickly because the House isn't scheduled to return to Washington until January 17, while the Senate won't return until January 23. The goal is to extend all the expiring programs for a full year—except for the physician payment cut reprieve, which is to be extended for two years until a better long-term solution can be found.

Additionally, the Centers for Medicare & Medicaid Services (CMS) have extended the annual Medicare participation enrollment period through February 14. The



previous deadline was December 31. The effective date for any participation status change during the extension, however, remains January 1, and will be enforced for the entire year. According to CMS, contractors will accept and process any participation elections or withdrawals made during the extended enrollment period that are post-marked on or before February 14.

The Good and The Bad

On January 1, the relative values (RVUs) for many codes commonly used by optometrists to report the physician services they provide to Medicare patients were scheduled to increase. However, these increases will not be realized on January 1 because of the 0% carry-forward of the existing fee schedule. CMS could not put into effect only the changes in RVUs without incorporating the entire impact of the SGR formula, and that meant taking the 27.4% cut as well.

The professional political organizations, such as the American Medical Association and American

Optometric Association, are advocating their members to urge Congress and the President to support doctors and patients by providing stability to the Medicare physician payment system. The threatened cut must be corrected without any further delays and a bipartisan initiative to repeal the flawed and disruptive SGR formula that is causing this crisis should be put in place.

The AOA is specifically requesting doctors and students to support the AOA's direct advocacy efforts on Capitol Hill by contacting their Senators and House members through AOA's Online Legislative Action Center (<http://app1.vocusgr.com/WebPublish/Controller.aspx?SiteName=AOAGR&Definition=Issues&Juris=US>).

More To Come

2012 should prove to be an interesting year with concerns about E-prescribing penalties, PQRS incentives, audit concerns, new CPT and ICD-9 codes just to name a few. I look forward to keeping all of you informed and engaged. ■

Don't forget to send your questions and comments to CodingAbstract@gmail.com.

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¹ The Ocular Surface. April 2007, 5(2).

² modified from: Gilbard JP, Rossi SR Ophthalmol, Apr 1992, 99(4):600-4.

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TheraTears[®] — Complete Dry-Eye Relief for Everyone

Seeing the Future with Contact Lenses

With advances in technology and improvements in materials, contact lenses aren't just for vision correction anymore. **By David L. Kading, O.D., and Katherine Shen, O.D.**

More than 24 million Americans wear contact lenses—but we could see that number skyrocket even higher with the advent of contact lenses that do more than offer vision correction.¹ Imagine a day when you could use contact lenses to monitor and deliver medication to your glaucoma patients, or treat your allergy or dry eye patients. Better yet, imagine if you were able to repair a damaged cornea and restore vision noninvasively by using stem cells embedded on a contact lens.

You won't have to stretch your imagination too far because researchers are already developing unique contact lens designs that could achieve those aims and more. In a few years, you might even find yourself fitting contact lenses on patients with perfectly healthy vision who aren't in need of any correction, but rather are looking for convenience. As technological advancements continue to revolutionize the contact lens industry, these visions will soon become realities.

Disease Monitoring

In recent years, several researchers have been looking into whether contact lenses could provide a noninvasive means of monitoring blood glucose levels or intraocular pressure that improves comfort and convenience without sacrificing accuracy.

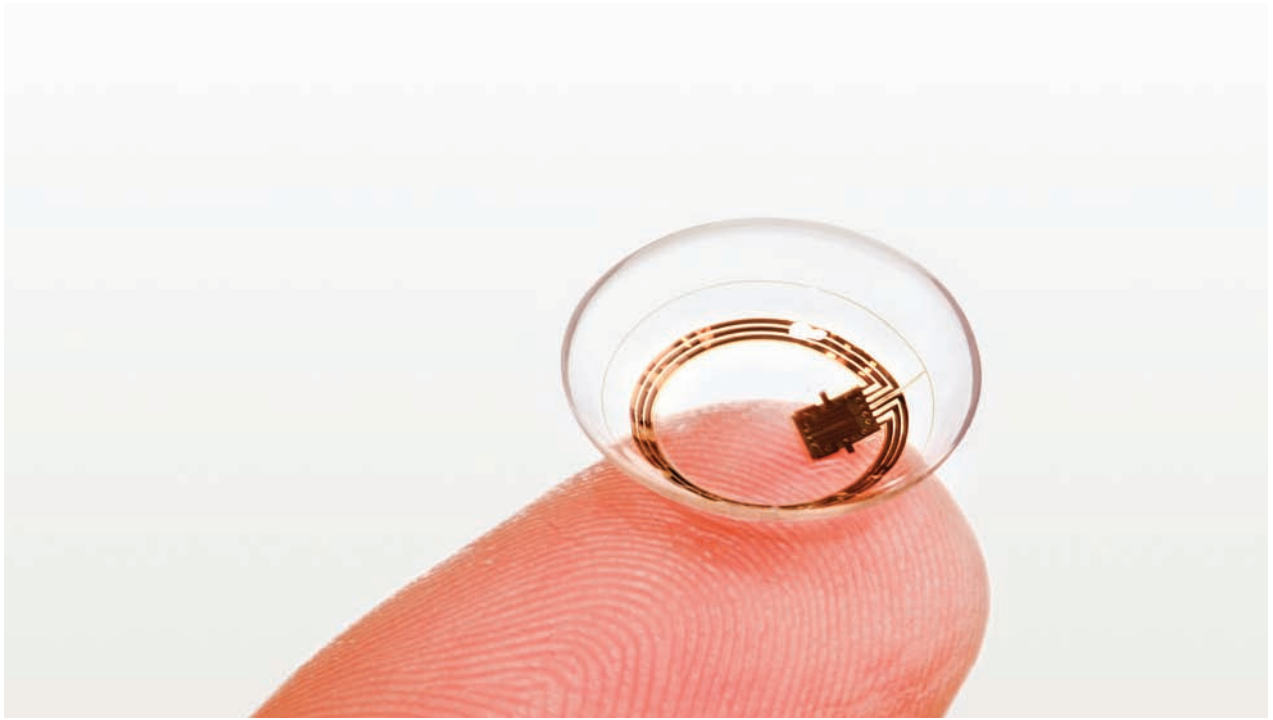
- **Blood glucose.** A professor at the University of Western Ontario has been developing a contact lens to measure glucose levels using tear film on the eye.² The lens contains nanoparticles that give it a reddish hue when exposed to a certain concentration of glucose, which would alert wearers to adjust their blood sugar.²

The lenses have been successfully tested in the lab using artificial tears, but there's still much work to be done—including developing a portable reader to provide specific measurements and an understanding of the connection between glucose levels in tears and in the blood.²

A Seattle researcher took a slightly different approach to diabetes detection, creating a contact

lens prototype that utilizes LED lights.³ Using sets of electrodes, it runs tiny currents through the tear fluid and measures them to detect very small quantities of dissolved sugar. Preliminary tests suggest that the sensors can accurately detect even very low glucose levels.³ The design would also call for a portable device to be worn by the patient that would wirelessly receive information from the contact lens, allowing the patient to adjust their medication and diet as necessary.³

- **Intraocular pressure.** In September 2011, Sensimed released the very first commercial smart contact lens that records changes in corneal curvature secondary to intraocular pressure fluctuation.³ The Triggerfish lens (*figure 1*) transmits wireless measurements at regular intervals to a portable recording device worn by the patient.³ The disposable lenses are designed to be worn just once for 24 hours. Patients wear them once or twice a year for one day so that providers can measure diurnal pressure (*figure 2*).³



1, 2. Sensimed's Triggerfish lens features highly sensitive platinum strain gauges that record changes in corneal curvature that correspond directly with intraocular pressure.

This information allows doctors to schedule medication more appropriately for better IOP control. In November 2011, the company completed its first U.S. clinical study, and recruitment for a second currently is underway.^{4,5}

Drug Delivery

In addition to looking for a more effective means of monitoring, the eye care industry has long been seeking techniques to optimize drug delivery for the treatment of chronic eye diseases, such as cataracts, glaucoma and age-related macular degeneration. Topical eye drops are one of the most frequently used treatments, but they can be cumbersome and inefficient. Some users have difficulty



instilling drops because of the way the bottles are designed. In these cases, the medicine often flows away from the eye, draining into the nasal cavity and then entering the bloodstream, which can lead to drug waste and unwanted side

effects.⁶

For years, researchers have been particularly interested in using contact lenses as a delivery vehicle for various classes of ophthalmic drugs. With advances in lens design and the availability of new

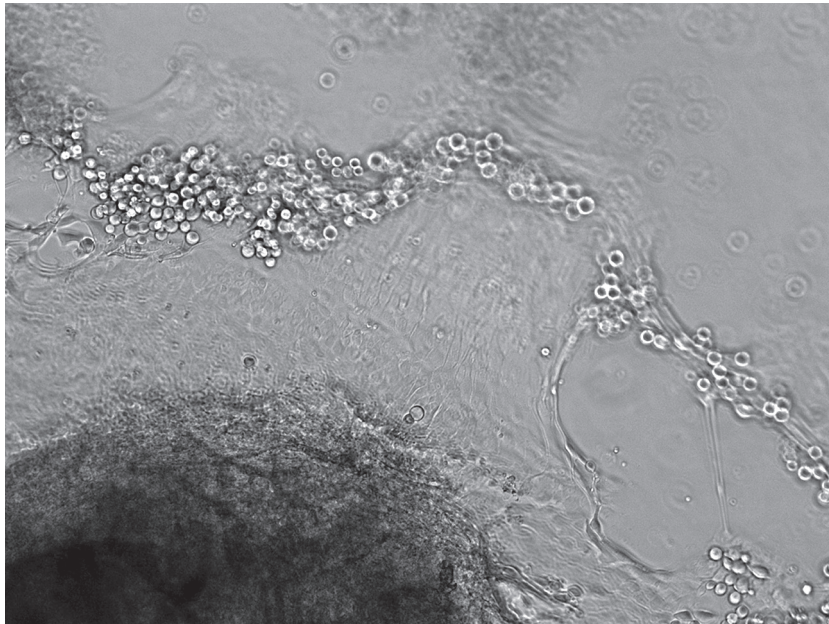


Photo: Nick Di Girolamo Ph.D.

Stem Cell-Coated Lenses

Taking a step into uncharted territory, scientists in Australia have been looking into the possibility of using a contact lens to restore vision in patients with blindness caused by corneal damage. Researchers at the University of New South Wales infused a contact lens with a patient's own stem cells (*figure 3*)—an idea that came about from the observation that stem cells from the cornea stick to contact lenses.⁹ They took three subjects who were blind in one eye, then obtained stem cells from their healthy eyes and cultured them in extended wear contact lenses for 10 days. Next, they cleaned the surfaces of the patients' corneas and inserted the contact lenses.

Within 10 to 14 days, the stem cells began to recolonize and repair the cornea.⁹ Two of the three patients went from being legally blind to being able to read some of the eye chart. The third patient actually regained enough sight to pass a driving exam. Researchers are still monitoring the stability of the treatment, but the early results seem promising. The simplicity and low cost of the technique also means that it could be utilized in poorer countries. If the stem cell corneal method shows additional potential, the procedure could open doors in the future so that patients with compromised ocular structures may not have to wait for a donor.

Photochromatic CLs

Patients even may be able to avoid eye damage before it happens by using contact lenses with UV protection. Long-term exposure to UV radiation can lead to cataracts, skin cancers around the eyelids, and other eye disorders.

3. Stem cells, such as these, have been cultured on a common therapeutic contact lens in order to repair corneal damage.

polymers, it's becoming a more realistic option.⁶ One new technique involves mixing the medication with a pre-polymer liquid, and then polymerizing the combination to create a transparent contact lens coating.⁷ This new approach has shown great promise in contact lens drug delivery devices, but its effect depends on the drug's solubility.

If the drug is water-soluble, it will be trapped within a network of tiny, interconnected, water-filled channels in the material. If the drug is water-insoluble, it will be trapped within nano-spaces in the polymer matrix, and slowly filter out into the channels.⁷ Upon contact with fluid on the eyeball, these channels will open, releasing the drug. By varying the water content, the channel size can be adjusted, and the rate at which the drug is dispensed onto the eye can be controlled.⁸

One major obstacle eye care professionals have faced with such

lenses in the past is how to ensure that sufficient oxygen gets through to the eye. Otherwise, neovascular blood vessel growth can occur. With its interconnected channels, the nanostructure of the new lenses allows gases, salts and nutrients to travel easily across the contact lens barrier.⁷ After testing the nano-engineered lenses to release a water-soluble glaucoma medication and a water-insoluble antibiotic onto the cornea, a research team was able to sustain controlled drug release over a few hours and even a few days.⁷

Ideally, these drug-eluting contact lenses eventually would be available in daily, two-week and monthly lenses so that patients could receive effective, controlled doses of medication while maintaining vision correction. Using a model of drug delivery like this could increase treatment compliance and stabilization of many ocular diseases that our patients encounter.



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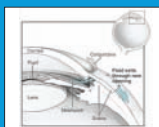
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Contact Lenses

Adapting the flexibility of UV protection into contact lenses by adding a photochromic effect could benefit many patients who find sunglasses to be a nuisance. (Many contacts already have UV protection in them.)

Conventional light-responsive sunglasses (such as Transitions) are coated with millions of molecules of photochromic dyes, which are transparent when out of the sun.¹⁰

These molecules change shape when exposed to sunlight, which allows them to absorb the UV radiation, triggering a response that darkens the lens. When UV light disappears, the molecules return to their original shape and transparent appearance.

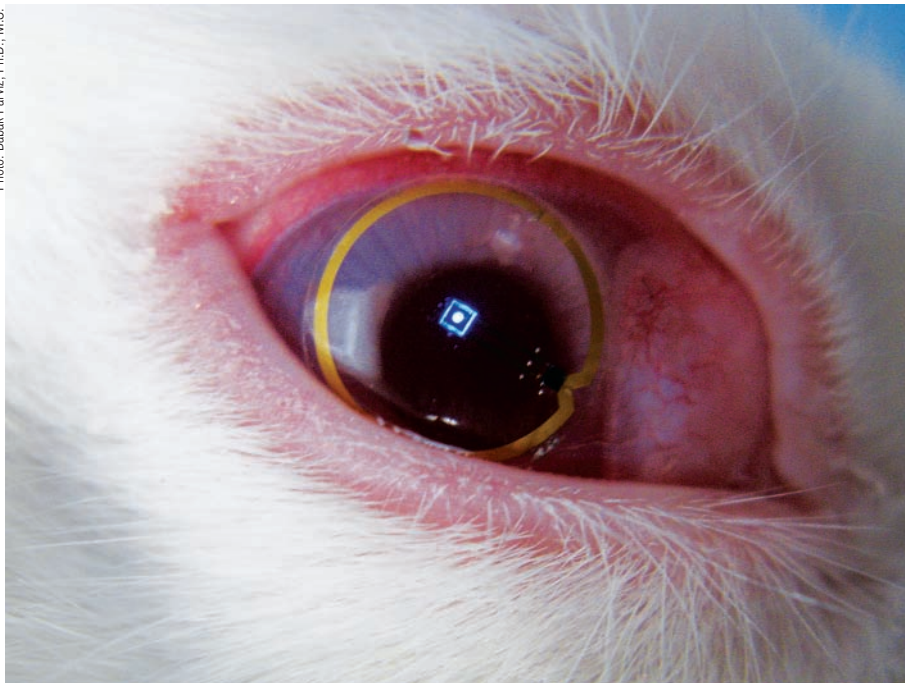
Attempts have been made to design similar light-responsive contact lenses; however, the struggle of applying a dye coating uniformly along the surface of the lens has proven difficult. Researchers at the Institute for Bioengineering and Nanotechnology in Singapore have managed to overcome this hurdle and successfully develop photochromic contact lenses that darken when exposed to UV light and return to normal in its absence.¹⁰

They contain an intricate network of nano-sized tunnels that can be filled with dyes and transition in 10 to 20 seconds—faster than light-sensitive sunglasses on the market today, according to preliminary studies the researchers have conducted.¹⁰ The team is currently working to make the lenses commercially available.

Electronic Viewing

One of the most exciting uses

Photo: Babak Parviz, Ph.D., M.S.



4. Placed on the eye of a live rabbit, this contact lens display is powered by a dipole antenna, showing bright emission from the on-lens pixel.

of contact lenses is the addition of augmented reality on top of our regular vision, which combines real and virtual surroundings to create an “integrated world.” Containing hundreds of LEDs, these lenses would form images in front of the eye that integrate words, photographs and diagrams so that wearers can navigate their surroundings and view displayable information through their contact lenses—much like Arnold Schwarzenegger’s character in “The Terminator” movies and, more recently, as seen in “Mission: Impossible Ghost Protocol.”

While it may seem like science fiction, such bionic technology is much closer to becoming a reality than you might think. Researchers at the University of Washington have created semi-transparent contact lenses with built-in electronics, control and communication cir-

cuits, and miniature antennas.¹¹

They tested a single-pixel, wireless contact lens display on live, anesthetized rabbits and found no adverse effects (*figure 4*).¹¹ Lead researcher Babak Parviz, Ph.D., M.S., suggests that even a lens with a single pixel could aid individuals with hearing impairments.¹² Furthermore, with the addition of color and increased resolution, he believes it could translate into speech captions, visual cues and displayed texts.¹²

Challenges

Advances in contact lens technology take considerable time. For a new refractive error lens to reach the market, it must go through a stringent research and development process in addition to rigorous studies and trials. This process will be even more significant for lenses geared toward applications

beyond vision correction.

Variables, such as lens temperature, oxygen/water content and duration of wear time, should be considered for the safety of the patient. It will take additional efforts, including further scientific research, testing and development, to ensure that they meet the same health and safety standards as current lenses.

As eye care providers, such advances could bolster our practices and improve our reach by increasing the market for contact lens wearers. As these changes come about, it's crucial that we maintain our stance that any contact lens is a medical device that needs to be fitted by a trained eye care professional to ensure the highest level of safety for patients and their eye

health.

If we look at our current patient base, we can readily see how contact lenses change lives daily—from the patient who has keratoconus to the child who plays sports. They are integral to improving vision and, in many cases, quality of life. ■

Dr. Kading owns Specialty Eyecare Group, a Seattle-based practice with multiple locations. His emphasis is on specialty contact lenses and new technologies. Dr. Shen is an associate at Specialty Eyecare Group where she specializes in pediatrics, binocular vision and ocular pathology.

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*Nichols KK, Foulks GN, Bron AJ, Glasgow BJ, Dogru M, Tsubota K, Lemp MA, Sullivan DA. The International Workshop on Meibomian Gland Dysfunction, Investigative Ophthalmology & Visual Science, Special Issue 2011, Vol. 52, No. 4



Injection: The Third Method of Drug Administration

Even if you don't perform injections in your office, you should know the different types of injections in eye care, and how they're performed. **By James L. Fanelli, O.D.**

With dramatic changes in the delivery of health care and the advent of newer diagnostic and treatment modalities, optometrists are in a unique position to deliver care to an organ system that truly has system-wide implications. Not only can the treatment we render to an eye have systemic ramifications, so too can systemic problems manifest in the eye.

A large number of ophthalmic conditions can be treated with topical and oral pharmaceutical agents. But we need to keep in mind that when we discuss "topical" and "oral," we are not so much discussing medications directly but rather we are describing routes by which medication can be delivered. In fact, several medications, such as steroids for example, can be delivered in a variety of ways. We are familiar with topical and oral steroid uses, but many of those same steroids can be delivered by way of injection,

inhalation and transdermal administration.

This article focuses on our third most common route of administration of medications used to treat conditions of the eye and adnexa: injections.

Reasons for Injections

Just as in the use of oral and topical medications, injectable medications can be divided into those that are used for diagnostic purposes and those that are used for therapeutic purposes. And, of those injectable medications used for therapeutic purposes, they can be further divided into primary treatment therapies and adjunctive treatment therapies. (See "Uses of Eye Care Injections," page 36.)

When using oral and topical medications, the onus of proper medication administration usually falls on the shoulders of the patients, and we as eye care providers often give too little thought to the actual delivery of

the treatment. However, when utilizing injectable medications for managing eye conditions, the onus of proper administration of the injectable medications falls squarely on the shoulders of the provider. As such, any eye care provider who utilizes injectable medications must be trained in the proper administrative techniques involved.

In addition, the provider obviously must be familiar with the medications themselves—including dosages, indications, contraindications, side effects and expected outcomes—as well as manage complications that result from their administration.

So, it is absolutely imperative that eye care providers who utilize injections are trained in the actual techniques of injectable administration because there are many types of injections germane to eye care. Furthermore, state licensing laws must be adhered to when prescribing any medications.

(See “Authority of Optometrists to Administer Drugs Via Injection,” page 34.)

Types of Injections

The types of injections used for conditions of the eye and adnexa include intramuscular, intradermal, subcutaneous, subconjunctival and sub-Tenon’s, intravitreal, intravenous and intracameral injections.

Let’s look at each in turn.

Intramuscular Injections

When we think of intramuscular (IM) injections, we tend to think of significant volumes of medication delivered through large, long needles into the muscles of the extremities. But this type of IM injection has relatively limited use in eye care.

Often, IM injections in the setting of eye care are used to treat systemic infections that have

ocular manifestations, such as gonococcal infections, in which the typical treatment consists of an IM injection of Rocephin (ceftriaxone, Roche).¹⁻³ Locations for placement of these IM injections are the deltoids and quadriceps femoris muscles, and the hip.³

Sometimes, IM injections are used to treat contact dermatitis reactions of the eye and adnexa, as in cases of poison ivy, for example.

Nowadays, one of the more common types of facial injections is also technically an intramuscular injection. These injections are most often utilized by dermatologists and plastic surgeons for cosmetic purposes.⁴⁻⁶ Botox Cosmetic (onabotulinumtoxin A, Allergan) for the reduction of glabellar lines and facial wrinkles falls into this category. Botulinum toxin interrupts the normal neuromuscular junction transmission, resulting



Botox is used not only for cosmetic purposes but also for therapeutic purposes, such as hemifacial spasm (pictured here), blepharospasm and migraine headaches.

in paralysis of the striated muscle supplied by that neuromuscular junction (NMJ).

Ideally, Botox is administered directly into the desired muscle, making it an IM injection. However, Botox is often administered as a subcutaneous facial injection in the tissue overlying the target



Photo: CDC/Gabriele Berenson

A general example of intradermal injection: Placement of this injection involves inserting the needle bevel slowly at a 5° to 15° angle. The needle bevel is advanced through the epidermis, just under the skin surface, so that the entire bevel is covered.

Authority of Optometrists to Administer Drugs Via Injection

STATE:	USE OF INJECTABLE DRUGS FOR DIAGNOSTIC AND TREATMENT PURPOSES ¹ [including the treatment of anaphylaxis]	USE OF INJECTABLE DRUGS CURRENTLY LIMITED TO TREATMENT OF ANAPHYLAXIS ONLY
ALASKA	YES	
ALABAMA		YES
ARIZONA		YES
ARKANSAS		YES
CALIFORNIA		YES
COLORADO		YES
CONNECTICUT		YES
D.C.		YES
HAWAII		YES
IDAHO	YES	
ILLINOIS		YES
IOWA		YES
KENTUCKY	YES	
LOUISIANA		YES
MAINE		YES
MARYLAND		YES
MINNESOTA		YES
MISSISSIPPI		YES
MONTANA	YES	
NEW HAMPSHIRE		YES
NEW JERSEY		YES
NEW MEXICO	YES	
NORTH CAROLINA	YES	
NORTH DAKOTA	YES	
OHIO		YES
OKLAHOMA	YES	
OREGON	YES	
TENNESSEE	YES	
TEXAS		YES
UTAH	YES	
VERMONT		YES
VIRGINIA		YES
WASHINGTON		YES
WEST VIRGINIA	YES	
WISCONSIN	YES	

¹ The authority to administer injectable drugs for diagnostic and treatment purposes may be limited by the state optometry act or regulations.

Source: American Optometric Association State Government Relations Center

muscle, and diffusion of the neurotoxin into the underlying muscle produces the desired effect.

While Botox is often considered a cosmetic treatment, there are indications where Botox is used for therapeutic purposes, such as blepharospasm, hemifacial spasms, extraocular muscle disturbances and, most recently, migraine headaches.^{5,7} While the actual mechanism of headache reduction is unknown, botulinum toxin is believed to block nociceptive neuropeptides, which are released in situations of chronic type pain.

Intradermal Injections

This type of injection has limited use in eye care. Because intradermal tissue is poorly vascularized, it is a good site for observing localized immune-type reactions, such



Subcutaneous injections are used to anesthetize the eyelids for repair of lacerations, biopsy of lesions, removal of foreign bodies of the lid, electroepilation, chalazia removal and thermal punctal cauterization.



as those that occur when purified protein derivative (PPD) testing is used to determine an individual's exposure to tuberculin.

Subcutaneous Injections

The subcutaneous tissue lies beneath the dermis and above the muscle tissue. It consists of connective tissue and fat, and is mini-

mally vascularized. It is not tightly adherent to the over- and underlying tissues and, as such, can hold larger volumes of medications.

Injectable anesthetics are administered subcutaneously in the periocular tissues. These may be delivered as a local type of injection (targeting a specific area, such as the temporal aspect of a lid),

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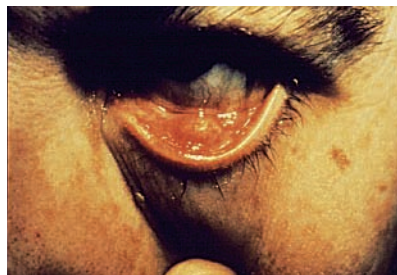
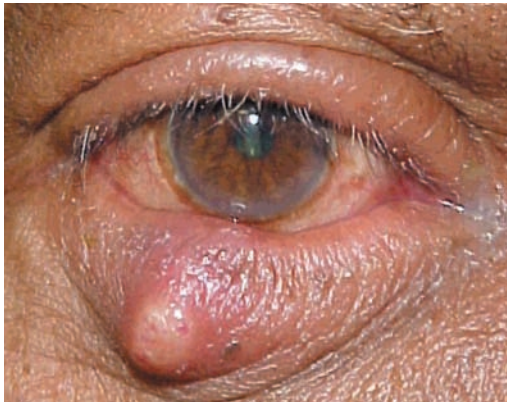
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When considering an injectable steroid for an eyelid “stye,” make sure that the condition is in fact a chalazion and not something else.

A chalazion may “point” externally more than internally (or vice versa). Regardless, the steroid is injected into the subcutaneous space.

or as a more regional, nerve block injection, targeting a larger area. These are used primarily for anesthesia of the upper and/or lower eyelids during repair of lacerations, biopsy of lesions, removal of foreign bodies of the lid, electroepilation, chalazia removal and thermal punctal cautery.

The subcutaneous tissue of the

eyelid, because of its physical characteristics (ability to hold larger volumes of medications) and its close proximity to the tarsal plate, make it an ideal tissue reservoir for steroids to treat chalazia. Chalazia are inflammatory processes of the meibomian glands. They may begin spontaneously due to localized irritation, but most often develop following an infectious process.

Steroids are useful in the management of chalazia, but they are contraindicated in active infectious eyelid processes. Therefore, when considering an injectable steroid for the treatment of an eyelid “stye,” make sure that the condition is in fact a chalazion and not something else.

From an anatomical perspective, chalazia develop within the meibomian gland. Meibomian glands are located within the tarsal plate. The tarsal plate consists of loose connective tissue, and is cartilaginous in nature. As such, it is

rather dense, and inserting a fine needle into tissue of this density is difficult. However, as chalazia develop, they invariably enlarge in the x-, y- and z-axes. Some chalazia may “point” internally more than externally, and some vice versa.⁸ With either presentation, the most appropriate location for the administration of the injectable steroid is still the subcutaneous space.

On the conjunctival side of the lid, the tarsal conjunctiva is tightly adherent to the underlying tarsal plate, and as such, there is very little subconjunctival space. Attempting to administer an injectable steroid into this space is difficult at best.

While chalazia vary in size, duration and firmness, steroid injections generally work very well if two conditions are met: the appropriate amount and concentration of steroid is used, and the chalazion is of recent origin (generally less than four to six months) and so is still relatively soft. As a chalazion becomes more compact over time and more granulation tissue develops inside the affected meibomian gland, it becomes more dense. These respond more slowly to steroid injections, and may require a second injection. Older chalazia—especially if they have been present for longer than six months—may not respond to steroids and must be incised and drained or excised.

The most effective steroid for the treatment of chalazia is Kena-log-40 (Bristol-Myers Squibb), which is triamcinolone acetonide in a 40mg/cc concentration. Kena-log-40 is a thick, viscous, white suspension, and is not quickly

Uses of Eye Care Injections

Diagnostic Injections

- IV fluorescein angiography
- IV indocyanine green angiography
- IV Tensilon testing for myasthenia gravis
- Intradermal TB testing

Primary Therapeutic Injections

- IM antibiotics for gonorrhea
- Translesional steroids for chalazia
- Subcutaneous anesthetics for the periorcular region
- IM/subcutaneous botulinum toxin
- Intravitreal injections for AMD
- Intravitreal injections for macular edema
- SC, IM or IV epinephrine for anaphylaxis

Secondary and Adjunctive Injections

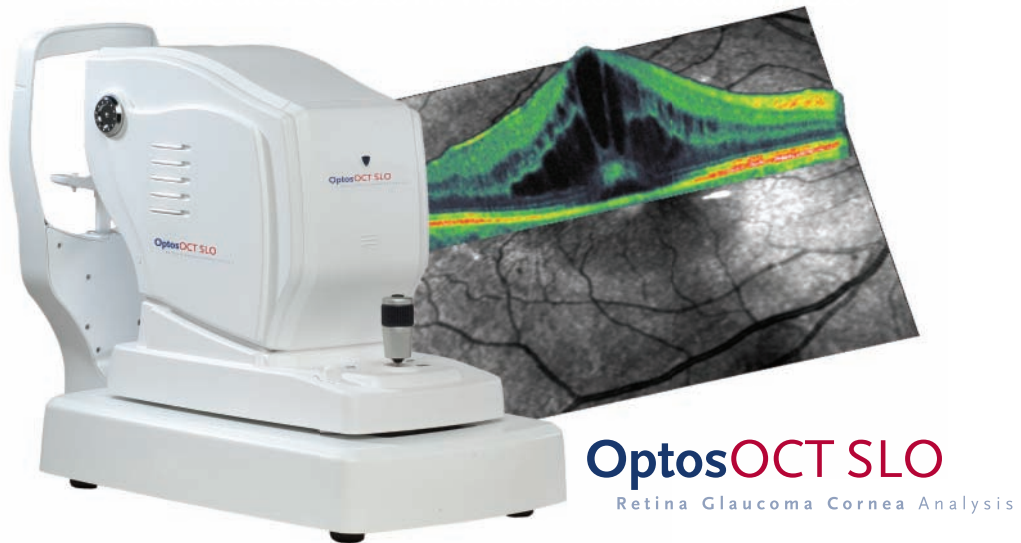
- Subconjunctival steroids for uveitis
- Sub-Tenon’s injections of steroids for uveitis, pars planitis, posterior pole inflammations
- Intravitreal injections for AMD and macular edema

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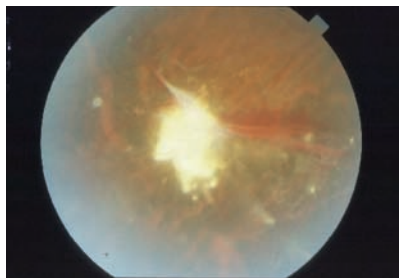
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absorbed into the systemic circulation. It is ideal for the treatment of chalazia for this and several other reasons. While the eyelid subcutaneous space can hold a (relatively) larger volume of injectable medications, what is administered in this area should be not excessively voluminous. Depending on the size and duration of the chalazion, anywhere between 0.2cc to 1.0cc of Kenalog-40 is used.

Because it is a viscous suspension that is slowly absorbed, the bolus of medication tends to sit in the subcutaneous space for an extended period of time, and gradually works to reduce the chronic inflammatory response that is the basis of the chalazion's recalcitrance. As such, it is quite normal to be able to visualize the triamcinolone remaining under the dermis and epidermis of the lid for several weeks. This effect can remain visible for several weeks and happens primarily because of the density of the steroid itself, as well as the translucence of the thin eyelid skin. Sometimes, this residual steroid is misdiagnosed as a side effect of injectable triamcinolone: depigmentation of the skin. In any case, the patient should be forewarned and educated about this possibility.

Some chalazia, especially those in the initial stages, are very soft, and the affected meibomian gland is not fully distended. This situation lends itself to a direct intralesional injection of the steroid. However, even in



Uveitis, such as this inflammatory chorioretinitis, may require steroid injection administered to the sub-Tenon space or intravitreally.

these situations, there still is not much physical space inside the meibomian gland to deposit much steroid. So, it is not critical that the injection be made intralesionally; paralesional injections work just as well.

From a safety perspective, when eyelid steroid injections are made, it is important to keep the needle tip moving, and not remain stationary in one space. So, it is possible that one injection of the steroid, made in a translesional approach, can encompass both an intralesional and a paralesional injection.

Subconjunctival and



A prime example of intravenous (IV) administration in eye care: fluorescein angiography.

Sub-Tenon's injections

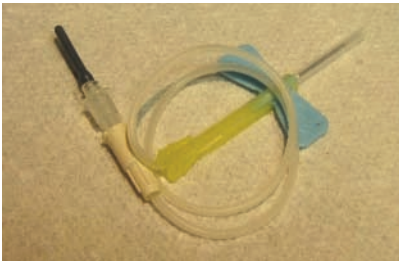
The indications for these types of injections often overlap; while the techniques for administering these injections are similar in approach, they are fundamentally different.

As the names indicate, the injected medication ultimately is delivered to either the subconjunctival space or the sub-Tenon's space. Access to the subconjunctival space is easier than the sub-Tenon's space. Also, given that the subconjunctival space is more anterior than the sub-Tenon's space, subconjunctival injections have a more pronounced effect in the anterior segment, while sub-Tenon's injections have more of an effect on the posterior segment.^{8,9} In some situations, administering a subconjunctival injection of lidocaine prepares the tissue for a subsequent sub-Tenon's injection.⁹

Both types of injections are used primarily for the treatment of inflammation. Chronic, recalcitrant anterior uveitis can often be supplementally managed with a subconjunctival injection of steroid.^{8,9} It is important to note that when considering a subconjunctival injection of a steroid to facilitate control of recalcitrant uveitis, the patient must first demonstrate poor or minimal response to aggressive topical therapy. In other words, subconjunctival steroid injections should not be used unless aggressive topical therapy with cycloplegics and topical steroids fail to control the anterior segment inflammation. Also, subconjunctival



Two common tools for injection: a 3cc syringe with a 1.5" 23-gauge needle (left) and a 1cc syringe with 3/8" 30-gauge needle (right).



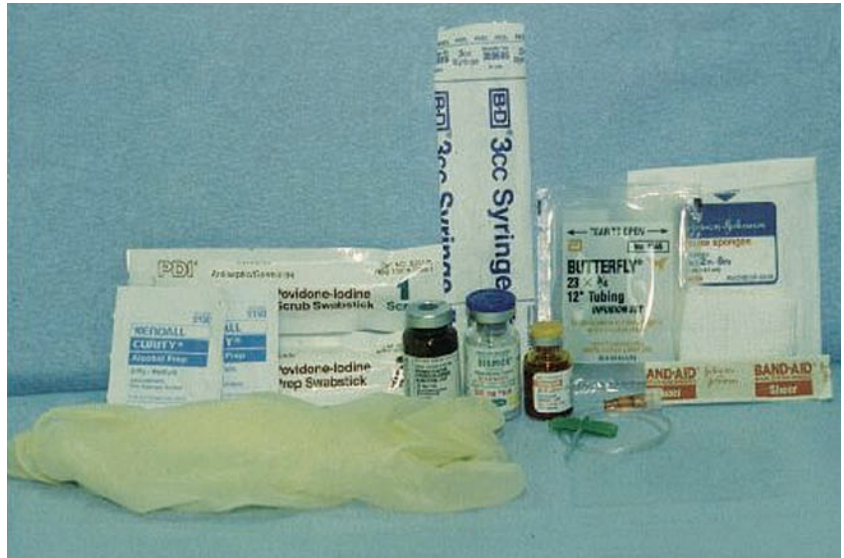
A 23-gauge butterfly setup, as used in the intravenous (IV) administration of fluorescein.

steroid injections are not to be used in lieu of topical therapy, but used as adjunctive therapy along with the topical medications. Sub-Tenon's injections are used to treat inflammations of the posterior segment, although with the advent of safer delivery systems and medications, intravitreal injections are also frequently used.

Intravitreal Injections

While these injections are not used by many optometrists, retinologists use them regularly in modern practice. Intravitreal injections are used to treat posterior segment disorders—mainly those of the macula. Given that the vitreous is avascular, absorption of intravitreally administered drugs is usually slow.

Angiogenic age-related macular degeneration is characterized by



Common Supplies Needed for Injections

- Syringes, usually 1cc to 3cc
- Needles, usually 27ga to 30ga 3/8" to 5/8" for periocular, 23ga 1.5" for extremity IM (insulin or TB syringes work well for periocular injections)
- Alcohol wipes
- Adhesive bandages
- Sterile swabs
- Topical anesthetics
- Topical antibiotic drops and ointments
- Medication ampules or vials
- Forceps
- Lid speculum

Last but not least: Informed consent document

increased fluid accumulation in the subretinal space and/or in the intraretinal spaces. There are a variety of forms of wet AMD, but all are predicated upon the breakdown of the blood retinal barrier and the development of new blood vessels originating in the choroid. Anti-VEGF therapy targets certain growth factors that are responsible for the genesis of neovascular membranes, which are susceptible to leakage and hemorrhage; this is the basis of vision loss in patients with angiogenic AMD.

When administered into the vitreous cavity, anti-VEGF agents are in close proximity to the neovascular membranes, and produce

fewer systemic side effects than if given by other routes of administration. Lucentis (ranibizumab, Genentech) and off-label Avastin (bevacizumab, Genentech) are prime examples of anti-VEGF medications that are administered by intravitreal injection.

Steroids such as Kenalog are also administered intravitreally, although for different reasons. Macular edema—whether from diabetic maculopathy, vein occlusions or other non-angiogenic diseases where fluid accumulates in the central macula and threatens vision—has been found to respond to intravitreal Kenalog (IVK) administration. Though

other treatments are often used in conjunction with IVK therapy (focal laser, for example), IVK remains a viable treatment option for many cases of macular degeneration.

Intravenous Injections

Intravenous (IV) injections are usually used only in diagnostic procedures in the context of ophthalmic care. Fluorescein angiography, indocyanine green angiography and, less frequently, Tensilon (edrophonium, Valeant Pharm.) testing are all performed with IV-administered drugs.

Both the advantage and disadvantage to IV injections lies in the almost instant absorption of the drug into the vascular system. Desired effects, such as being able to evaluate fluorescein flow through the eye, happen quickly. But, unwanted side effects also occur quickly and more noticeably when drugs are administered via the IV route.

Intracameral Injections

These injections are usually reserved for operation room procedures during intraocular surgery. These would not be used in day-to-day clinical eye care.

The injectable route of administration is simply an alternative way to deliver medicine to the body. Injectable medications can be used as primary therapies and as adjunctive therapies. Being familiar with their indications and uses is imperative in current optometric practice. ■

Dr. Fanelli is in private practice in Wilmington, N.C., writes Review of Optometry's "Glaucoma Grand Rounds" column, and lectures on glaucoma and other clinical topics.

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Pataday™

(olopatadine hydrochloride ophthalmic solution) 0.2%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE

PATADAY™ solution is a mast cell stabilizer indicated for the treatment of ocular itching associated with allergic conjunctivitis.

DOSAGE AND ADMINISTRATION

The recommended dose is one drop in each affected eye once a day.

DOSAGE FORMS AND STRENGTHS

Ophthalmic solution 0.2%: each ml contains 2.22 mg of olopatadine hydrochloride.

CONTRAINDICATIONS

None.

WARNINGS AND PRECAUTIONS

For topical ocular use only: not for injection or oral use.

Contamination of Tip and Solution: As with any eye drop, to prevent contaminating the dropper tip and solution, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle. Keep bottle tightly closed when not in use.

Contact Lens Use: Patients should be advised not to wear a contact lens if their eye is red. PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% should not be used to treat contact lens related irritation. The preservative in PATADAY™ solution, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses and whose eyes are not red, should be instructed to wait at least ten minutes after instilling PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% before they insert their contact lenses.

ADVERSE REACTIONS

Symptoms similar to cold syndrome and pharyngitis were reported at an incidence of approximately 10%. The following ocular adverse experiences were reported in 5% or less of patients: blurred vision, burning or stinging, conjunctivitis, dry eye, foreign body sensation, hyperemia, hypersensitivity, keratitis, lid edema, pain and ocular pruritus. The following non-ocular adverse experiences were reported in 5% or less of patients: asthenia, back pain, flu syndrome, headache, increased cough, infection, nausea, rhinitis, sinusitis and taste perversion. Some of these events were similar to the underlying disease being studied.

USE IN SPECIFIC POPULATIONS

Pregnancy: Teratogenic effects: Pregnancy Category C.

Olopatadine was found not to be teratogenic in rats and rabbits. However, rats treated at 600 mg/kg/day, or 150,000 times the MROHD and rabbits treated at 400 mg/kg/day, or approximately 100,000 times the MROHD, during organogenesis showed a decrease in live fetuses. In addition, rats treated with 600 mg/kg/day of olopatadine during organogenesis showed a decrease in fetal weight. Further, rats treated with 600 mg/kg/day of olopatadine during late gestation through the lactation period showed a decrease in neonatal survival and body weight. There are, however, no adequate and well-controlled studies in pregnant women. Because animal studies are not always predictive of human responses, this drug should be used in pregnant women only if the potential benefit to the mother justifies the potential risk to the embryo or fetus.

Nursing Mothers: Olopatadine has been identified in the milk of nursing rats following oral administration. It is not known whether topical ocular administration could result in sufficient systemic absorption to produce detectable quantities in the human breast milk. Nevertheless, caution should be exercised when PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in pediatric patients below the age of 2 years have not been established.

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

NONCLINICAL TOXICOLOGY

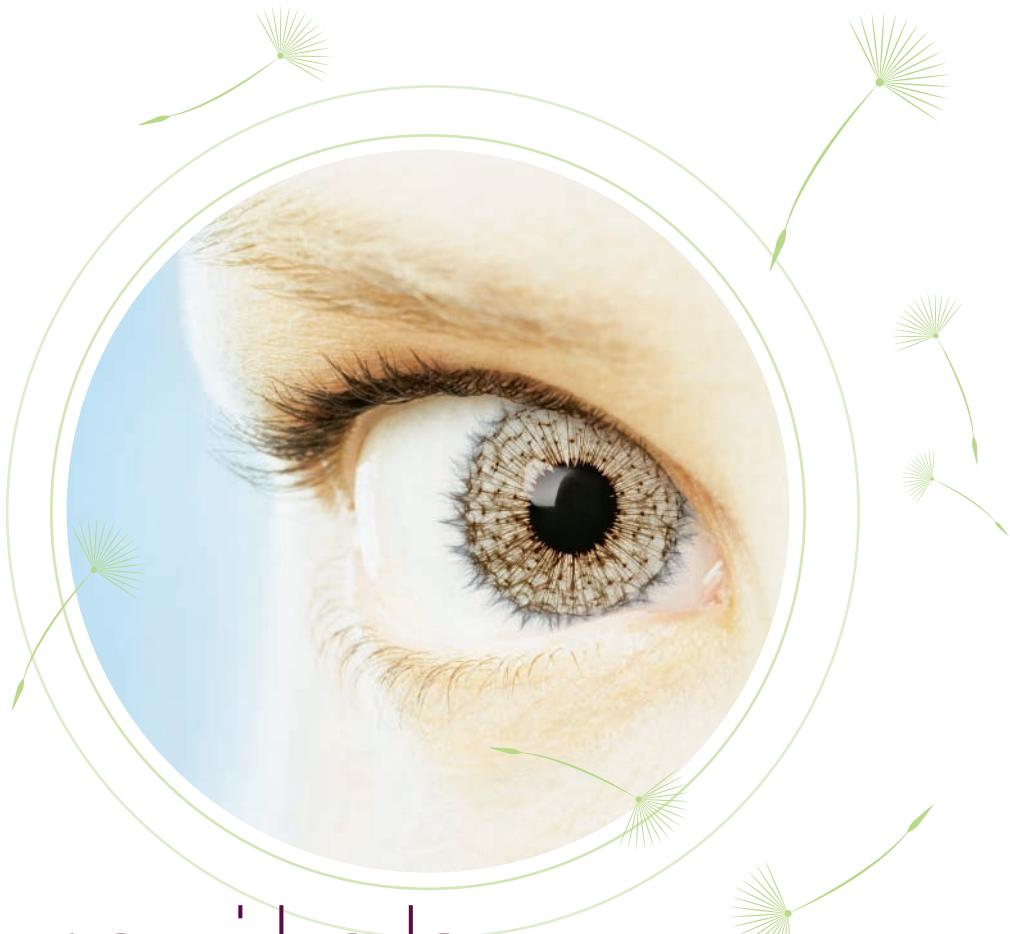
Olopatadine administered orally was not carcinogenic in mice and rats in doses up to 500 mg/kg/day and 200 mg/kg/day, respectively. Based on a 40 µL drop size and a 50 kg person, these doses were approximately 150,000 and 50,000 times higher than the maximum recommended ocular human dose (MROHD). No mutagenic potential was observed when olopatadine was tested in an *in vitro* bacterial reverse mutation (Ames) test, an *in vitro* mammalian chromosome aberration assay or an *in vivo* mouse micronucleus test. Olopatadine administered to male and female rats at oral doses of approximately 100,000 times MROHD level resulted in a slight decrease in the fertility index and reduced implantation rate; no effects on reproductive function were observed at doses of approximately 15,000 times the MROHD level.

U.S. Patents Nos. 5,641,805; 6,995,186; 7,402,609

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ophthalmic solution) 0.2%

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- **Start:** As soon as 3 minutes following allergen challenge, 60% of patients achieved Zero-itch*†
- **Finish:** At 16 hours, 60% of patients had Zero-itch*†

INDICATION AND DOSING

PATADAYTM Solution is a mast cell stabilizer indicated for the treatment of ocular itching associated with allergic conjunctivitis. The recommended dose is one drop in each affected eye once a day.

IMPORTANT SAFETY INFORMATION

PATADAYTM Solution is for topical ocular use only. It is not for injection or oral use.

To prevent contaminating the dropper tip and solution, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle. Keep the bottle tightly closed when not in use.

PATADAYTM Solution should not be used to treat contact lens-related irritation. The preservative in PATADAYTM Solution, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses should be instructed to wait at least ten minutes after instilling PATADAYTM Solution before they insert their contact lenses.

Symptoms similar to cold syndrome and pharyngitis were reported at an incidence of approximately 10%.

For additional information about PATADAYTM Solution, please refer to the brief summary of prescribing information on the following page.

*Post-hoc analysis of combined data from two studies using a contralateral conjunctival allergen challenge (CAC). Based on a scale of itching scores of 0-4, with 0 as no itching and 4 as severe itching. Ocular itching was evaluated 3 minutes after allergen challenge at onset and at 16 hours.

†(N=85; 95% CI=48.8, 70.5)

‡(N=82; 95% CI=48.3, 70.4)

References: 1. IMS Health, IMS National Prescription AuditTM, August 2010 to February 2011, USC 61500 OPHTH ANTI-ALLERGY. 2. Data on file.



Alcon[®]

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The Increasing Value of OCT Imaging

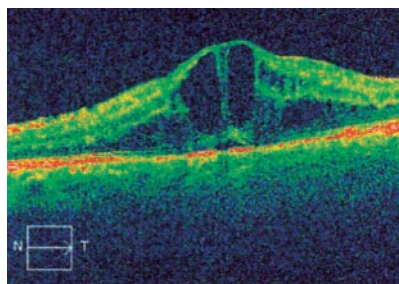
Ocular coherence tomography has become an all-around useful tool for glaucoma, retina and even anterior seg evaluation. Not every office needs one, but we certainly couldn't do without ours. **By Kenny Bumgarner, O.D.**

Today's optometrists are increasing their utilization of non-invasive technologies to aid in the observation of retinal tissues.

Ocular coherence tomography (OCT) facilitates evaluation, monitoring and follow-up for glaucoma and macular pathologies. In addition, OCT can be beneficial in numerous retinal manifestations to establish tissue thickening or thinning when compared to the normal population database.

While fluorescein angiography has been the "gold standard" of evaluation and management of macular and retinal pathologies, OCT has augmented or even replaced angiography in particular cases. Cystoid macular edema, central serous retinopathy and diabetic maculopathy are only a few of the macular pathologies where OCT proves to be extremely beneficial.¹

Since the introduction of the OCT, optometrists have rewritten our optometric algorithms in the diagnosis, management and follow-up of patients. This technology has



OCT may have already supplanted the 'gold standard' fluorescein angiography for detecting some retinal etiologies, such as cystoid macular edema.

allowed for increased efficacy of referrals and continued in-office management of patients. In this regard, the wider use of OCT may have a beneficial impact for the patient, as well as the doctor.

This article examines the OCT's revolutionary technology, addressing benefits from a clinical view, in addition to an investment standpoint. It also discusses professional opinions, clinical diagnoses, management and follow-up modalities that suggest the OCT is not only important in primary care optometry, but becoming a valuable

adjunctive asset for optimal patient care.

Clinical Uses for OCT

OCT affords us a highly quantifiable, accurate and repeatable technology that is best utilized in conjunction with other available subjective and objective technologies. We can utilize OCT for many types of posterior polar evaluations, such as optic nerve abnormalities, glaucoma progression, macular and retinal pathologies.

Resolution of this technology in the last several years has gone from approximately 10 μ m microns to 4 μ m. This instrument is essentially able to measure within the accuracy of two-thirds the width of one red blood cell. (Practically speaking, we are able to examine living tissue that is 0.25mm thick, containing 10 layers of highly specialized, nearly transparent cells. It shows us a living histology slide of the sensory retina and retinal pigment epithelium.)

Scanned information is then compared to "normal population"

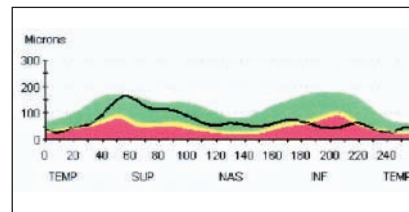
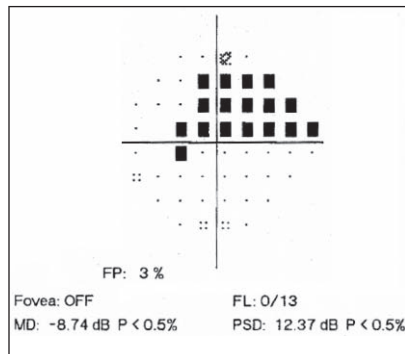
data in a way that is repeatable and highly accurate, with a high degree of sensitivity and specificity. Scanning devices allow us to pinpoint the exact layer damaged in glaucoma, optic nerve and macular processes.

Let's look at each of these applications individually:

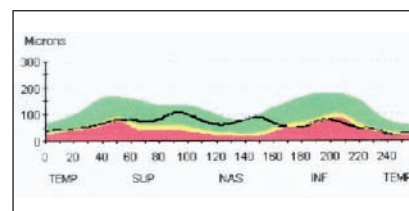
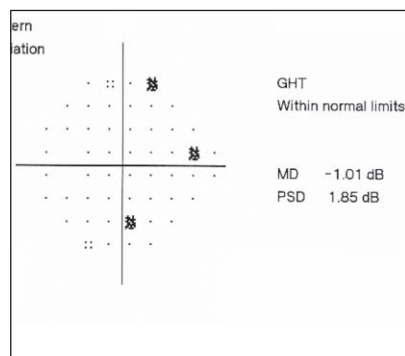
- **Glaucoma.** There is no question that nerve fiber loss and structural damage appear much earlier in the disease process than functional subjective visual field defects.² OCT technology facilitates our ability to more accurately confirm or dismiss a diagnosis of glaucoma at an earlier stage of development. This fact promotes earlier intervention long before 40% of the nerve fiber loss manifests (which is necessary for subjective visual field depressions to be recorded).² When combining scanning technologies with other clinical data, interventional therapy may be initiated much earlier when including OCT findings with visual fields.

However, OCT is not a stand-alone test for glaucoma evaluation and management. It should be viewed as a critical piece of information in the decision-making process. A good example is when the retinal nerve fiber layer (RNFL) shows superior and/or inferior thinning that is suspicious for glaucoma while the visual fields remain normal. OCT allows an added point of reference in following a patient from normal to glaucoma suspect, and finally to glaucoma patient.

The valuable OCT data should be clinically confirmed by the examination findings, including the "multifactorial glaucoma risk pie." This analysis includes history, age, race, family history, longevity and general health information. Clinical testing to be added to the decision-making process includes important



This patient's visual field (left) correlates with his OCT TSNIT graph, which corroborates the findings of nerve fiber thinning.



However, this patient's visual field (left) does not agree with her OCT TSNIT graph, which suggests early nerve fiber thinning not yet apparent with perimetry.

information, such as pachymetry, serial intraocular pressures, visual fields, stereophotography and finally clinical experience. In short, you would rarely start therapy based only on OCT thinning, but consideration of RNFL is a valuable contribution to the decision-making process.

Looking forward, the most powerful use of this technology in glaucoma will take place when we can "marry" OCT structural analysis with functional testing of visual field analysis. (Several companies are now working on producing this combined testing technology.) Although this information is not diagnostic, it does provide relevant structural analysis to verify stability vs. progression.

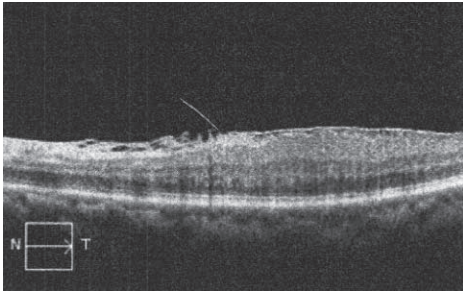
- **Optic nerve abnormalities.** In addition to glaucoma, OCT can be used in contributing information in the areas of optic nerve drusen, optic pits, optic atrophy disorders and optic nerve edema.³ Another

example would be in progressive thinning in the optic atrophies by serial analysis.

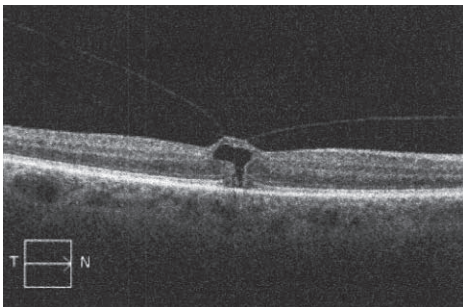
RNFL analysis can be helpful in assessing nerve head thickening in a patient with suspected pseudotumor cerebri (PTC). I have found it especially helpful in follow-up monitoring of disc changes once treatment has begun for PTC.⁴ This management, in conjunction with other ocular, physical and neurological examinations, can be helpful in assessing the patient's treatment progress.

- **Macular abnormalities.** OCT analysis of the macula includes a range of disorders, such as:

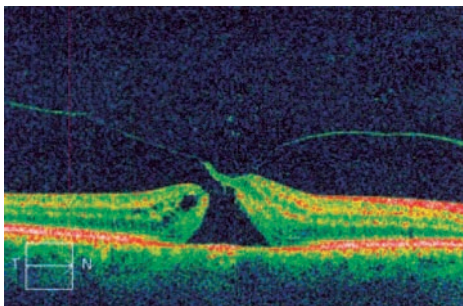
- Macular degeneration/choroidal neovascular membrane.** Scanning technology is important for initial substantiation of macular thickening when combined with clinical findings of deep and superficial retinal hemorrhages, deep and superficial exudate, fluorescein angiography, as well as subjective



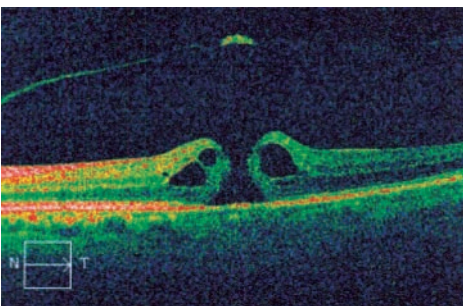
This series of OCT images, from different patients, demonstrates the development of a macular hole, starting here with epiretinal membrane with subsequent macular thickening.



An impending macular hole secondary to vitreomacular traction.



An early macular hole.



A macular hole with overlying posterior vitreous detachment.

symptoms of metamorphopsia. OCT can show structural thickening and distortion of the macular anatomy. In addition, its usefulness has been documented to follow the success or failure of anti-VEGF therapies; it allows us serial review post-injection to structurally document the reduction of macular thickening.⁵ This becomes a valuable tool in guiding therapy and follow-up decisions for re-injection vs. monitoring.

—*Vitreomacular traction syndromes.* Scanning technology is valuable in the verification and diagnosis of these syndromes as they progress from preretinal fibrosis, classic vitreomacular traction syndrome and, finally, differentiation of early macular hole formation to complete macular hole formation. As with other disorders, this information provides structural evidence of deviation from normal macular anatomy in its various forms.

—*Macular edema.* OCT has become a valuable instrument in the evaluation of macular distortion, whether from diabetic, cystoid, central serous or other disorders. It also has become valuable for the retinologist, as well as the co-managing optometrist, in post-treatment patients to monitor for resolution, exacerbation or stability of macular integrity.

—*Retinal arteriovenous occlusive diseases.* OCT is used to monitor central retinal venous and branch occlusions as they approach the macula. In addition, arteriolar occlusive disease with secondary macular and retinal edema

can be seen initially. In long-term follow-up, thinning of the affected macular and adjacent retina will be noted in many post-arterial occlusive cases. OCT is useful in initial evaluation of macular thickening as it relates to other clinical objective and subjective findings. This becomes helpful in deciding when to refer to the retinal specialist (not to mention concurrently orchestrating multidisciplinary management of underlying systemic disease). Finally, as with the macular edemas, OCT can be useful in post-treatment monitoring for improvement, stability or exacerbation of the macular anatomy.

• *Anterior segment evaluation.* OCT scanning devices, although not necessarily diagnostic, can be helpful in evaluating anterior segment disorders, such as narrow angles, anterior chamber or iris abnormalities when combined with additional clinical testing, such as gonioscopy, ultrasound and other clinical apparatus.

In terms of iris thickening disorders, including iris cysts and melanomas, OCT helps us to more critically differentiate between solid vs. cystic defects.

OCT for Communication

OCT is becoming an invaluable tool not only for clinical diagnosis but also for patient education, doctor-to-doctor communication and comanagement.

Consider these uses:

• *Cataract surgical referrals.* In special instances, OCT provides potential contributory information in patients with reduced visual acuity that is not clearly defined by other techniques. It can offer information when there is question as to degree of, for example, cataract vs. retinal effect on visual acuity. This is helpful in macular degeneration,

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Estimated Return on Investment

Example: ZEISS CIRRUS HD OCT models 4000 and 400

ESTIMATED REIMBURSEMENT	Patients per day				
	2	3	4	5	6
CPT 92133 / 92134, \$44 per patient (nat'l avg)	\$44	\$44	\$44	\$44	\$44
MONTHLY reimbursement (20 days)	\$1,760	\$2,640	\$3,520	\$4,400	\$52,800
ANNUAL Reimbursement (240 days)	\$21,120	\$31,680	\$42,240	\$52,800	\$63,360
5 YEAR PROJECTION	\$105,600	\$158,400	\$211,200	\$264,000	\$316,800

	CIRRUS 4000	CIRRUS 400
MONTHLY BREAK EVEN	Patients	Patients
Number of patients needed to make monthly payment	25	21

Lease payment divided by CPT 92133 / 92134 (\$44)

Does not include 92132, which may increase reimbursement to approx \$80 / patient

SALES PRICE	CIRRUS 4000	CIRRUS 400
	\$73,950	\$63,950

ESTIMATED TAX CONSIDERATIONS

IRS Section 179 Accelerated Depreciation

NET PRICE AFTER TAX CONSIDERATIONS	\$34,418	\$29,218
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Source: Carl Zeiss Meditec USA

THE ABOVE INFORMATION IS NOT LEGAL OR TAX ADVICE. Please verify reimbursement with your local medicare carrier and verify your tax implications with your CPA/tax advisor

is not used in an illustrative manner. The ability to actually show the patient normal vs. abnormal tissue is highly educational. This gives the patient a better understanding of his or her disease process and/or reason we are referring for further evaluation. It also provides patients with a clear understanding of why we are monitoring their retinal tissue. This is a valuable asset in instilling patient confidence and allowing the patient to “actually see what is wrong.”

Both my patients and I have found the OCT images intuitive and

easy to understand. And, patients require very little training to see how their “abnormal scan” differs from the normal.

- **Teleophthalmology.** The OCT provides an excellent opportunity for us to communicate with other secondary and tertiary ophthalmic physicians “over the airwaves.” This often results in a more effective referral as well as saving the patient time and money monitoring certain retinal disorders “in-house.” A simple phone call along with an e-mail of the OCT allows us a “telecommunicative consultation” to facilitate clinical decision-making between the primary and secondary care providers.

retinal macular fibrosis or any case of suspected macular abnormality. Although mere thickness readings cannot guarantee acuity assessment, these findings can help the practitioner more accurately predict post-surgical cataract outcomes.

In addition, OCT can be useful for visualizing macular structural abnormalities that are not clearly seen through a dense cataract. This may include subtle macular edema from diabetes, small focal venous occlusive disorders, preretinal fibrosis or more subtle early macular hole formation. Although this cannot be used as a routine test when considering cataract referral, it can be valuable information in commu-

nication with secondary or tertiary care providers in the event they are concerned that macular abnormalities may affect the visual acuity outcomes post-cataract surgery. In the same way, it can be helpful in certain refractive surgical candidates.

In short, OCT should be used in a case of “reasonable suspicion of abnormality” when we are unsure of the total visual acuity due to cataract, or a combination of cataract and macular abnormality. This technology allows the primary care practitioner a better and higher degree of competency and referral.

- **Patient education.** On a personal note, there isn’t a day that goes by in our office that OCT

Management and Follow-up

Management of numerous posterior segment disorders with the aid of serial OCT is becoming more important in daily practice. With our present technology, follow-up of macular thickening from tractional or edematous disorders can be precisely measured to within microns. In addition to macular and retinal evaluation, no one can argue the benefit of OCT in the management and predictive value in glaucoma evaluation, both in-office and for referral to secondary glaucoma specialties.

Comanagement is becoming a big issue in following the post-treatment macular degeneration patient who has had anti-VEGF, intraocular steroid injections, photodynamic therapeutics, or a combination of any of these. This gives the retinal specialist and the practicing optometrist the confidence in each other to comanage these patients. In particular, it gives the ophthalmologist the assurance that subtle edemas and retinal thickening, as well as other retinal fluctuations, can be more precisely monitored in the primary care office.

Because many of our retinal specialists are overworked, they welcome the opportunity to send the patient back to the referring physician's office. The patient benefits because he is back with "the doctor I know and trust." The cost to the health care system is arguably lower in primary vs. secondary and tertiary office settings. In addition, the patient benefits in less travel time and less wait time. Importantly, the average O.D. spends more "one-on-one" time with the patient.

Return on Investment

Even with a reduction in office reimbursements, the familiar argument that OCT is not a revenue producer is unfounded. Our office performs more than 120 OCT scans a month. True, reimbursements for OCT itself have gone down; yet, the added financial enhancement comes not only in OCT coding, but also in the charges for office visits and verification of acuities, which often requires refractions (an additional charge). This makes OCT an excellent revenue producer in the office. (See "Estimated Return on Investment," page 46.)

Since the advent of scanning technologies in the 1980s, OCT has become more affordable and, more importantly, more valuable in patient care. The impact of this technology will make it one of the most important clinical tools available in our decade, and will only get better as technology and integration with other testing modalities improves.

The value of OCT, as with any testing technology,

can only be maximized if we become familiar with the strengths and weaknesses of the equipment. Learning and experience, with a keen eye on the literature, can allow us to maximize utilization of scanning devices to enhance our ability to provide quality eye care to our community. ■

Dr. Bumgarner is in a primary care group practice in Pinehurst, N.C. He has no financial relationships to disclose. He thanks Andrew Apple and Nicole Piatt, fourth year optometry students at Pennsylvania College of Optometry at Salus University, for their assistance with this article.

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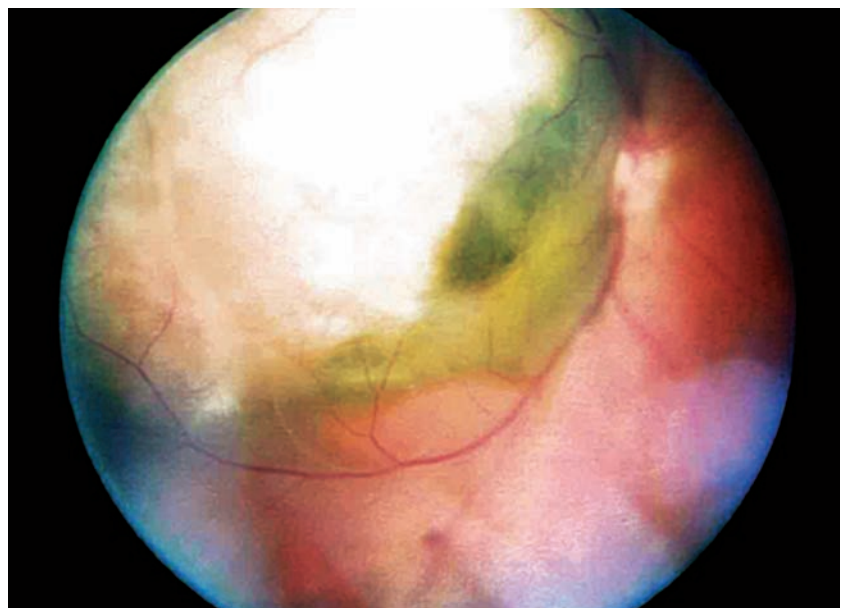
Choroidal Melanoma is a Life Sentence

A thorough knowledge of treatment options and associated risks is crucial to ensure the best possible outcome with this dire condition. **By Sara Weidmayer, O.D.**

A 65-year-old white male inmate presented at a state correctional facility's eye clinic with a complaint of "bugs" in the vision of his left eye. This had been happening for about three weeks; he denied photopsia. The patient's systemic history was significant for hypertension, heart problems (having had two valve replacements) and type 2 diabetes that was controlled with oral medications.

His ocular history was significant for "long-standing" blindness of his right eye.

Upon further questioning about this long-standing blindness, we learned that, 15 years earlier, the patient had seen a retinal specialist, who told him he had a "freckle" in his eye. He reported that he had lost his vision in the right eye about 10 years ago, but hadn't seen a specialist since that visit 15 years ago.



1. A fundus photograph of our patient's right eye.

Diagnostic Data

Upon examination, the patient's visual acuity was light perception only O.D., and 20/25 O.S. Pupils measured 4mm O.D. and 3mm

O.S. and were both round and reactive, with a 3+ afferent defect O.D. Anterior segment evaluation was remarkable for nuclear sclerotic cataracts. We performed

a dilated fundus examination, and found that the patient's chief complaint was due to a posterior vitreous detachment O.S., which caused dense, central vitreous syneresis. We found no retinal breaks or anything else of clinical significance in that eye.

Dilated fundus examination of his right eye revealed a large elevated lesion, approximately 12 disc diameters in size, extending from—and including—the temporal optic disc, past the temporal macula and beyond the superior arcade. It was gray-white in color with what appeared to be some fluid content in the inferior-nasal aspect (*figure 1*).

Diagnosis

We made a provisional diagnosis of choroidal melanoma with serous retinal detachment, and documented it with fundus photos.

Treatment and Follow-Up

We referred the patient to the facility's staff ophthalmologist for further evaluation and treatment. About two weeks later, the ophthalmologist saw him and took a fluorescein angiogram as well as an A- and B-scan ultrasound. The interpretation reports for these procedures were fairly rudimentary; they stated that the A- and B-scans revealed a "solid mass," and the fluorescein showed that the mass was filled with dye early and then slowly disappeared. After these studies, the ophthalmologist posed differentials of malignant melanoma or disciform macular degeneration. He referred the patient to a retinal specialist, who saw him just days after his ophthalmology consultation.

The retinal specialist immediately diagnosed a choroidal melanoma and sent the patient to an

oculoplastics specialist that day to discuss treatment. Diagnostic testing results from the retinal specialist and oculoplastics specialist were not available to us in the patient's record. An abdominal CT scan with contrast and a chest X-ray were also ordered.

The patient and oculoplastics

groups.^{1,3,4} They occur mostly in light-skinned persons with blue or green irides, and are rarely found in blacks or Asians.^{1,4}

Patients with choroidal melanomas are often asymptomatic, but may present with decreased vision, visual field defects, floaters, photopsias or, in rare instances,

Choroidal melanomas often display an abrupt elevation from the choroid, subretinal fluid, orange pigmentation over the lesion's surface and growth over time.

specialist decided on treatment with enucleation, and the patient was scheduled for surgery the following week. The enucleation was successful; however, the patient unfortunately experienced difficulty with the anesthesia and died two days after the enucleation. The abdominal CT and chest X-rays had not yet been completed.

Discussion

Choroidal melanomas are relatively rare, with an incidence of approximately five to six cases per one million people, which equates to about 1,400 cases in the United States each year.^{1,2} They are found mostly in adults (with the peak around age 55), generally are not familial, and show a slight male predilection for most age

pain.^{4,5} If pain does occur, it is usually a result of secondary glaucoma or tumor necrosis; choroidal melanomas also can cause pain by impinging on underlying posterior ciliary nerves, but this seldom occurs.^{3,5}

These lesions usually are elevated and may appear mottled, dark brown, dull-gray, gray-green or yellow (amelanotic).⁴⁻⁶ They may assume a mushroom or dome shape with congested blood vessels within the tumor—this configuration represents the 20% of choroidal melanomas that erupt through Bruch's membrane and the retinal pigment epithelium (RPE).^{1,4,5}

Choroidal melanomas often display an abrupt elevation from the choroid, subretinal fluid, orange pigmentation over the lesion's surface and growth over time.⁴

Malignant Transformation

Risk factors for malignant transformation of choroidal nevi include:⁴

- Thickness > 2mm.
- Subretinal fluid.
- Presence of symptoms.
- Prominent orange pigment overlying the lesion.
- Location < 3mm from the optic disc.

**If two or more factors are present, the lesion is likely a choroidal melanoma.*

Case Report

Subretinal fluid with resultant underlying serous retinal detachment results from RPE breakdown. These serous detachments often shift, and may appear to contain blood if the tumor has traversed Bruch's membrane.⁵

Overlying orange pigmentation is lipofuscin; this pigment is composed of proteins, lipids and small chromophores, and it accumulates in the RPE as a result of cell degeneration and incomplete digestion of the photoreceptor outer segments.^{6,7} Lipofuscin is not specific to melanomas; it may also be associated with choroidal nevi or other benign choroidal tumors. However, lipofuscin is much more commonly seen with melanomas than with benign counterparts.⁵

Other possible signs associated with choroidal melanomas include vitreous hemorrhages or pigmented vitreous cells, drusen on the surface of the tumor, choroidal neovascular membranes, or even proptosis if the tumor invades the orbit.⁴

Differential Diagnoses

There are a plethora of differential diagnoses for melanotic and amelanotic choroidal melanomas, which vary on the prognostic continuum of severity.

- *Choroidal nevi* are a major differential. They are common, benign melanocytic tumors and are found in approximately 2% to 6.5% of the white population.⁸⁻¹⁰ Nevi usually are slate-gray and relatively flat (less than 2mm thickness), although there is significant size overlap between small melanomas and larger nevi.^{8,9} Like choroidal melanomas, they also may show overlying drusen or lipofuscin (figure 2). Statistically, of every 500 choroidal nevi, one will undergo malignant transfor-

mation if followed for 10 years; the estimated annual rate of malignant transformation is one in 8,845.^{6,9}

There are multiple known risk factors for such transformation (see "Malignant Transformation," page 49).^{4,8} The most important appears to be initial nevus thickness of greater than 2mm, but a large base diameter (greater than 7mm) also suggests premalignancy of the nevus.^{8,9} The absence of drusen is a good prognostic indicator.⁷

Whereas choroidal melanomas tend to grow relatively rapidly, choroidal nevi may enlarge slowly over a period of several years, not necessarily indicating malignant transformation. Such non-malignant growth is more common in younger patients and tends to stabilize with age.¹¹ Thus, slow growth of choroidal nevi is not invariably a sign of malignancy, especially in younger patients without other risk factors.¹¹

Interestingly, pigmented choroidal lesions with none of the above risk factors have a 3% chance of growth in five years; such lesions are usually choroidal nevi.⁹ Presence of one of the above factors carries a 38% chance of growth, and more than a 50% chance of growth exists if two or more risk factors are present.⁹ The relative risk of growth climbs from 1.9 times to 27.1 times for the presence of one vs. all five risk factors.⁹

- *Choroidal metastasis* refers to a tumor that has spread to the choroid via hematogenous routes



2. Choroidal nevus with overlying drusen.

from a primary malignancy elsewhere in the body. Thus, they are not primary tumors like choroidal melanoma—most often, they are metastases from breast or lung cancer. These lesions usually appear dome-shaped and creamy-yellow in color, and often induce retinal detachments. Choroidal metastases are often bilateral or multifocal and do not appear mushroom-shaped, unlike amelanotic melanomas.¹

- *Congenital hypertrophy of the RPE* presents as single or multifocal, darkly-pigmented, flat lesions, often with hypopigmented lacunae. They are benign, usually do not change with time, and require no treatment.¹

The resultant serous retinal detachment and retinal elevation secondary to exudative age-related macular degeneration (AMD) poses another differential for choroidal melanoma. AMD may show subretinal hemorrhaging, lipid or

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turbid exudation, dirty-gray or yellow macular edema, choroidal folds, pigment epithelial detachments or disciform scarring.¹ Fluorescein angiography aids in differentiating between these conditions.

- *Melanocytomas* are darkly pigmented and found on or around the optic disc (*figure 3*). Unlike melanomas, they are congenital and often occur in individuals with dark pigments. They usually are inactive, but may grow and rarely develop into melanomas.¹

- *Choroidal hemangiomas* are benign dilations of choroidal blood vessels and are often associated with Sturge-Weber syndrome. They appear elevated and are red-orange in color. Like choroidal melanomas, they may induce serous retinal detachments.¹

- *Choroidal osteomas* are yellow-orange placoid masses. Interestingly, they are composed

of mature bone tissue. They may allow choroidal neovascularization and subretinal bleeding to develop. Very characteristic features make them easy to differentiate from choroidal melanomas using ultrasonography or CT scan.¹

- Additional differentials for amelanotic or melanotic choroidal melanomas include choroidal detachment, lymphoma, metastatic carcinoma, subretinal or sub-RPE hematoma, localized suprachoroidal hematoma, nodular posterior scleritis, reactive hyperplasia of RPE or massive gliosis of the retina.^{4,5}

Additional Testing

Various instruments may assist in the diagnosis of choroidal melanomas.

A- and B-scan ultrasonography not only aids in diagnosis, but also may provide more accurate measurements of the tumor. A-scan usually reveals low internal reflectivity within the tumor; oscillation in height of the echoes within the lesion may correspond with the patient's pulse, which indicates the presence of intralesional vascularity.^{1,5}

B-scan shows a solid mass with an acoustically bright anterior aspect with internal and basal darkness; the

cross-sectional shape typically is bi-convex, but may appear mushroom-like. Choroidal excavation and orbital shadowing may also be seen.^{1,5}

Fluorescein angiography typically shows hyperfluorescence of the tumor's vessels and diffuse late staining.¹ However, the fluorescein pattern depends on the tumor's size, shape, pigmentation, integrity of the RPE and whether there is a corresponding serous retinal detachment, among other variables.⁵ Fluorescein angiography does not yield pathognomonic signs of choroidal melanoma.³

In this particular patient, choroidal melanoma was diagnosed based on fundoscopic examination, A- and B-scan ultrasounds, and fluorescein angiography (without biopsy).

This patient's clinical presentation alone was highly suggestive—basically unequivocally—of choroidal melanoma. He possessed four of the five aforementioned risk factors for malignancy: thickness > 2mm, subretinal fluid, symptoms/visual loss, and a location < 3mm from the optic disc. Though the dimensions of the melanotic lesion based on ultrasonography are not available, it was clearly thickened more than 2mm and had a basal diameter larger than 7mm (the widely accepted upper limits of benign nevi).^{8,12}

Additionally, the mass exhibited blatantly invasive features, such as encroachment onto the optic disc.^{8,12}

Management and Prognosis

When a suspicious ocular mass is found, it is important to ask the patient whether they have had any ocular surgery or trauma; a history of cancer; or any systemic symp-



3. Melanocytoma are darkly pigmented and found on or around the optic disc.

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DESCRIPTION: Tears Again® HYDRATE™ is a Medical Food specially formulated for the Dietary Management of Dry Eye Syndrome, Blepharitis, and Meibomian Gland Dysfunction. Tears Again® HYDRATE™ is intended to be used under the supervision of a physician. Tears Again® HYDRATE™ is formulated to contain the omega-3 fatty acid, Flaxseed Oil, but has also included Evening Primrose Oil (EPO), a substance having exceptionally high content of the even rarer essential fatty acid, gamma linolenic acid (Omega-6) as well as Bilberry Extract (BBE). The clinically supported tear specific ingredients in Tears Again® HYDRATE™ are microencapsulated through a unique proprietary liposome process to enhance absorption. This delivery system, termed Hypersorb®, eliminates digestive problems commonly associated with EFAs and is especially suited for the oral delivery of ingredients with poor bioavailability.

CONTAINS: Flaxseed Oil 1000 mg, Evening Primrose Oil 500 mg, Bilberry Extract 40 mg

Flaxseed Oil is derived from the seeds of the flax plant and is one of the richest sources of the Omega-3 fatty acid, Alpha-Linolenic Acid (ALA). Recent reports have linked dietary Flaxseed Oil to increased comfort when used to manage Dry Eye Syndrome.¹ Essential Fatty acids such as Flaxseed Oil work throughout the body to protect cell membranes. Omega-3 Fatty Acids (Flaxseed Oil) have a competitive inhibitory effect on the arachidonic acid (omega-6 derivative) inflammatory cascade and a modular effect on immune cells.^{2,3} Flaxseed Oil also improves meibomian gland secretion by increasing its fluidity, thereby 'unplugging' the gland orifices. A recent study demonstrated that increased intake of Omega-3 fatty acids improved the profile of the polar lipid fraction of meibomian secretions in patients with Dry Eye Syndrome. By enhancing the lipid fraction of the tear film, evaporative loss will decrease.⁴ Chronic blepharitis has associated meibum abnormalities that explain defects in the tear lipid layer that may result in a frankly unstable tear film and that explain the frequently associated evaporative dry eye.⁵

Evening Primrose Oil is an Omega-6 gamma linolenic acid (GLA) that is a downstream metabolite of Omega-6 linoleic acid. This compound is a necessary component in the downstream metabolism of Omega-6 fatty acid to the series one anti-inflammatory prostaglandins (PGE1s), which are associated with healthy mucosal tissue and tear film. The human body cannot metabolize Omega-3 fatty acids to these specific anti-inflammatory prostaglandins. Omega-6 fatty acids that are successfully metabolized or those that have the metabolic advantage of containing GLA reduce inflammation after further metabolizing to dihomogamma-linolenic acid (DGLA), which also blocks, when appropriate, the pro-inflammatory arachidonic acid conversion. They also enhance the delta-6 and delta-5 desaturase enzymatic conversion of Omega-3 alpha-linolenic acid to EPA/DHA and the series 3 anti-inflammatory prostaglandins.⁷ The body requires all of the essential fatty acids for optimal health. They are particularly important for the patient that has Dry Eye Syndrome because PGE1s from Omega-6 (EPO) interrupt the inflammatory loop associated with chronic Dry Eye Syndrome.

Bilberry Extract is a botanical source of an important bioflavonoid for eye health. Bioflavonoids are water-soluble plant-based antioxidants that have additional beneficial effects on eye health. The eyes are supplied with oxygen and nutrients through minute capillaries; bioflavonoids may increase the integrity of these blood vessels, enhancing their function. Research has also demonstrated improvements in patients with retinopathy by supporting blood vessels of the eyes and thereby reducing exudations (fluid seeping through blood vessels).

OTHER INGREDIENTS: Safflower Oil, Lecithin (Precept 8120), d-alpha Tocopheryl Acetate, Gelatin, Glycerin, Purified Water, Caramel Powder.

CONTRAINDICATIONS: Should not be used by the patient without physician supervision, or in those persons showing hypersensitivity to any component of this preparation. Avoid taking at the same time with other medications and/or supplements. Pregnant women should not take Evening Primrose Oil due to increased risk of pregnancy complications. Evening Primrose Oil may lower the seizure threshold and precipitate seizures in patients taking phenothiazines.

WARNINGS: Before taking, tell your doctor if you are pregnant or breastfeeding. Flaxseed Oil may cause ileus (intestinal blockage). Flaxseed Oil may cause a thyroid problem. Flaxseed Oil and Evening Primrose Oil reduce blood vessel platelet aggregation. If already taking aspirin or blood thinners consult your physician – you may need to have your clotting time checked. Evening Primrose Oil may lower the seizure threshold and precipitate seizures in patients taking phenothiazines.

PRECAUTIONS: Keep out of the reach of children. In case of emergency contact a Poison Control Center Immediately. Avoid taking at the same time with other medications and/or supplements.

Pregnancy Category C: It is not known whether this Medical Food can cause fetal harm when administered to a pregnant woman or affect reproduction.

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

ADVERSE REACTIONS: Other than the possibility for allergic reaction in susceptible individuals, no serious side effects are associated with consumption of normal amounts. Flaxseed Oil increases stool bulk and frequency of defecation. It is not recommended for those with bowel obstruction, irritable bowel syndrome or diverticular disease. Stop taking immediately and talk to your doctor if you have any of the following side effects: Breathing problems or tightness in your throat or chest, chest pain, skin hives, rash, or itchy or swollen skin, headache, GI upset, nausea.

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Case Report

toms of cancer, such as anorexia, weight loss, general fatigue, malaise or illness. While 98% of patients with choroidal melanomas do not have metastatic disease detectable at the time of diagnosis, metastasis must be ruled out.⁵ This would most appropriately be handled by an ocular oncologist, so such a referral should be made. Tests include a complete blood count, liver enzymes, abdominal CT, MRI or ultrasound, and a chest X-ray.^{4,5} Several treatment

A few factors to consider are tumor size and location, metastasis status, the visual status of both the affected and unaffected eyes, and the patient's age and overall health.

options are available for choroidal melanomas, but many have high risks involved; therefore, the treating practitioner must carefully weigh many variables when selecting the appropriate treatment modality for each particular patient. A few factors to consider are tumor size and location, metastasis status, the visual status of both the affected and unaffected eyes, and the patient's age and overall health.⁵ Depending on these factors, observation may be a viable management plan if the patient has serious concurrent medical issues, but generally is not advised.

One very aggressive treatment is enucleation, but it comes with significant risks. Half of the patients who are treated with enucleation eventually die of metastatic melanoma. This invasive treatment option is more often discussed if the affected eye is blind, painful, shows optic disc involvement, or if the tumor is very large.⁵

Most small choroidal mela-

nomas are treated with locally destructive therapies, such as thermotherapy, radiotherapy or irradiation.¹² Various types of radiation may be used as treatment.^{4,5} The most common is plaque brachytherapy, which utilizes a radioactive plaque that is sutured on the surface of the globe exterior to the tumor.

This is more commonly attempted with smaller tumors that are 3 disc diameters or more away from the disc and fovea.

Approximately 10% to 15% of patients treated in this way experience local tumor relapse after treatment. Post-treatment, the patient's vision usually remains the same as it was before treatment, but there is a chance that it may improve. However, vision may be subsequently reduced due to secondary effects, such as radiation retinopathy, optic papillopathy, cataracts or neovascular glaucoma.

Photocoagulation may be attempted for small tumors (< 3mm thickness, < 7mm basal diameter).^{4,5} Like photocoagulation for any other reason, a permanent scotoma will result in the photocoagulated areas. Other laser treatments can also be used, including transpupillary thermotherapy, which utilizes a low-power, long-duration infrared laser.^{5,12} This technique may be used in conjunction with plaque radiotherapy, but has not shown a significant improvement in local tumor control.¹³

Other, less common treatments

include local resection, photodynamic therapy or cryotherapy.^{4,5} Often, multiple treatments are used as part of a combination approach.

Sadly, the prognosis for patients with choroidal melanoma often is poor. Despite treatment, 30% to 50% of patients eventually develop metastatic disease; this occurs preferentially to the liver, but also to lung, bone, skin, lymph nodes or central nervous system.^{3,11,14} The same proportion of patients will die within 10 years from diagnosis, usually due to metastatic spread.^{3,11}

Once metastasized, fatality is almost certain.¹¹ The highest incidence of metastatic detection is within a year from choroidal melanoma diagnosis, though it may not occur until years later. Several factors correlate with increased mortality rate, including larger melanoma size, anterior location, extrascleral extension, growth through Bruch's membrane, optic nerve extension, lack of pigmentation, and aggressive cell type and/or mitotic activity.³

It is apparent that this patient did not receive adequate care at his first appointment with the optometry clinic; however, despite appropriate referrals following his second appointment, the chances of a successful outcome were reduced markedly.

Though a delay in referral of a few months may not have significantly altered outcomes in this case, this situation emphasizes the importance of thorough case history and effective doctor-patient communication. It also highlights the importance of appropriate referrals and additional work-ups, regardless of the "long-standing" nature of a condition.

While the prognosis for patients with choroidal melanoma may seem bleak, eye care professionals must institute appropriate treatment as soon as they discover such a lesion to improve the patient's chances of having positive secondary outcomes, including preservation of vision. ■

Dr. Weidmayer practices with a group of optometrists at Eye Center of Lenawee, P.C., in Adrian and Brooklyn, Mich.

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13th Annual Dry Eye Report

Keep On Plugging

Punctal plugs are an effective treatment option for many patients with dry eye syndrome. But, they also may be used in a variety of other ways to facilitate improved ocular health. **By Walt Whitley, O.D., M.B.A.**

When we hear about punctal plugs, the first thing that comes to mind is dry eye treatment. Although frequently used for this purpose, there are several other conditions in which punctal occlusion can be implemented into your treatment plan. Indeed, punctal and canalicular occlusion may enhance the effects of topical medications, help catalyze postoperative healing and improve contact lens wear.

Here, I'll review how punctal occlusion may be used to treat not only dry eye syndrome (DES) but other ocular surface conditions. Also, I'll discuss several non-traditional uses for occlusion that may greatly enhance your patient's overall ocular health and comfort.

The Traditional Use for Occlusion

The most common indication for punctal occlusion is DES.¹

This multifactorial disease of the tears and ocular surface results from insufficient tear production, excessive tear evaporation or abnormal tear composition.^{2,3} DES causes symptoms of burning, itching, redness, photophobia and reduced visual acuity.^{2,4,5}

Multiple studies have shown that nearly five million Americans age 50 or older are affected significantly by DES.⁶ Additionally, millions more Americans experience varying, less severe symptoms of DES.

There are many underlying risk factors for dry eye, including age, female gender, systemic diseases, medications, environmental considerations, contact lens wear and refractive eye surgeries.⁷ The Beaver Dam Eye Study showed that age was the biggest contributory factor to dry eye syndrome.⁷ And, because a large percentage of Americans are now more than 50 years of age, we can expect to see

even more patients present with dry eye symptoms.

There are two subcategories of DES: aqueous-deficient and evaporative. Aqueous-deficient causes include Sjögren's syndrome, lacrimal gland insufficiency, lacrimal duct obstruction and reflex hyposecretion.² Evaporative mechanisms include meibomian gland dysfunction, eyelid aperture disorders, lid-globe incongruity, blink disorders and ocular surface irregularities.²

Irrespective of the subcategory, DES produces tear hyperosmolarity and/or ocular surface inflammation. Tear hyperosmolarity can be caused by either low aqueous flow of excessive tear film or excessive tear film evaporation—both of which can damage the ocular surface by causing an epithelial inflammatory response.^{2,8}

Proper diagnostic testing for DES often helps guide treatment decisions. Diagnostic screening

assesses tear film stability, tear production and flow, and ocular surface stability. Typical tests for DES include tear film break-up time, Schirmer testing, corneal topography, impression cytology, and rose bengal, lissamine green or fluorescein dye testing.² Additionally, tear film osmolarity testing (i.e., TearLab) has been used to identify dry eye patients.

No matter which testing you perform, the first step to effective DES management is to determine whether the patient has ocular surface inflammation. If so, you should consider the use of anti-inflammatory therapies, such as cyclosporine and omega-3 fatty acids supplementation, to optimize ocular surface health before recommending punctal occlusion.

Sometimes, a short course of topical steroids—used simultaneously with the initiation of cyclosporine—will help reduce the sting, allow for rapid symptomatic relief, and/or facilitate the long-term success of cyclosporine dosing. Implementing this strategy to first identify patients with inflammation prevents the potential accumulation of inflamed tears after punctal occlusion, which may exacerbate the ocular surface disease.

The Dry Eye Workshop (DEWS) and the International Task Force (ITF) Delphi Panel on Dry Eye outlined the primary signs and symptoms that could be used to determine DES severity.⁹ More specifically, the ITF created a four-tier, step-wise approach to DES grading and management:^{3,9}

- **Severity Level 1** includes mild to moderate signs and no symptoms. Recommended treatment includes patient education and artificial tears.

- **Severity Level 2** includes

Types of Occlusion

Punctal occlusion may be performed in a variety of ways:

- Temporary occlusion with collagen plugs may help identify patients who likely would benefit from permanent occlusion, or may be an ideal option for individuals who are apprehensive about more permanent treatment options. Collagen plugs typically dissolve within four to seven days.
- Semi-permanent occlusion with punctal plugs are comprised of silicone or thermal labile acrylic polymers, and may last for several months.
- Permanent occlusion can be achieved with the use of silicone plugs and thermal or laser cautery. While silicone plugs do not dissolve, it is important to note that they may extrude or migrate out of the puncta over time.

moderate to severe symptoms, tear film signs, mild corneal punctate staining, conjunctival staining and visual signs. Recommended treatment includes preservative-free artificial tears, gels, ointments, nutritional supplements, cyclosporine A and topical corticosteroids. (Most patients present to our offices when they reach Level 2 signs and symptoms.)

- **Severity Level 3** includes severe symptoms, marked corneal superficial punctate keratitis, central corneal staining and filamentary keratitis. Recommended treatment includes all Level 2 options as well as tetracyclines and punctal plugs.

- **Severity Level 4** includes severe symptoms, such as significant corneal staining, corneal erosion and conjunctival scarring. Recommended treatment includes systemic anti-inflammatory therapy, oral cyclosporine, moisture goggles, acetylcysteine and punctal cautery.^{2,9}

Again, many patients who suffer from DES have found success with topical artificial tears and anti-inflammatories as well as omega-3 fatty acid supplementation. Whether using medications short- or long-term, ocular surface diseases are chronic, co-morbid conditions that wax and wane over time.

Although many practitioners have been able to effectively treat DES with various medications, such as topical cyclosporine, punctal occlusion is often a forgotten adjunct tool.

One reason for this potential oversight may be related to the current FDA labeling of Restasis (cyclosporine, Allergan), which indicates that increased tear production was not documented in patients who were taking topical anti-inflammatory drugs or using punctal plugs.¹⁰ This, however, does not mean that these patients did not experience tear production.

Because patients with punctal plugs already have increased tear volume due to mechanical blockage—and those on anti-inflammatory agents show decreased inflammation associated with many of the symptoms of dry eye—it was more difficult to document statistical improvement in these patient groups.¹⁰

In 2007, Calvin Roberts, M.D., and associates noted a synergistic effect with punctal occlusion and topical cyclosporine use.¹¹ They concluded that, “while topical cyclosporine is being used more and more for the treatment of moderate dry eye, our study results indicate clinicians should not abandon punctal plugs nor

Importance of Patient Education and Punctal Occlusion

Be sure to communicate these points to your patients when considering occlusion:

- Punctal occlusion is a safe, quick, reversible and widely performed procedure.
- Punctal plugs help alleviate symptoms of dry eye, but do not cure the disease.
- Punctal plugs will help increase the contact time of your drops.
- Punctal plugs will not interfere with tear production.
- Collagen plugs are temporary and are often used for diagnostic purposes.
- Permanent silicone plugs do not dissolve and can be removed if complications arise.

consider punctal plugs and cyclosporine mutually exclusive.”¹¹

Non-traditional Uses for Occlusion

According to the ITF, punctal occlusion is recommended for dry eye patients at Severity Level 3.² However, punctal plugs can be used to treat conditions other than dry eye that may disrupt normal tear volume and balance.

• **Compliance issues.** Punctal plugs may play an indirect role in addressing patient non-compliance. Factors that influence patient compliance include: cost of medication, dosing schedule, inability to take drops/meds, forgetfulness, poor understanding of the condition and insufficient trust in their doctors. No matter the reason, poor compliance and adherence to our recommendations can have a significant impact on final outcome.

Simpler, less frequent dosing results in better compliance in a variety of therapeutic classes.¹² Additionally, plugs are not dependent on patient adherence or dexterity for therapeutic efficacy. In fact, punctal plugs may help eliminate compliance issues by reducing the need for consistent artificial tear instillation.¹¹ Furthermore, punctal occlusion increases the residence time of topical therapeutic agents that may be prescribed to address other co-morbid conditions, such

as ocular surface disease.^{11,12}

• **Contact lenses.** Patient discomfort is the leading reason for contact lens dropout. When compounded with poor lens care compliance, discomfort can make successful management of the contact lens wearer almost impossible.

Multiple factors can help facilitate an optimal contact lens wear experience, including good ocular and systemic health, proper lens care, satisfactory compliance, and the type of contact lens material. Once lens wear is optimized, contact lens rewetting drops and punctal occlusion are viable options to address comfort throughout the day and increase tear volume. One study indicated that patients with punctal plugs experienced a 34.6% increase in contact lens comfort within three weeks of occlusion.¹³

The best way to maximize contact lens success is to evaluate the ocular surface prior to the initial fit. If patients show any signs or symptoms of ocular surface disease, treat it accordingly. If patients present with any signs of conjunctival or corneal staining, topical therapy and punctal occlusion may be indicated.

You may need to consider both the lens material and wear schedule for your patients, and transition them into daily wear lenses with low water content, if necessary. Once improved, the

patient will be ready to wear contact lenses comfortably. Additionally, making patients comfortable and happy will keep them in their lenses longer and may prevent them from seeking refractive surgical options.

• **Acute ocular conditions.** As primary eye care providers, we often encounter patients who present with various acute conditions. Although the signs and symptoms may vary, patients often come to us for our expert medical opinion.

Depending on the presentation, appropriate treatment options may include topical lubricants, antibiotics, steroids and combination medications. According to our corneal specialist, John Sheppard, M.D., temporary punctal occlusion increases the contact time of topical medications, which allows the drugs to penetrate into ocular surface tissues and render their desired effect with greater efficacy.

Common conditions in which punctal occlusion may yield some benefit include corneal infiltrates, corneal abrasions, recurrent corneal erosions, filamentary keratitis, superior limbic keratitis, trachoma and neurotrophic keratopathy.

A recent example in our clinic was a 32-year-old white male who was referred for a bacterial corneal ulcer secondary to contact lens wear. He presented with pain O.S., mild mucous discharge and light sensitivity. Clinical findings included 1+ lid edema, 3+ conjunctival injection, 3.0mm mid-peripheral corneal ulcer at 5 o'clock and 1+ anterior chamber cells. We performed cultures to determine the causative pathogen.

We prescribed homatropine 5% t.i.d. O.S., moxifloxacin 0.3% q1h (throughout the night) O.S., a 0.4mm temporary collagen plug



For patients with decreased tear production presumed to be due to ocular inflammation associated with chronic Dry Eye
Don't Wait Until Their Own Real Tears Are Too Low
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Only RESTASIS® is approved to help your patients make more of their own real tears

Indications and Usage: RESTASIS® Ophthalmic Emulsion is indicated to increase tear production in patients whose tear production is presumed to be suppressed due to ocular inflammation associated with keratoconjunctivitis sicca. Increased tear production was not seen in patients currently taking topical anti-inflammatory drugs or using punctal plugs.

Important Safety Information
Contraindications: RESTASIS® is contraindicated in patients with active ocular infections and in patients with known or suspected hypersensitivity to any of the ingredients in the formulation.

Warning: RESTASIS® has not been studied in patients with a history of herpes keratitis.

Precautions: The emulsion from one individual single-use vial is to be used immediately after opening for administration to one or both eyes, and the remaining contents should be discarded immediately after administration. Do not allow the tip of the vial to touch the eye or any surface, as this may contaminate the emulsion. RESTASIS® should not be administered while wearing contact lenses. If contact lenses are worn, they should be removed prior to the administration of the emulsion.

Adverse Reactions: The most common adverse event was ocular burning (upon

instillation)—17%. Other events reported in 1% to 5% of patients included conjunctival hyperemia, discharge, epiphora, eye pain, foreign body sensation, pruritus, stinging, and visual disturbance (most often blurring).

Please see brief Prescribing Information on adjacent page.

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PRECAUTIONS

General: For ophthalmic use only.

Information for Patients

The emulsion from one individual single-use vial is to be used immediately after opening for administration to one or both eyes, and the remaining contents should be discarded immediately after administration.

Do not allow the tip of the vial to touch the eye or any surface, as this may contaminate the emulsion.

RESTASIS® should not be administered while wearing contact lenses. Patients with decreased tear production typically should not wear contact lenses. If contact lenses are worn, they should be removed prior to the administration of the emulsion. Lenses may be reinserted 15 minutes following administration of RESTASIS® ophthalmic emulsion.

Carcinogenesis, Mutagenesis, and Impairment of Fertility

Systemic carcinogenicity studies were carried out in male and female mice and rats. In the 78-week oral (diet) mouse study, at doses of 1, 4, and 16 mg/kg/day, evidence of a statistically significant trend was found for lymphocytic lymphomas in females, and the incidence of hepatocellular carcinomas in mid-dose males significantly exceeded the control value.

In the 24-month oral (diet) rat study, conducted at 0.5, 2, and 8 mg/kg/day, pancreatic islet cell adenomas significantly exceeded the control rate in the low dose level. The hepatocellular carcinomas and pancreatic islet cell adenomas were not dose related. The low doses in mice and rats are approximately 1000 and 500 times greater, respectively, than the daily human dose of one drop (28 µL) of 0.05% RESTASIS® BID into each eye of a 60 kg person (0.001 mg/kg/day), assuming that the entire dose is absorbed.

Cyclosporine has not been found mutagenic/genotoxic in the Ames Test, the V79-HGPRT Test, the micronucleus test in mice and Chinese hamsters, the chromosome-aberration tests in Chinese hamster bone-marrow, the mouse dominant lethal assay, and the DNA-repair test in sperm from treated mice. A study analyzing sister chromatid exchange (SCE) induction by cyclosporine using human lymphocytes *in vitro* gave indication of a positive effect (i.e., induction of SCE).

No impairment in fertility was demonstrated in studies in male and female rats receiving oral doses of cyclosporine up to 15 mg/kg/day (approximately 15,000 times the human daily dose of 0.001 mg/kg/day) for 9 weeks (male) and 2 weeks (female) prior to mating.

Pregnancy-Teratogenic Effects

Pregnancy category C.

Teratogenic Effects: No evidence of teratogenicity was observed in rats or rabbits receiving oral doses of cyclosporine up to 300 mg/kg/day during organogenesis. These doses in rats and rabbits are approximately 300,000 times greater than the daily human dose of one drop (28 µL) of 0.05% RESTASIS® BID into each eye of a 60 kg person (0.001 mg/kg/day), assuming that the entire dose is absorbed.

Non-Teratogenic Effects: Adverse effects were seen in reproduction studies in rats and rabbits only at dose levels toxic to dams. At toxic doses (rats at 30 mg/kg/day and rabbits at 100 mg/kg/day), cyclosporine oral solution, USP, was embryo- and fetotoxic as indicated by increased pre- and postnatal mortality and reduced fetal weight together with related skeletal retardations. These doses are 30,000 and 100,000 times greater, respectively than the daily human dose of one-drop (28 µL) of 0.05% RESTASIS® BID into each eye of a 60 kg person (0.001 mg/kg/day), assuming that the entire dose is absorbed. No evidence of embryofetal toxicity was observed in rats or rabbits receiving cyclosporine at oral doses up to 17 mg/kg/day or 30 mg/kg/day, respectively, during organogenesis. These doses in rats and rabbits are approximately 17,000 and 30,000 times greater, respectively, than the daily human dose.

Offspring of rats receiving a 45 mg/kg/day oral dose of cyclosporine from Day 15 of pregnancy until Day 21 post partum, a maternally toxic level, exhibited an increase in postnatal mortality; this dose is 45,000 times greater than the daily human topical dose, 0.001 mg/kg/day, assuming that the entire dose is absorbed. No adverse events were observed at oral doses up to 15 mg/kg/day (15,000 times greater than the daily human dose).

There are no adequate and well-controlled studies of RESTASIS® in pregnant women. RESTASIS® should be administered to a pregnant woman only if clearly needed.

Nursing Mothers

Cyclosporine is known to be excreted in human milk following systemic administration but excretion in human milk after topical treatment has not been investigated. Although blood concentrations are undetectable after topical administration of RESTASIS® ophthalmic emulsion, caution should be exercised when RESTASIS® is administered to a nursing woman.

Pediatric Use

The safety and efficacy of RESTASIS® ophthalmic emulsion have not been established in pediatric patients below the age of 16.

Geriatric Use

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

ADVERSE REACTIONS

The most common adverse event following the use of RESTASIS® was ocular burning (17%).

Other events reported in 1% to 5% of patients included conjunctival hyperemia, discharge, epiphora, eye pain, foreign body sensation, pruritus, stinging, and visual disturbance (most often blurring).

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Based on package insert 71876US14B Revised February 2010

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U.S. Patent 5,474,979

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Dry Eye

Complications of Occlusion

With the exception of thermal/laser cauterization, punctal occlusion is a reversible procedure that is widely used within eye care. Regarding most types of plugs, the safety profile is very high; epiphora, conjunctival irritation and extrusion typically are the most common—yet infrequent—complications. Other rare complications include canaliculitis and dacryocystitis, which are caused by common bacterial pathogens, such as *Actinomyces israelii*, staphylococci, streptococci and diphtheroids. In the event of complications, patients may require plug removal as well as therapeutic treatment anti-inflammatories and antibiotics. Rarely, surgical removal of plugs is indicated.

O.S. (lower puncta) and had him return the following day. Upon examination, the infection improved with resolution of the cellular reaction and the medications were continued for the following week until complete resolution. Although the condition could have improved without occlusion, the punctal plugs helped increase the contact time of the medications.

- **Glaucoma.** Similar to topical treatment of ocular surface disease, punctal occlusion may benefit glaucoma patients by increasing the penetration and efficacy of IOP-lowering medications. Additionally, occlusion may decrease systemic absorption of glaucoma medications, such as beta blockers, and therefore reduce unwanted side effects. One study group found that nasolacrimal duct occlusion reduces the amount of systemic absorption by up to 60%.¹⁴ Nonetheless, clinical studies have found conflicting results regarding the statistical significance of punctal occlusion for glaucoma therapy.^{15,16}

- **Prevention of herpes simplex keratitis (HSK) reactivation.** Herpes simplex virus (HSV) is the most common cause of corneal blindness secondary to infection in the United States and developed countries.¹⁷ The recurrence rate of ocular HSV within two years ranges from 23% to 33%, and about 20% to 25% of those with ocular HSV infections develop T-cell mediated stromal HSK. In addition to stromal inflammation, HSK patients often exhibit decreased tear production and dry eyes.^{18,19}

Thermal cautery could facilitate therapy simply by enhancing the effectiveness of topically applied drugs. Both thermal cautery and cyclosporine discourage HSV reactivation by targeting dry eye, improving tear film quality and decreasing ocular surface irritation.²⁰

Interestingly, Dr. Sheppard suggested that punctal occlusion—in conjunction with topical cyclospo-

rine—might reduce the frequency and duration of HSV recurrences and consequently minimize the risk of serious ocular damage.²⁰ This may be a more agreeable option for patients who are wary about the permanence of cauterization.

• **Refractive surgery.** Optometrists play an integral role in the peri-operative care of refractive surgery patients. Whether patients are undergoing cataract surgery with premium IOL implantation, laser vision correction, or any other ocular surgical procedure (pterygium removal, glaucoma surgery, etc.), an ocular surface evaluation is an essential aspect of the preoperative evaluation.

Prior to any ocular surgery, our role is to prepare the ocular surface in order to maximize patient outcomes as well as minimize the risk for surgical complications. Pre-existing ocular surface disease, such as DES, can have a tremendously negative impact on the accuracy of the preoperative measurements and overall patient comfort throughout the procedure.

If we can identify these dry eye patients and aggressively treat

their ocular surface with artificial tears, anti-inflammatories, nutrients and/or punctal occlusion before surgery, we can ensure a more favorable postoperative outcome. Although punctal plugs cannot address all preoperative ocular surface concerns, occlusion can help address tear volume levels.

The incidence of dry eye after cataract surgery may be as high as 87%.²¹ Both cataract surgery and laser vision correction transect the corneal nerves, causing deinnervation that typically persists for one to three months following the procedure.

This may produce a neurotrophic cornea, as well as yield clinical signs of epitheliopathy with little to no symptoms other than decreased visual acuity. Other consequences of postoperative dry eye include ocular discomfort, decreased or fluctuating vision and refractive regression.

Whenever patients present with decreased vision following refractive surgery, aggressive ocular surface treatment may determine whether patients need additional surgery. Punctal occlusion may be a viable alternative for these

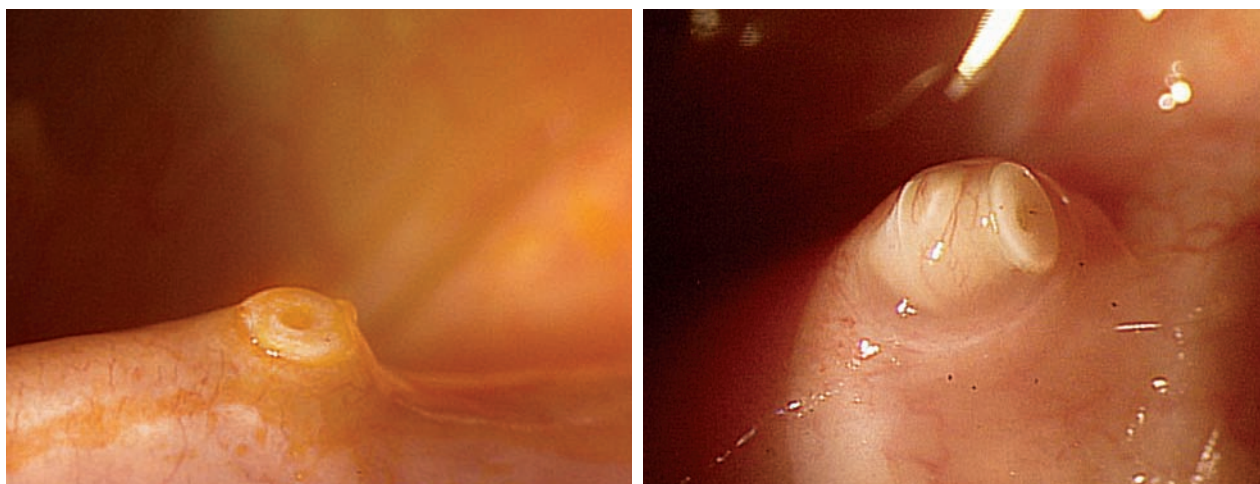
individuals.

Additionally, punctal occlusion promotes wound healing and better visual acuity after certain refractive procedures, such as LASEK. One study documented a lower fluorescein staining score, a better uncorrected distance acuity, and a lower incidence of haze in LASEK patients who received punctal plugs.²²

Future Developments in Punctal Occlusion

Currently, punctal plug delivery systems are being developed that can distribute a sustained time-release dose of glaucoma medication over a three-month period.²³ The Punctal Plug Delivery System (QLT Inc.) is designed to provide a consistent dose of prostaglandin analog in glaucoma patients.

The initial results of the technology's phase II clinical study revealed that 60% of subjects experienced an IOP reduction of 5mm Hg or greater at four-week follow-up.²³ Even more impressive, the Punctal Plug Delivery System's four-week retention rate was 95%.²³ Clearly, such a device could markedly reduce compliance issues for glaucoma patients.



Proper fit of the Odyssey Parasol Plug (left). Pyogenic granuloma formation following punctal plug insertion (right).

Optometry plays an integral role in the treatment and management of many ocular surface conditions. Today, we have several treatment options to address not only DES, but also many other conditions that could be improved by increased tear volume and medication efficacy.

Although punctal plugs chiefly have been used to treat dry eye conditions, practitioners now can consider other potential therapeutic benefits. ■

Dr. Whitley is the director of optometric services at Virginia Eye Consultants in Norfolk, Va.

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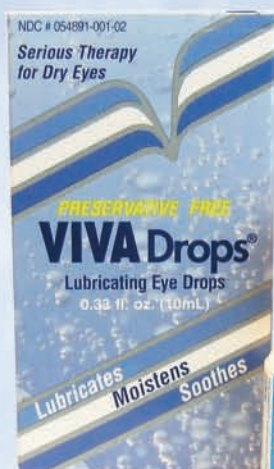
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13th Annual Dry Eye Report

The Impact of Autoimmune Disease

Inflammatory autoimmune conditions can cause significant ocular surface disease and systemic complications. Here's how to manage patients who present with Sjögren's syndrome, rheumatoid arthritis and multiple sclerosis. **By Andrew Morgenstern, O.D.**

KIM KARDASHIAN! Now, I have your attention. Evidently, you can't even read an eye care article today without seeing her name. So, what on earth does Kim Kardashian have to do with optometry? Well, the A-list socialite released a statement in July 2011 indicating that she has the autoimmune disease psoriasis.

She, like many others, has a 30% chance of developing psoriatic arthritis and other advanced inflammatory conditions that could contribute to significant dry eye as well as heart disease, heart

attack, diabetes, high blood pressure, obesity and depression.

We know that inflammation is a primary contributor to many common ocular conditions, including ocular surface disease, age-related macular degeneration and uveitis. This explains why topical corticosteroids and other potent anti-inflammatory drugs are now mainstay therapeutic options for such conditions that we once treated very passively.

Clearly, eye care is not the only branch of medicine to confront problems with inflammation. Inflammation, and more impor-

tantly chronic inflammation, is a fundamental issue for nearly all bodily systems as well as individual anatomical components.

And, for optometrists, it is essential to know that a host of inflammatory autoimmune conditions can have adverse effects on one or many components of the eye.

Here, we'll explore how three of the most common inflammatory autoimmune processes—Sjögren's syndrome, rheumatoid arthritis (RA) and multiple sclerosis (MS)—impact both ocular and systemic health. Additionally, we'll discuss

Release Date: January 2012

Expiration Date: January 1, 2015

Goal Statement: Inflammation is a primary contributor to many common ocular conditions, including ocular surface disease, age-related macular degeneration and uveitis. Here, we'll explore how three of the most common inflammatory autoimmune processes—Sjögren's syndrome, rheumatoid arthritis (RA) and multiple sclerosis (MS)—impact both ocular and systemic health.

Faculty/Editorial Board: Andrew Morgenstern, O.D.

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

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

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

a variety of diagnostic and management strategies for each condition.

Sjögren's Syndrome

In August 2011, tennis superstar Venus Williams pulled out of the U.S. Open, citing fatigue and other issues associated with Sjögren's syndrome. She said, "I have been recently diagnosed with Sjögren's syndrome, an autoimmune disease which is an ongoing medical condition that affects my energy level and causes fatigue and joint pain."¹ Ms. Williams is not alone. More than one million Americans have the disease—90% of whom are women.²

The sicca complex of Sjögren's syndrome presents with a hallmark triad of xerostomia (dry mouth), xerophthalmia (dry eye) and arthritis. These hallmark clinical signs of Sjögren's syndrome are caused by an overproduction of B-lymphocytes (antibodies).³ The onslaught of B-lymphocytes clogs and destroys exocrine glands. In fact, some Sjögren's patients even have trouble perspiring because of obstructed sweat glands.

The most common findings associated with Sjögren's syndrome include:⁴

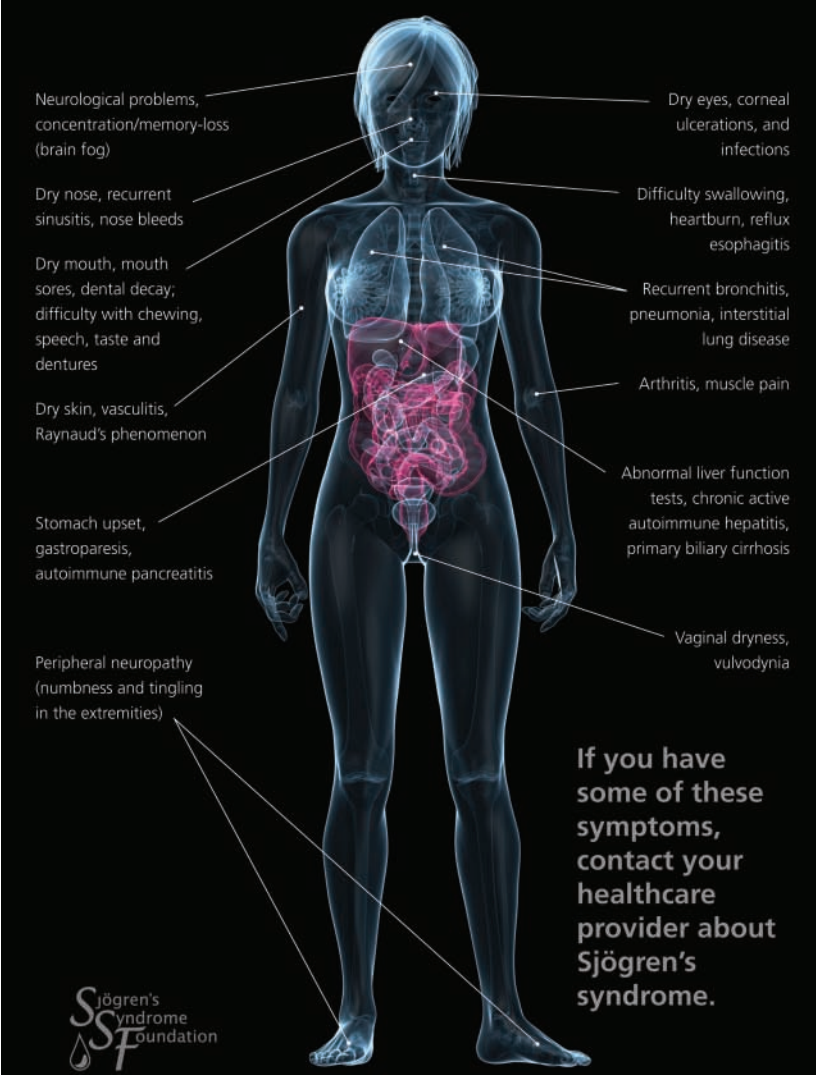
- **Extremely dry eyes.** This includes a gritty or sandy sensation; burning; and redness.

- **Excessive dry mouth and throat.** Patients often experience difficulty chewing and swallowing; a decreased sense of taste; difficulty speaking; an increased incidence of cavities and other periodontal disease; and a dry cough or lingering hoarseness. Additionally, individuals with Sjögren's syndrome may exhibit enlarged or infected parotid glands, which are located at the angle of the jawbone.

- **Persistent fatigue.** This includes lingering aches and pains

Imagine having Sjögren's

Below are the numerous ways Sjögren's can affect your body.



Neurological problems, concentration/memory-loss (brain fog)

Dry nose, recurrent sinusitis, nose bleeds

Dry mouth, mouth sores, dental decay; difficulty with chewing, speech, taste and dentures

Dry skin, vasculitis, Raynaud's phenomenon

Stomach upset, gastroparesis, autoimmune pancreatitis

Peripheral neuropathy (numbness and tingling in the extremities)

Dry eyes, corneal ulcerations, and infections

Difficulty swallowing, heartburn, reflux esophagitis


Recurrent bronchitis, pneumonia, interstitial lung disease

Arthritis, muscle pain

Abnormal liver function tests, chronic active autoimmune hepatitis, primary biliary cirrhosis

Vaginal dryness, vulvodynia

If you have some of these symptoms, contact your healthcare provider about Sjögren's syndrome.



For additional information about Sjögren's contact us at 800-475-6473 or visit online at www.sjogrens.org

Image courtesy of the Sjögren's Syndrome Foundation (www.sjogrens.org).

in both muscles and joints.

Sjögren's patients also may experience other less common symptoms, such as irritation of the nerves in the arms, hands, legs or feet (neuropathy); thyroid gland abnormalities; skin rashes; memory loss or confusion; feeling of numbness or tingling; gastrointestinal problems; inflammation

of the lungs, kidneys, liver or pancreas; and cancer of the lymphatic tissue (occurs in up to 5.8% of patients with the disease).⁵

There are two forms of Sjögren's syndrome—primary and secondary. Primary Sjögren's syndrome is not associated with any other illness. The chief clinical finding of primary Sjögren's syndrome

Lip Biopsy in Detail

Sjögren's syndrome is characterized by chronic inflammation of the glands that produce saliva and tears. An effective way to confirm a diagnosis of Sjögren's syndrome is to biopsy these glands to determine whether inflammation is present.²⁴ If inflammation is noted, the clinician may also determine the type and severity of the underlying condition.

The minor salivary glands located just under the inner surface of the lip are the most accessible of these glands. (These are the "cobblestones" that you can feel when you rub your tongue along the inner surface of your lower lip.) In performing a lip biopsy, the surgeon typically makes a shallow, half-inch incision on either side of the inner lip, after numbing the area with a local anesthetic. Approximately five to seven glands are removed with sterile tweezers. Then, the incision is closed with resorbable sutures. The patient may report soreness of the lip for a few days afterward. Approximately 1% to 2% of individuals will notice localized numbness for two to three months following surgery.²⁵

The lip biopsy has to be evaluated for the presence of a lesion by a pathologist with special training. The offending lesion associated with Sjögren's syndrome is termed "focal lymphocytic sialadenitis," and is characterized by one or more tightly aggregated lymphocytes that are located adjacent to normal gland tissue, surrounding a duct in a 4mm² area of gland tissue. The lip biopsy may reveal the presence of other types of glandular inflammation and/or point to alternative diagnoses, such as sarcoidosis or lymphoma.

It is worth noting that optometrists cannot perform lip biopsies. Instead, it is best to refer the patient to an oral surgeon or an otolaryngologist.

is ocular dryness. This may be confirmed with several diagnostic examinations, including phenol red thread testing, lissamine green staining, Schirmer testing, tear film break-up time, corneal topography or photokeratoscopy, and tear film osmolarity testing.

Additional laboratory tests may also indicate that dry eyes and mouth are caused by associated autoimmune mechanisms. Some examples include testing for the presence of autoantibodies in the blood, known as anti-Ro/SSA or anti-La/SSB.

In challenging cases, inner lip biopsy may be performed to confirm the diagnosis of primary Sjögren's syndrome (*see "Lip Biopsy in Detail," above*). The biopsy may reveal that inflammation is damaging the salivary glands. Some patients, however, have described the lip biopsy as a painful procedure that yields a slow recovery time.

Secondary Sjögren's syndrome develops in the presence of another

autoimmune disease such as RA, systemic lupus erythematosus (SLE) or vasculitis. The condition generally is diagnosed when someone with an established, underlying autoimmune disease experiences extreme dryness of the eyes and mouth. Fortunately for the patient, a diagnosis rarely requires a lip biopsy.

When attempting to make a positive diagnosis of Sjögren's syndrome in our office, we typically refer the patient to a rheumatologist for several reasons. First, rheumatologists are familiar and comfortable with ordering the required blood testing for Sjögren's screening. Also, if the rheumatologist confirms a positive diagnosis, the patient likely will exhibit additional associated symptoms that require management. Finally, a strong relationship with a rheumatologist can be extremely beneficial, because they can refer RA, SLE and sarcoidosis patients to you for dry eye consultations.

Management of Sjögren's Syndrome

Your patients likely will benefit from a frank discussion about the nature of treating a chronic condition. One of my favorite lines to tell a patient at the first visit: "We are going to become long-term friends. I don't have a magic pill to cure you today, and if I can resolve 85% of your problems, we are all going to be happy in the end." Be sure to set your patient expectations accordingly.

Long-term Restasis (cyclosporine, Allergan) use likely will become a part of the treatment regimen. Also, consider a short-term course of corticosteroids to treat significant ocular surface disease and tear dysfunction. According to corneal specialist Stephen Pflugfelder, M.D., "Patients treated with steroid eye drops often have a rapid improvement in ocular irritation symptoms and severity of corneal-epithelial disease. Steroids can jumpstart therapy when they are combined with other therapies, such as topical cyclosporine or oral omega-3 essential fatty acid supplements that require weeks to show improvement."⁶ Dr. Pflugfelder notes that he typically uses corticosteroids (e.g., Lotemax [loteprednol, Bausch + Lomb]) for approximately four weeks, while watching for steroid-related complications.⁶

According to ocular surface research pioneer, Scheffer Tseng, M.D., one of the unique identifying features of Sjögren's syndrome is decreased reflex tearing.⁷ As a result, if you notice a decrease in reflex tearing, punctal occlusion in combination with supplemental artificial tears may be warranted.

In addition, you may consider prescribing one or more medications to treat Sjögren's-associated

dry mouth. One common therapeutic option is Evoxac (cevimeline, Daiichi Sankyo). Evoxac is a cholinergic agonist that binds to muscarinic receptors. In sufficient dosages, muscarinic agonists can increase saliva and sweat secretion from exocrine glands, as well as enhance smooth muscle tone in the gastrointestinal and urinary tracts.

Typically, Evoxac is taken three times a day by mouth. It is contraindicated in patients with uncontrolled asthma, those with a known hypersensitivity to cevimeline, and in instances when miosis is undesirable, (e.g., in patients with acute iritis or narrow-angle closure glaucoma). Common side effects of Evoxac use include excess sweating, nausea and diarrhea.⁸

Ultimately, however, I have limited experience with the treatment of dry mouth and would prefer to comanage such cases with a rheumatologist.

Rheumatoid Arthritis

RA is a systemic collagen vascular inflammatory disease that has a significant impact on ocular surface health. In addition to dry eye, inflammation secondary to RA can cause episcleritis, uveitis and scleritis.

Nearly 1.3 million Americans have RA. The majority of these individuals are 65 years of age or older.⁹ And, according to a 2010 report from the Centers for Disease Control and Prevention, by the year 2030, 65 million Americans age 18 and older will have diagnosable arthritis.⁹

Research has shown that lymphocyte infiltration of the lacrimal glands is the primary cause of associated dry eye.^{10,11} It is interesting to note that about 90% of the RA patients who present to my office with ocular dryness are women over the age of 60.⁸

Management of RA

Management includes systemic suppression of the autoimmune reaction. Unfortunately, however, the indicated oral medications often yield several side effects, including dry eye and decreased aqueous production. These side effects, in combination with the underlying ocular inflammatory effects of RA, often cause moderate to severe ocular surface disease. Common ocular treatment includes artificial tears, corticosteroids, cyclosporine and nutritional supplements.

Disease-modifying antirheumatic drugs (DMARDs) frequently are prescribed to RA patients to slow or sometimes prevent joint inflammation and destruction.¹² Initiating early treatment with DMARDs can reduce the severity of the disease. These medicines are most effective when used long-term.

DMARDs can be divided into two general categories: oral and biologic (*see, "Oral and Biologic DMARDs," right*). Oral DMARDs interfere with the formation or function of immune cells that cause joint inflammation. Biologic DMARDs are administered via intramuscular injection, and act in several different ways to affect immune cell function.

Keep in mind that DMARDs are very potent drugs. Any patient who is taking either oral or biologic DMARDs for RA must be followed closely by a rheumatologist. Additionally, the patient should undergo frequent blood tests and urinalysis to monitor for potentially negative effects on the bone marrow, kidneys and liver.

As is the case with any drug class, patients who use DMARDs may experience several side effects. Some of the most common systemic side effects caused by either oral or biologic DMARDs include:¹³

Oral and Biologic DMARDs

Commonly used oral DMARDs:

- Hydroxychloroquine
- Chloroquine
- Leflunomide
- Methotrexate
- Sulfasalazine

Less commonly used oral DMARDs:

- Azathioprine
- Cyclophosphamide
- Cyclosporine
- Gold salts
- Minocycline
- Penicillamine

Biologic DMARDs:

- Abatacept
- Adalimumab
- Anakinra
- Etanercept
- Infliximab
- Rituximab
- Tocilizumab

- Low white blood cells counts.
- Blood or protein in the urine.
- Liver and/or lung damage.
- Bone marrow toxicity.
- Skin rashes, nausea and hair loss.

In addition to these systemic side effects, patients on DMARD therapy may experience a variety of ocular side effects. These include:¹⁴

- "Bull's eye" maculopathy, which can cause a reduction in best-corrected visual acuity and impaired central vision.
- Retinal pigment epithelium damage.
- Loss of color sensitivity and central vision loss.

Before initiating DMARD therapy, patients should undergo a baseline retinal evaluation, including an optical coherence tomography scan. Then, after beginning therapy, re-evaluate the patient's retinae every six to 12 months for changes.

American-European Consensus Criteria for Sjögren's Syndrome²⁶

In order to make a diagnosis of Sjögren's syndrome, the following criteria must be met:

1. Ocular Symptoms (at least one)

- Symptoms of dry eye that persist for at least three months.
- A foreign body sensation in the eyes.
- Use of artificial tears three or more times per day.

2. Oral Symptoms (at least one)

- Symptoms of dry mouth that persist for at least three months.
- Recurrent or persistently swollen salivary glands.
- Need for liquids to swallow dry foods.

3. Ocular Signs (at least one)

- Abnormal Schirmer's test (without anesthesia; ≤ 5 mm/5 min).
- Positive vital dye staining of the eye surface.

4. Histopathology

- Lip biopsy that shows focal lymphocytic sialadenitis (focus score ≥ 1 per 4mm²)

5. Oral Signs (at least one)

- Unstimulated whole salivary flow (≤ 1.5 mL in 15 minutes).
- Abnormal parotid sialography.
- Abnormal salivary scintigraphy.

6. Autoantibodies (at least one)

- Anti-Ro/SSA or anti-La/SSB, or both.

For a primary Sjögren's syndrome diagnosis:

- Any four of the six criteria; must include criteria from either category 4 or category 6.
- Any three of the four objective criteria (categories 3, 4, 5, 6).

For a secondary Sjögren's syndrome diagnosis:

• In patients with another well-defined major connective tissue disease, the presence of one ocular or oral symptom, in addition to two of the three objective criteria for categories 3, 4 or 5, is indicative of secondary Sjögren's syndrome.

Exclusion Criteria

- Past head and neck radiation treatment.
- Hepatitis C infection.
- Acquired immunodeficiency syndrome (AIDS).
- Pre-existing lymphoma.
- Sarcoidosis.
- Graft vs. host disease.
- Current use of anticholinergic drugs.

Other medications, such as NSAIDs and corticosteroids, often are used to treat the symptoms of RA—not the primary autoimmune condition.¹⁵ Therefore, in cases of dry eye secondary to RA, it is very useful to include a corticosteroid in the treatment regimen to maximize patient relief.

In non-uveitic dry eye secondary to RA, consider Lotemax q.i.d. for one month as well as a long-term

course of Restatis. Because these patients have chronic inflammatory disease, the long-term use of steroids is not advised because of the potential for IOP increase and cataract development.¹⁶

From clinical experience, there is great benefit to long-term Restasis use in most patients who experience dry eye secondary to an autoimmune condition. In addition to improved ocular surface

comfort, many of my patients seemingly have avoided the onset of recurrent uveitis.

Multiple Sclerosis

The relationship between MS and ocular surface disease is very interesting. In MS patients, associated ocular surface disease may be triggered by the underlying condition itself or may occur secondary to neural interruption.¹⁷

MS causes the myelin sheath around some of the most important neurologic components to become inflamed. Myelin is a critical component in the conduction of electrical impulses for proper nerve function. When inhibited, sensory impulses are not conducted properly and can result in poor motor control. Also, the motor response itself can be interrupted by poor signaling/conduction to the end target.

For eye care providers, it is important to know that MS patients can develop severe ocular surface disease from insufficient tear production (which is a motor response to a dry eye stimulus), failure to recognize dryness (corneal sensory issues), lagophthalmos (the lid failing to close properly due to muscle limitations) and an increased risk for uveitis.¹⁸⁻²⁰

Management of MS

If your MS patients experience insufficient tear production, punctal occlusion may be a suitable treatment option. In most cases, punctal occlusion should be combined with artificial tears, omega-3 fatty acid supplementation, artificial tear gel at bedtime, and sometimes topical cyclosporine.

For MS patients with lagophthalmos, you may wish to recommend the addition of a humidifier to the room that they sleep in.

Also, be sure to consider the use of dry eye goggles, Lacriserts (hydroxypropyl cellulose ophthalmic insert, Aton Pharma) and lid scrubs.

Remember, many MS patients may have experienced a previous episode of optic neuritis. If so, their best-corrected visual acuity may be unsatisfactory. So, creating a healthy ocular surface is paramount for achieving the highest possible vision quality.

Additionally, some MS patients have mobility issues that physically restrict their ability to use drops, gels and lid scrubs effectively. So, this potential complication must be actively addressed and considered when prescribing an appropriate treatment regimen.

Generally speaking, excessive mobility restrictions secondary to MS also may result in decreased ocular surface hygiene. Physically restricted patients have a higher incidence of anterior and posterior blepharitis.^{21,22}

Treatment of blepharitis, as outlined by the International Workshop on Meibomian Gland Dysfunction, is essential to optimal vision and overall ocular health.²²

Systemic treatment of MS includes the use of Betaseron (interferon beta-1b, Bayer Health-Care Pharmaceuticals) and Avonex (interferon beta-1a, Biogen Idec.) to either reduce or eliminate inflammatory flareups.²³

The fundamental treatment goal for MS is not to eradicate the disease, but curtail its progression. By minimizing the frequency and severity of associated flareups, the patient has a reduced likelihood of progressive mobility changes, vision loss and associated ocular surface disease.

Remember, individuals with MS have a chronic condition that must

be monitored and managed for life. Unfortunately, because of the progressive nature of MS, a particular treatment may be effective one day, but not two years later.

Ocular surface disease secondary to inflammatory autoimmune disease is very common. When a patient presents to your practice with a prior diagnosis of Sjögren's syndrome, RA or MS, you should consider the likelihood of concomitant ocular surface disease until proven otherwise. In fact, the probability that a patient with autoimmune disease will present with at least one or more signs or symptoms of ocular surface disease is extremely high.

However, by knowing how to effectively treat the associated ocular manifestations of various autoimmune processes, you can ensure that your patients experience the best vision and ocular health possible. ■

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OSC QUIZ

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- Which condition is NOT associated with advanced inflammatory conditions?
 - High blood pressure.
 - Obesity.
 - Dry eyes
 - Epstein-Barr virus.
- What major medication class is often used to treat ocular inflammation?
 - Topical fluoroquinolones.
 - Topical corticosteroids.
 - Anti-malarials.
 - Disease modifying antirheumatic drugs (DMARDs).
- What percentage of Americans with Sjögren's syndrome are women?
 - 60%.
 - 70%.
 - 80%.
 - 90%.

4. Which symptom is NOT part of the hallmark triad of Sjögren's syndrome?

- Arthritis.
- Dry eyes.
- Dry skin.
- Dry mouth.

5. The overproduction of B-lymphocytes is thought to destroy which glands?

- Meibomian.
- Krause's.
- Wolfring's.
- Exocrine.

6. All are forms of dry eye testing and evaluation EXCEPT:

- Tear film osmolarity.
- Ocular Response Analyzer (ORA).
- Corneal topography.
- Tear film break-up time.

7. A biopsy of what anatomical component is an effective way to confirm a diagnosis of Sjögren's syndrome?

- Conjunctiva.
- Meibomian glands.
- Inner lip.
- Lacrimal glands.

8. According to Stephen Pflugfelder, M.D., which drug should be used for four weeks to treat dry eye in patients with Sjögren's syndrome?

- Lotemax (loteprednol, Bausch + Lomb).
- Zirgan (ganciclovir, Bausch + Lomb).
- Nevanac (nepafenac, Alcon).
- Lastacaft (alcaftadine, Allergan).

9. According to Scheffer Tseng, M.D., what is a unique identifying feature of Sjögren's syndrome?

- Lagophthalmos that is greater in the dominant eye.
- Posterior blepharitis without anterior blepharitis.
- Increase in ptosis of the unaffected eye.

d. Decreased reflex tearing.

10. What drug is often used to treat dry mouth in patients with Sjögren's syndrome?

- Hydroxychloroquine.
- Methotrexate.
- Azathioprine.
- Cevimeline.

11. By the year 2030, approximately how many Americans over age 18 will have a diagnosable form of arthritis?

- 1.3 million.
- 6.5 million.
- 13 million.
- 65 million.

12. Common ocular treatments for inflammation and dry eye associated with rheumatoid arthritis include all of the following EXCEPT:

- Artificial tears.
- Biologic DMARDs.
- Corticosteroids.
- Cyclosporine.

13. What are the two primary categories of DMARDs?

- Anti-malarial and biological.
- Biological and anti-arthritis.
- Anti-arthritis and oral.
- Oral and biological.

14. How are biologic DMARDs administered?

- Intramuscular injection.
- Intranasal spray.
- Topical eye drop.
- Orally.

15. What clinical finding is NOT a common ocular side effect of DMARD use?

- Bull's eye maculopathy.
- Cataract formation.
- Retinal pigment epithelium damage.



Bust That Rust!

You *can* remove foreign bodies and rust rings, even those located centrally—if you do it properly and use the right tools. **By Paul C. Ajamian, O.D.**

Q A patient presented to my office five days after getting a piece of metal in his eye. The foreign body was central and seemed deep, so I referred the patient out. Was that the right move?

A There are several reasons why the O.D. might refer out a patient with a foreign body (FB). But, usually these are also the same reasons why we should seize the opportunity to remove the foreign body ourselves:

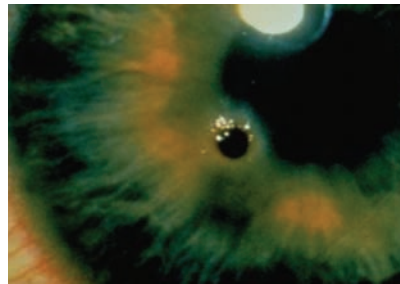
- *The foreign body is central and might leave a scar.* If the FB is going to leave a scar, it's going to leave a scar no matter who removes it. So the sooner you get it out, the better.

- *The patient is in pain.* If so, that's a good reason to remove it right away so that the cause of the pain will be eliminated.

- *The foreign body appears deep.* Many FBs that appear deep are no deeper than the epithelium or anterior stroma. But even if it's in the deep stroma, you can usually take the FB out yourself, as long as you do it properly. Here's how to do it:

- 1. Anesthetize the eye.** Use proparacaine or, if the patient is very sensitive or the FB is at the limbus, consider using Akten (lidocaine 3.5%, Akorn Inc.), an ophthalmic gel that provides deeper anesthesia.

- 2. Identify the foreign body.** While a lot of the FBs we see are metallic, don't assume this one is. But, even if it is an unusual object—such as bugs' wings, spatulas of latex paint, droplets of Super Glue, or just an unidentified foreign body—that in itself shouldn't



Corneal foreign body with rust ring.

prompt you to refer the patient.

Some doctors advocate allowing rust to remain for a few days to allow it to “rise to the surface” or “soften up.” I disagree. The longer rust remains in the cornea, the more likely it will cause an immune response. We've actually seen an immune ring that formed around a FB and started to thin the cornea. So, no matter what the foreign body is, including rust, just get it out.

- 3. Use the proper tools.** You'll need a pair of jeweler's forceps, a foreign body spud (like a golf club spud) and an Alger brush.

Focus your attention first on removing the object (using the spud), and worry about the rust after the FB is out. (You don't want to start with the Alger brush and turn one piece of metal into a million fragments, when you could have taken it out in one piece.)

If there *is* rust left behind, nothing gets it out better than an Alger brush. Place the instrument right on the edge of the crater and apply pressure to clean out the area. Don't worry; if you push too hard or too deep, the drill motor will stop on its own.

- 4. Make sure it's all gone.** With any FB, make sure to evert the upper and lower lids. We occasionally see what's described as an “infectious ulcer,” which is really a FB abrasion caused by an embedded lash or hidden foreign body in the upper or lower lid.

- 4. Prevent infection and pain.**

We no longer patch eyes, even those with large FBs or abrasions. So, to prevent infection of the open epithelial defect, prescribe a short course (up to seven days) of a topical antibiotic. To take the edge off the pain, prescribe a topical NSAID. For patients in severe pain, be prepared to prescribe an oral narcotic analgesic. Copious artificial tears also help with comfort and healing.

- 5. Be on call.** Be available after hours for these patients. Give them your cellphone number or some way to reach you directly if there's a problem. Telling patients to go to an emergency room after hours is a recipe for disaster—and a lawsuit.

- 6. Follow up in 24 hours.** Have the patient come back the next day so you can check the eye's condition. Also, use this as an opportunity to educate the patient about the importance of protective eyewear.

Also, don't forget to document everything in the patient's chart—what you did, what instructions you provided, and any precautions you gave for the future. Last but not least, if you have any suspicion that the FB caused a penetrating injury, perform gonioscopy and dilation. If it does appear to have penetrated, get an X-ray of the eye. ■

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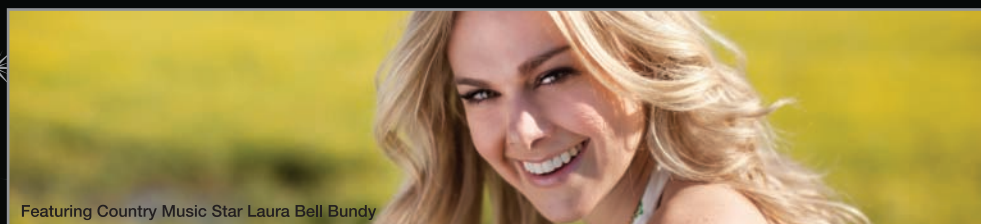
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On Thin Eyes

While CXL complications are rare, operating on a cornea that's too thin can result in some unexpected—and unwanted—snags. **Edited by Joseph P. Shovlin, O.D.**

Q I just had a patient who had bilateral corneal collagen cross-linking (CXL) for post-LASIK ectasia. In one eye, she developed corneal edema and a somewhat significant hypopyon immediately following her procedure. What may have caused this? Is there anything we can do to avoid it in the future?

A Your first step should be to rule out the possibility of infection. “While infection is a rare complication of CXL, there have been a few reported cases of post-CXL infection in the literature,” says Peter Hersh, M.D., director of the Cornea and Laser Eye Institute–Hersh Vision Group in Teaneck, N.J. “Most are bacterial in origin, although herpes simplex keratitis and *Acanthamoeba* have been reported.”

Post-CXL infection is likely a result of the decreased epithelial barrier to microbial infiltration during the healing process after CXL rather than during the procedure itself because CXL kills bacteria and fungi in addition to damaging keratocytes.¹ In some cases, a bandage contact lens that is used postoperatively may contribute, Dr. Hersh says.

A number of interventions may help to avoid infections after CXL. First, you should perform a thorough preoperative examination to look for any ocular surface disease and treat any instances of dry eye or blepharitis.

Dr. Hersh recommends the use of warm compresses, lid hygiene,

tear replacement therapy, topical cyclosporine drops and topical antibiotics, as necessary to address ocular surface disease preoperatively. But, he warns against treating patients with pre-existing herpetic keratitis because of the risk of precipitating recurrent infection.

In the early postoperative period with epithelial removal, the patient should be followed carefully until complete re-epithelialization. Typically, prophylactic antibiotics (usually a broad-spectrum fluoroquinolone) are used until the epithelium has healed completely.

“To help avoid infection, the contact lens should be removed as soon as any epithelial defect from the CXL procedure is healed,” Dr. Hersh says. “If any infiltrate, edema or hypopyon is noted, cultures should be taken as indicated and early treatment with broad-spectrum antibiotics instituted.”

Outside the setting of microbial infection, severe inflammation with hypopyon and corneal edema would be quite rare after CXL, Dr. Hersh says. Typically, a fine corneal haze is seen after cross-linking. Over time, this dust-like haze evolves into a demarcation line,

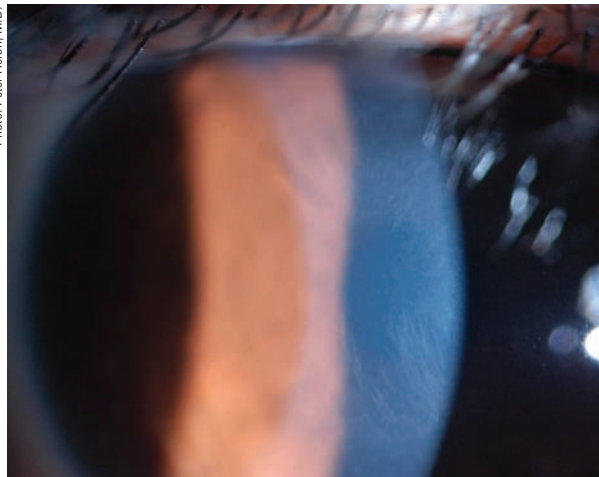


Photo: Peter Hersh, M.D.

Typically, a fine corneal haze is seen after cross-linking. Over time, this dust-like haze evolves into a demarcation line, and subsequently resolves.

and subsequently resolves.² In rare cases, a focal, deep, scar-like haze has been described. Risk factors for such a focal scar remain unclear, but may include pre-existing corneal opacities.

Corneal edema itself is a risk of CXL. “If the cornea is too thin, the interaction of riboflavin and the ultraviolet light potentially may damage the corneal endothelium leading to corneal swelling,” Dr. Hersh says. “Treatment of eyes with appropriate corneal thickness, intraoperative stromal swelling and proper riboflavin diffusion timing should help avoid such complications.” ■

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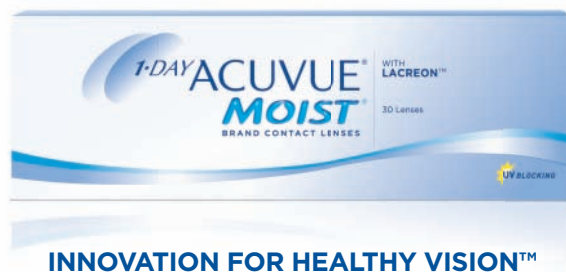
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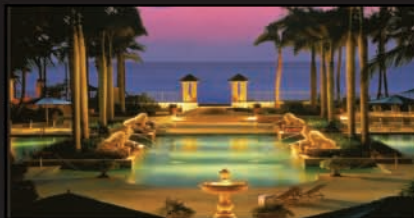
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MS and the Eye (Part 1)

In any patient with unexplained neurologic deficits, consider disease that damages the myelin sheath. **By Carlo J. Pelino, O.D., and Joseph J. Pizzimenti, O.D.**

A demyelinating disease is any condition that results in damage to the myelin sheath that surrounds nerve fibers in the brain and spinal cord. When the myelin sheath is damaged, nerve impulses slow or even stop, causing neurological problems.

Demyelinating diseases may result in vision or hearing loss, headache, seizures, muscle spasms and weakness, loss of coordination, paralysis and loss of sensation.

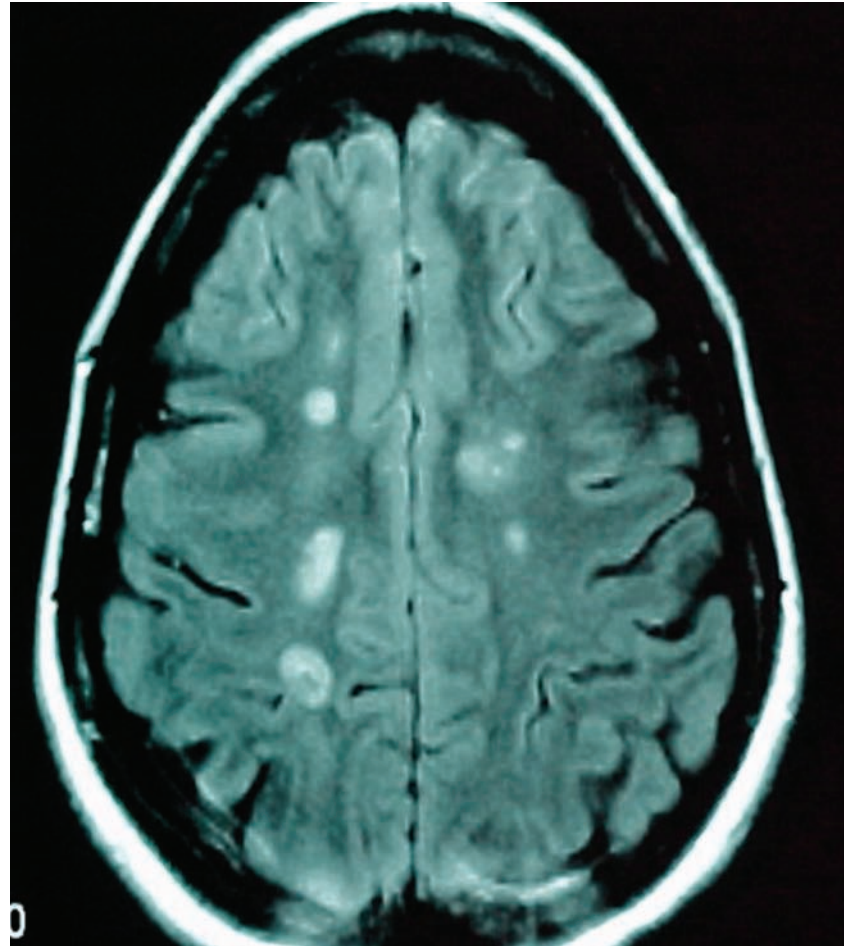
In the first part of this two-part column, we present an overview of demyelinating disease, with a focus on multiple sclerosis (MS)—the most common demyelinating disorder.

Myelin Basics

Myelin is a collection of lipid fats and proteins that covers the long extensions of axons. It considerably increases the speed that the action potentials move down the axons. At rest (resting potential), the neuron and the surrounding space act as a “capacitor” storing current, which is released during the action potential.¹

Myelin is in both the central nervous system (CNS) and the peripheral nervous system (PNS). In the PNS, Schwann cells produce and maintain the myelin; whereas glial cells called oligodendrocytes produce and maintain the myelin in the CNS.^{2,3}

Disorders that affect myelin interrupt nerve transmission.



Notice the multiple ring and ovoid lesions in the periventricular and deep white matter in this patient with MS.

Patient symptoms may reflect deficits in any part of the nervous system. The most commonly affected areas in the CNS are the brain, spinal cord and optic nerves.^{3,4}

In primary demyelinating disorders, the cause is unknown, but an autoimmune mechanism is suspected because the disorder some-

times follows a viral infection or viral vaccination. Demyelination may also occur secondary to an infectious, ischemic, metabolic or hereditary disorder.⁴

MS is a chronic, recurrent disease characterized by disseminated patches of demyelination in the brain and spinal cord (*figure 1*).

But, there are also a number of other primary demyelinating disorders of the CNS (see “Non-MS Primary Demyelinating Disease of the CNS,” below).^{4,5}

A Demyelinating Myelopathy

MS affects approximately 350,000 Americans and 2.5 million people worldwide. The prevalence of MS in the United States ranges from 6 to 177 per 100,000.¹ In Western societies, MS is second only to trauma as the leading cause of neurologic disability beginning in early to middle adulthood.

The number of cases of MS appears to have steadily risen over the past century, and this increase has occurred primarily in women.¹ The disease is three times more common in women than in men, with onset occurring between ages 20 and 40.^{1,2} Approximately 10% of cases begin before age 18; however, the disease is relatively uncommon in children under 10. Geographically, the prevalence rates of MS increase at higher latitudes.

Inflammation, demyelination and gliosis occur in patients with MS. The course can be relapsing-remitting or progressive. Symptomatic episodes consistent with MS are typically “separated in

time and space”—that is, they occur months or years apart and affect different anatomical locations. As an example, a patient may present with paresthesias of the hand that resolve, followed a few months later by optic neuritis. MS is a clinical diagnosis supported by laboratory studies and neuroimaging findings.^{4,5}

What Causes MS?

A proposed explanation for the latitude effect on MS is the possible protective power of sun exposure. Ultraviolet radiation from the sun is an important source of vitamin D, and low levels of vitamin D are common at high latitudes, particularly in winter months. Prospective studies have shown that vitamin D deficiency is associated with increased MS risk, which could be explained by the immunoregulatory effects of vitamin D.^{1,3}

MS also correlates with high socioeconomic status, which may reflect better sanitation and thus delayed initial exposure to infectious agents. Some reports implicate specific infectious agents, such as human herpes virus type 6 or *Chlamydomyces pneumoniae*; although, in general, the available reports have been inconsistent. A number of epidemiologic and laboratory studies suggest that an Epstein-Barr infection rarely may play a role in MS. The virus may precipitate an autoimmune process that attacks myelin. At this time, however, a causal role for Epstein-Barr virus or any specific infectious agent remains uncertain.^{1,3}

Some evidence points to a genetic influence

on the development of MS, with scientists having identified a number of MS susceptibility genes.¹ Whites are at higher risk than Asians and people of African descent, even when residing in a similar environment. Genetic heterogeneity may also be present in MS, meaning that there are different causative genes in different individuals.^{1,3} The risk of developing MS is approximately 20 times greater in first-degree relatives of patients with the disease.³

Although the precise etiology of MS remains unknown, multiple factors appear to contribute. It is thought that the immune system attacks the myelin sheath or the cells that produce and maintain it. This causes inflammation and injury to the sheath—and ultimately to the nerve fibers that it surrounds—and may result in multiple areas of scarring (sclerosis).^{4,5}

We wish all our readers the best of systemic and ocular health in 2012! Keep an eye out for our next column in March, when we'll discuss how to classify, diagnose and manage MS. ■

To read more about MS, see also “The Impact of Autoimmune Disease,” page 64.

Non-MS Primary Demyelinating Diseases of the CNS Systems

- Optic neuritis: inflammation of the optic nerve in one or both eyes
- Neuromyelitis optica (Devic's disease): inflammation of the optic nerve and spinal cord
- Acute transverse myelitis: inflammation of the spinal cord
- Acute disseminated encephalomyelitis: inflammation of the brain and spinal cord
- Adrenoleukodystrophy and adrenomyeloneuropathy: rare, inherited metabolic disorders

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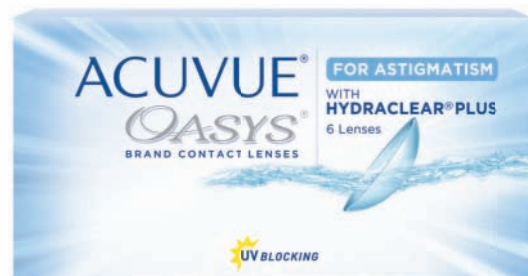


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Patient Sees Better in the Dark

This young patient presented with a 10-year history of extremely poor visual acuity and photophobia. **By Mark T. Dunbar, O.D.**

A 20-year-old Hispanic male presented with a chief complaint of slow, painless, progressive vision loss at distance and near. The patient reported that he had difficulty with his vision since he was 10 years of age. His only other symptom was photophobia. His medical and family ocular histories were unremarkable.

On examination, his best-corrected visual acuity measured 20/200 O.U. at distance and near. Confrontation visual fields were full to finger counting O.U. His pupils were equally round and reactive to light, with no afferent defect. Extraocular motility testing was normal. His anterior segment examination was unremarkable.

The dilated fundus examination revealed clear vitreous and relatively large optic nerves with moderate-sized cups O.U. The retinal vessels were of normal caliber, and

the maculae appeared healthy with a foveal light reflex O.U. We took fundus photographs (*figures 1 and 2*) and performed a spectral-domain optical coherence tomography (SD-OCT) scan (*figures 3 and 4*).

Take the Retina Quiz

1. What simple, in-office test would provide the most useful information about our patient?
 - a. Applanation tonometry.
 - b. Amsler grid.
 - c. Color vision testing.
 - d. Manifest refraction.
2. What additional testing is necessary to help confirm the diagnosis?
 - a. Electroretinogram (ERG).
 - b. Electrooculography (EOG).
 - c. Fluorescein angiography (FA).
 - d. Visual fields.
3. How would you interpret the

SD-OCT?

- a. Normal.
- b. Abnormally thick choroid.
- c. Loss of the photoreceptor integrity line (PIL).
- d. Occult choroidal neovascularization (CNV).

4. What is the most likely diagnosis?

- a. Cone dystrophy.
- b. Stargardt's macular dystrophy (SMD).
- c. Malingering.
- d. Functional vision loss.

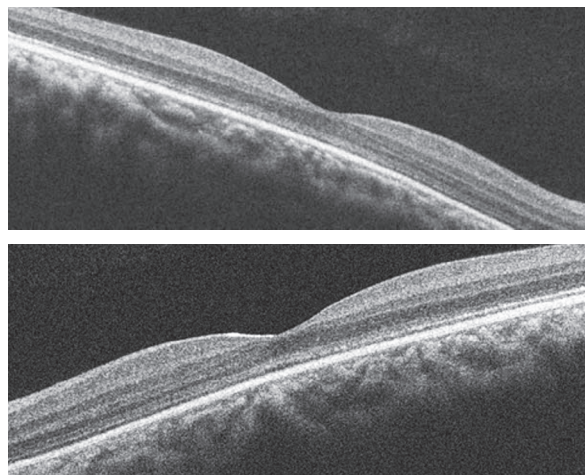
For answers, go to page 98.

Discussion

The dilated fundus exam of our patient showed essentially normal optic nerves and what appeared to be healthy maculae. There was no evident macular edema or any other overt macular pathology. In fact,



1, 2. Fundus photographs of our patient (O.D. left, O.S. right). What clinical finding do you notice?



3, 4. The SD-OCT scan of both maculae (O.D. top, O.S. bottom).

to be progressive in nature and is acquired later in life—unlike other congenital photoreceptor conditions, such as achromatopsia, in which the patient experiences poor vision from birth; nystagmus, an aversion to light (referred to as hemeralopia); and varying degrees of color vision loss.^{1,2}

a foveal light reflex was present in each eye.

So, what was wrong with our patient? Did he have some form of functional vision loss, or was our patient a malingerer who was seeking some financial gain? Our answer was embedded in the case history.

One of his primary complaints was photosensitivity. More specifically, he noted that, even on a cloudy day, he was bothered by light. In addition, he believed that he didn't see as well in normal lighting conditions and that his vision was actually better at night or when the lighting was dimmer. The final clue (which I intentionally omitted from the patient's history above) was color blindness. In fact, the patient missed all 15 plates O.U. on Ishihara color vision testing.

At this point, all the clues suggested that the patient had a cone dystrophy—although SMD was still a potential differential diagnosis. However, FA testing did not show the classic, quiet choroid that is associated with SMD.

Cone dystrophy is considered to be an acquired disorder that affects the cone photoreceptors. It tends

to be progressive in nature and is acquired later in life—unlike other congenital photoreceptor conditions, such as achromatopsia, in which the patient experiences poor vision from birth; nystagmus, an aversion to light (referred to as hemeralopia); and varying degrees of color vision loss.^{1,2}

There are several hereditary patterns of acquired cone dystrophy—all of which result in early loss of color vision as well as a progressive decline in visual acuity (to the level of 20/200 to 20/400).¹

In most cases, vision loss begins during the teenage years; however, initial symptoms may present even as late as the seventh decade of life. Interestingly, our patient was first seen at age 15, and at that time his best-corrected acuity was 20/60 O.U. Over the ensuing five years, his vision slowly dropped to the 20/200 level.

The primary difficulty in making the diagnosis is that, in many instances, the retinal exam can be completely normal. Bull's-eye maculopathy and temporal disc pallor has been reported.^{1,2} In our patient, the retinal exam appeared normal—although you could almost convince yourself that there may have been temporal optic nerve pallor.

The wide variety of clinical presentations illustrates why electrophysiology is so important in confirming the diagnosis. We performed ERG on our patient, which revealed significantly reduced and prolonged cone response and mildly

reduced rod response that was consistent with an acquired cone dystrophy.

The SD-OCT scan is quite interesting. At first glance, it appears normal. However, on careful inspection, you can see that the PIL is absent in the fovea, which suggests that a process is affecting the photoreceptors. The PIL can be seen as a highly reflective line that is located just above the retinal pigment epithelium. It is present outside the macula; but, as you follow it temporal to nasal through the macula, you can see that it disappears on each side.

There are a number of hereditary patterns for cone dystrophy, including autosomal dominant, autosomal recessive and X-linked. Some patients with the X-linked form may exhibit a golden, tapetal-like sheen that disappears on dark adaptation. But, because most cone dystrophies occur sporadically, many researchers believe that the majority of cases are autosomal recessive.¹ Our patient's family history was negative for cone dystrophy, so it is likely that his condition was indeed autosomal recessive in nature.

We referred our patient to the low vision service for evaluation and educated him about helpful programs that were offered by Florida's Division for Blind Services. Additionally, we completed the necessary forms that declared him legally blind, which will grant him access to special assistance programs. ■

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Sowka Down Under, Part II

Resistance is futile... But, it happens anyway.

By Joseph W. Sowka, O.D.

This past fall, I had the opportunity to lecture again at the Tasmania Lifestyle Congress in Hobart, Tasmania, Australia. I enjoy lecturing abroad because it gives me the opportunity to meet colleagues the world over and to see how optometry is practiced elsewhere.

From a therapeutic perspective, optometrists in both Australia and New Zealand are very sophisticated and keep abreast of the literature when delivering care to their patients. Our therapeutic options in the U.S. are very similar. In fact, we use the same glaucoma medications. But, there is one area where I see a major difference: topical antibiotics.

In the U.S., we have a plethora of later-generation fluoroquinolones, such as moxifloxacin, gatifloxacin, levofloxacin and besifloxacin. However, these medications are not available in Australia—and it is unclear if they ever will be. In Australia, the only fluoroquinolones available are ofloxacin and ciprofloxacin, and these medications typically are reserved for cases of bacterial keratitis.¹ Of course, these fluoroquinolones are still available as generics in the U.S., but are not often used because of the availability of newer alternatives as well as concerns of bacterial resistance.^{2,3}

Bacterial keratitis is as common in Australia as in the U.S., with contact lens wear and ocular surgery as precipitating events.^{4,5} Despite the absence of the later-generation fluoroquinolones, Australian eye care providers and their patients do



The author making friends with some of the locals.

not seem to be suffering. Interestingly, there are low resistance rates of *Pseudomonas aeruginosa* and *Staphylococcus aureus* to ciprofloxacin in Australia.¹ You have to wonder how clinicians Down Under can be so successful, yet we in America seem to have significant issues with resistance and consistently need to develop new drugs to combat these heartier microbes.

Emerging Resistance

Antibiotic resistance among ocular pathogens seems to be increasing in correlation with the prevalence of resistant bacteria associated with systemic infections. In other words, systemic treatment of bacterial infections seems to be promoting the development of organisms that are heartier and more resistant to antimicrobial therapy.⁶ So, when one of these heartier organisms infects the conjunctiva or cornea, there may be fewer therapeutic options.

We have always looked upon heavy systemic use of antibiotics as well as antibiotic treatment of livestock feed as primary culprits

for ophthalmic resistance. And, although the rise in resistant ocular bacteria is apparently linked to the increase in resistant systemic pathogens, recent evidence has identified the emergence of resistant bacteria in the eye to prior topical antibiotic therapy.⁶ In any case, either of these postulations contributes to the emergence of resistance among ocular pathogens. (To that end, besifloxacin is one of the latest fluoroquinolones that has no systemic counterpart. It was developed exclusively for ophthalmic use with the hope that it will decrease the development of resistant microbes because of no systemic use.)

A recent surveillance study saw that a large proportion of *S. aureus* and coagulase-negative staphylococci isolates were resistant to oxacillin/methicillin, azithromycin and fluoroquinolones.⁷ Another study indicated that methicillin-resistant staphylococci were more likely to be resistant to fluoroquinolones, aminoglycosides and macrolides.⁸

There are two schools of thought regarding antibiotic choice and the development of resistance.

- *High risk.* This concept involves using the most potent antibiotics only in high-risk cases, such as bacterial keratitis. In most countries outside the U.S., topical fluoroquinolones—particularly those recently approved by the European Medicines Agency, including levofloxacin and moxifloxacin—rarely are used. The strategy of using topical fluoroquinolones as a last resort reflects a belief that the agents may enhance

the development of resistance, jeopardizing future availability of antibacterial treatment for ocular infections.⁹

- **High concentration.** The other school of thought suggests that the use of topical fluoroquinolones, which results in antibacterial concentrations at the ocular surface that can significantly exceed mutant prevention concentrations (approximately 10 times the minimum inhibitory concentration), should be preferentially used in order to prevent the development of resistance. In the case of later-generation fluoroquinolones such as topical moxifloxacin, a dual-step mutation is required for resistance to emerge. Moxifloxacin restricts the selection of resistant mutants, meaning that emergence of resistance is unlikely.⁹

It is not clear which strategy is most appropriate. Despite the availability of just two fluoroquinolones in Australia, resistance rates are quite low. A study from New South Wales, Australia, indicated that ocular infections—both conjunctivitis and keratitis—involving *S. aureus* in the U.S. exhibited greater resistance to antibiotics than those in Australia.¹⁰ Additionally, the researchers noted that isolates from corneal infections were more resistant to antibiotics than those from conjunctival infections, with isolates from the U.S. demonstrating the greatest resistance levels.¹⁰

Nonetheless, there have been some issues with bacterial resistance in Australia. For example, there is an emerging resistance to cefazolin, which commonly is used as a first-line antibiotic for gram-positive cocci.¹¹ However, most microorganisms isolated from patients with bacterial keratitis in Australia showed susceptibility to ciprofloxacin and aminoglycosides.¹²

So, if clinicians in Australia

typically restrict their use of the two available fluoroquinolones to cases of bacterial keratitis, what do they use for other conditions?

Chloramphenicol Revisited

In the January 2008 column, I discussed the widespread use of topical chloramphenicol in New Zealand. In Australia, as in New Zealand, topical chloramphenicol is the most widely used ophthalmic antibiotic. Moreover, it is used extensively throughout the world (except in the U.S.) for the treatment of acute bacterial infections and corneal trauma. Also, due to its excellent ocular penetration, chloramphenicol is very popular in surgical prophylaxis.

Chloramphenicol has a broad spectrum of both gram-positive and gram-negative antibacterial activity, and it is effective against anaerobic organisms, *Mycoplasma*, *Rickettsia* and *Chlamydia*. Especially noteworthy is its low rate of clinical and microbiologic resistance as well as its ability to conquer organisms that are resistant to more common antibiotics.¹³⁻¹⁶ Chloramphenicol's efficacy against ocular methicillin-resistant *Staphylococcus aureus* (MRSA) infections has also been documented in several investigative reports.¹⁷⁻²⁰

To date, there have been 23 cases of aplastic anemia (the majority were not fatal) reported in the U.S. that possibly were associated with the use of topical chloramphenicol.¹³ This purported association with aplastic anemia has curtailed use of the drug in the U.S.

We have long shied away from topical chloramphenicol due to its perceived threat to patient health and risk of death. But, are Australian optometrists and ophthalmologists worried about chloramphenicol

use? Doesn't seem so. How do I know? Because, in Australia, topical chloramphenicol is sold over the counter without a prescription! ■

Dr. Souka has no direct financial interests in any of the products mentioned.

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A Look At MSI

Multispectral imaging may help eye care providers diagnose retinal conditions earlier than conventional funduscopy. **Edited by Diana L. Shechtman, O.D., and Paul M. Karpecki, O.D.**

Multispectral imaging (MSI) is an emerging diagnostic technology that permits the clinician to dissect and visualize the retina in spectral slices, from the inner limiting membrane all the way to the choroid.

Most importantly, however, MSI may help clinicians detect and evaluate several sight-threatening retinal and choroidal diseases, including diabetic macular edema, retinal pigment epithelium atrophy, vitreomacular traction syndrome and wet age-related macular degeneration.

Here, we'll examine how MSI technology can help eye care providers effectively diagnose and manage retinal pathology.

Multispectral Overview

The concept of MSI was first described in 1977, when a research team combined a modified fundus camera with various interference filters to enhance the appearance of anatomical and pathological retinal features.^{1,2} However, this modified

camera design was hampered by the use of filters and a bandwidth that was too wide, which resulted in diminished contrast and poor spectral separation.

Current, commercially available MSI devices, such as the RHA instrument (Annidis Health Systems), use multiple monochromatic LED-sourced wavelengths—ranging from 550nm to 780nm—to illustrate the individual anatomic components of the retina. Generally, shorter wavelengths are used to reflect anterior retinal features and longer wavelengths are used to reflect deeper retinal features, including the choroid.

RHA also has additional after-image processing that highlights oxygenated and deoxygenated hemoglobin, which may yield representation of metabolic activity (*figure 1*).³ This may allow the eye care practitioner to accent the retinal structures associated with arterial and venous blood by non-invasive means. One study described this

process as a means of demonstrating oxygen saturation through morphological angiography.³

MSI in Clinical Practice

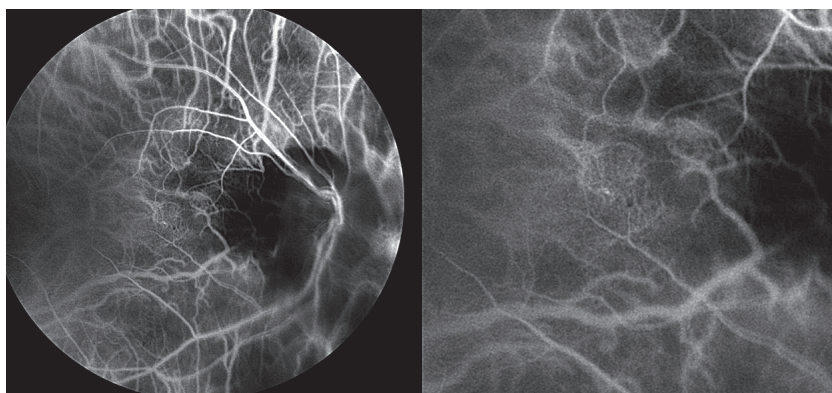
Today, MSI takes digital imaging to a new diagnostic level by yielding an enhanced view of the anatomical fundus layers. Separation of anatomical layers allows pathology-specific ophthalmoscopy and progressive views through the entire retina—from the internal limiting membrane to the choroid.

MSI is capable of creating a series of monochromatic, en face fundus spectral slices for added diagnostic insight. And, because conventional fundus photography is limited to parameters of the visible spectrum, it is less sensitive than MSI in detecting features that are reflected in the longer range of the spectrum such as deep retinal layers and the choroidal structures.

Furthermore, MSI may help a managing clinician make a more informed diagnosis. The device aids in:

- Localization of retinal morphological abnormalities.
- Interpretation of disease, based upon affected layer.
- Enhanced viewing of obscure, subtle or overlapping pathological structures.
- Enhanced viewing of deep retinal structures.

Preventative eye care is predicated on early detection, and being able to identify risk factors and preliminary clinical signs is essential. MSI affords



1. Oxy/deoxy map of a patient with myopic degeneration. Note the visualization of the choriocapillaris in the magnified image (right).



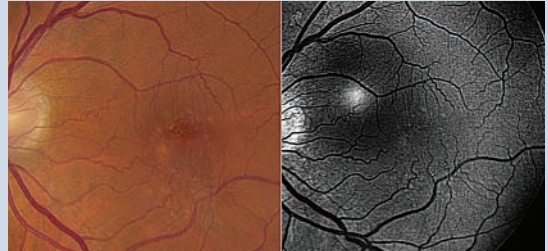
eye care providers the means to understand the anatomical aspects of specific pathologies, as well as perform further examination of vascular tissues, fluids and metabolic markers of retinal health and integrity. Ultimately, with increased use, MSI will help facilitate earlier and more accurate detection of many sight-threatening retinal conditions. ■

Thanks to Richard Maharaj, O.D., B.Sc., of Hamilton, Ontario, for contributing this column. Dr. Maharaj sits on the U.S. optometric advisory board for Annidis Health Systems, Inc.

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Case Report: MSI Helps Confirm Diagnosis of VMTS

A 54-year-old white male presented with signs of small-caliber drusen in his left eye that were evident with fundus imaging. Interestingly, his visual acuity measured 20/20 O.U. In addition to funduscopy, we performed multispectral imaging (MSI) and spectral-domain optical coherence tomography (SD-OCT).



Fundus (left) and MSI shorter wavelength (right) images of our patient's left eye reveal surface traction secondary to VMTS. Note the enhanced anatomical detail on MSI shorter wavelength.

Both MSI and SD-OCT indicated the presence of vitreomacular traction syndrome (VMTS). MSI shorter wavelength effectively captured the topographical striations associated with the VMTS. Also, MSI further identified an atrophic area associated with the decomposition of melanin in the retinal pigment epithelium (RPE). On SD-OCT, we saw that the photoreceptor integrity line was intact, which was why this patient—with significant RPE atrophy—remained asymptomatic.

We recommended oral carotenoid supplements as well as an AREDS-equivalent multivitamin. Additionally, we prescribed 1,000 IU vitamin D daily to address the inflammatory nature of the pathology.

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Product Review

Contact Lenses

Specialty Line

ABB Concise received FDA clearance to produce an entire line of specialty contact lenses in silicone hydrogel 60Dk, 74% H₂O Definitive material. This includes indications for use in toric, multifocal, multifocal toric and irregular cornea lenses as well as the KeraSoft IC Lens manufactured under the Bausch + Lomb license and offered exclusively in the Definitive material. ABB plans to launch the new product line in the first quarter of 2012.

Website Enhancement

VirtualTryOn

Polarized sunglass maker Maui Jim has added VirtualTryOn to its website. The new system provides enhanced information about each sunglass style and allows customers to see themselves in a pair of glasses and then post the images to Facebook. It has



two options—VirtualFitLive and VirtualFitPhoto.

VirtualFitLive is the live webcam option that projects a live image of the user's face; then the system superimposes sunglasses onto the image. If the user turns their face, the glasses move with them. VirtualFitPhoto lets users upload photos of their face, either by snapping photos through their computer's webcam or by uploading a photo from their files. The chosen sunglasses will appear on the photo, and can be saved or shared with friends on Facebook. To try it out, visit www.mauijim.com/tryonlive.html.

Frames

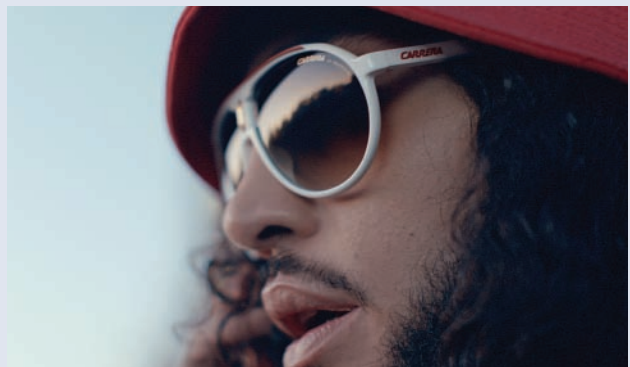


Aspex Grilamid TR90

The Aspex Grilamid TR90 was developed with thermoplastic polyamide, a new, advanced polymer developed exclusively for Aspex that is 20% lighter than other plastics. Frames using TR90 are now available in all Aspex brand lines, including EasyClip, Manhattan Design Studio and Takumi Magnetic Eyewear. In addition to being flexible, durable and lightweight, frames made from TR90 are temperature resistant, the company says. TR90 frames are also non-allergenic and block damaging UV exposure. For more information, visit www.aspexeyewear.com.

Carrera Champion Sunglasses

Worn by Gym Class Heroes lead singer Travis McCoy, Carrera's Champion sunglasses appeared in the band's latest music video "Ass Back Home." Sleeping on buses and in hotel rooms in different cities every night, the documentary-style video showcases the hard, taxing lifestyle of an artist while on tour. The Carrera "Champion" sunglass model is inspired by the original design first introduced in the early 1980s and produced in Safilo Group's Optyl, a lightweight, hypoallergenic material. Visit carreraworld.com.



Frames



Costa Double Haul

Serious anglers will appreciate Costa's signature vent system in Double Haul's frame front to alleviate lens fog in extreme weather conditions, as well as full-eye coverage to allow full range of vision while on the water. Double Haul features a large fitting frame with Hydrolite no-slip nose pads, sturdy integral hinges and durable co-injected molded temples for a comfortable fit. The new style is available in tortoise, black and the new translucent crystal frame colors.

Anglers can customize Double Haul in Costa's patented 580 lenses in either glass or polycarbonate (580P). The new style will retail from \$179 to \$249 depending on lens customization, and will be available at www.costadelmar.com and at authorized Costa retail outlets.

Karl Lagerfeld Eyewear Collection

Marchon debuted the Karl Lagerfeld Fall/Winter collection for men and women this season. Shapes from the women's sunwear collection are vintage-inspired and amplified by use of colors—dark hues that graduate to light and then are infused with a contrasting color burst or rich tortoise shells and horns. The men's collection showcases retro shapes with modern color gradients and the K temple exemplifies skilled craftsmanship. The collection features ophthalmic and sunwear styles, including:



KL747S

- K747S. A thin, polished metal bar is inlaid at the temples, beginning at the end pieces and continuing to the mid temples, punctuated by the "KL" logo. Lagerfeld enhances the depth of design by setting the inlaid metal against color gradients, specifically a purple/violet gradient and grey/orange gradient to enhance the cat eye shape.

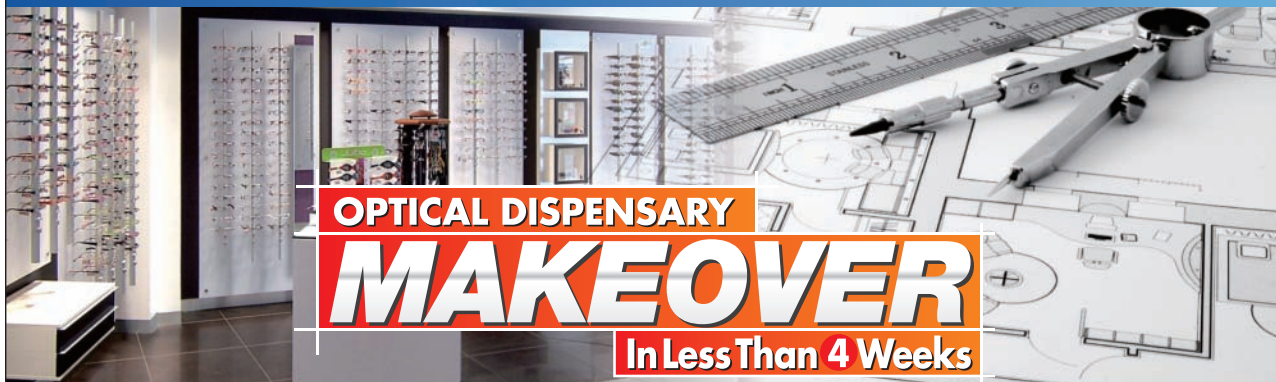


KL748S

- KL748S. This sister style to the KL747S is a modified butterfly, evident on the slightly waved brows crafted from rich zyl. Thin, polished metal beginning at end pieces and continuing down the temples toward the "KL" logo is prevalent set against black, Havana, light tortoise and sand colorations. ■

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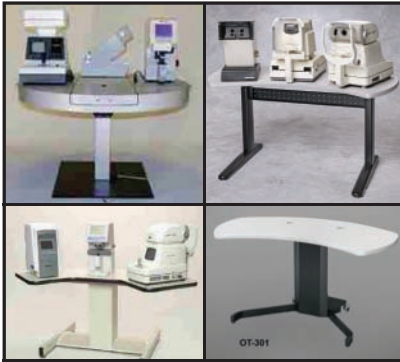
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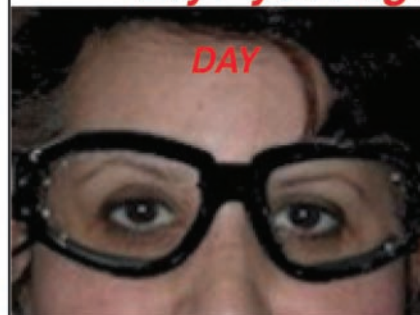
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- **27-29.** *2nd Annual Final Eyes CE Event.* Baptist Medical Center in the duPont Auditorium, Jacksonville, Fla. CE hours: 16. Contact Valerie Fernandez, CME Coordinator, at (904) 202-2080 or valerie.fernandez@bmcjax.com. For more information, visit FinalEyesCE.com.
- **28.** *Snow School 2012.* Great Wolf Lodge, Scotrun, Pa. Hosted by: The New Jersey Society of Optometric Physicians. CE hours: 6. Call (609) 323-4012 or visit www.njsop.org.

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- **4-9.** *26th Annual Eye Ski Conference.* The Lodge at Mountain Village, Park City, Utah. CE hours: 20. Contact Tim Kime, O.D., Meeting Director, at tandbkime@buckeye-express.com. For more information, visit www.eyeskiutah.com.
- **11-12.** *75th Great Lakes Optometric Congress.* Chicago/ Northbrook Hilton, Northbrook, Ill. Hosted by: The Optometric Extension Program Foundation. CE hours: 13. Contact John Loesch, O.D., at drjohnod1@gmail.com. For more information, visit www.oepf.org.
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- **13-14.** *OAOP Annual Spring Congress.* Embassy Suites & Conference Center, Norman, Okla. Hosted by: the Oklahoma Association of Optometric Physicians. CE hours: 21. For more information, visit www.oaop.org.
- **14-15.** *4th Annual Symposium on Ocular Disease.* Crowne Plaza Hotel, Tyson's Corner, Va. Hosted by: PSS EyeCare. CE hours: 16. Call (203) 415-3087 or e-mail education@psseyecare.com. For more information, visit www.psseyecare.com.
- **20-21.** *Educational Meeting 2012.* Mission Inn, Howey-in-the-Hills, Fla. Hosted by: the Florida Chapter of the American Academy of Optometry. CE hours: 10. For more information, contact Arthur T. Young, O.D., at eyeguy4123@msn.com or (239) 542-4627.
- **20-22.** *WFOA 2012 Spring Seminar.* Sandestin Hilton Beach Resort, Destin, Fla. Hosted by: the West Florida Optometric Association. CE hours: 18. For more information, contact Tom

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Streeter at (850) 279-4361 or opttom@hotmail.com. Visit <http://wfoameeting.com>.

■ **21-22.** *20th Annual Suncoast Educational Seminar.* Hyatt Regency Clearwater Beach Resort & Spa, Clearwater Beach, Fla. Hosted by: The Pinellas Optometric Association. For more information, contact Dr. Bruce Cochran at (727) 446-8186.

■ **25-29.** *10th Annual New Jersey Chapter—American Academy of Optometry.* Kingston Plantation, Myrtle Beach, S.C. CE hours: 16. For more information, contact Dennis H. Lyons, O.D., at (732) 920-0110 or dhl2020@aol.com.

May 2012

■ **3-5.** *MWCO Annual Congress.* Caesar's Palace, Las Vegas. Hosted by: Mountain West Council of Optometrists. Contact Tracy Abel, CMP, at (888) 376-6926 or tracyabel@earthlink.net. For more information, visit www.mwco.org.

■ **18-20.** *Nova Southeastern University's 16th Annual Clinical Eye Care Conference & Alumni Reunion.* NSU College of Optometry. CE hours: TBD. Contact Vanessa McDonald, MS, Manager of Continuing Education, at (954) 262-4224 or oceaa@nova.edu. For more information, visit <http://optometry.nova.edu/ce>.

June 2012

■ **10-24.** *Majestic China 2012.* Hosted by: iTravelCE, LLC. CE hours: 20. Contact Dr. Bridgitte Shen Lee, at (832) 390-1393 or info@itravelce.com. For more info, visit www.itravelce.com.

■ **21-24.** *Maui 2012.* Wailea Beach Marriott Resort & Spa, Maui, Hawaii. Contact Lois DiDomenico at ReviewMeetings@Jobson.com or (866) 658-1772. For more information, visit www.revoptom.com/conferences.

July 2012

■ **19-22.** *Caribbean 2012.* Ritz Carlton, San Juan, Puerto Rico. Contact Lois DiDomenico at ReviewMeetings@Jobson.com or (866) 658-1772. For more information, visit www.revoptom.com/conferences.

August 2012

■ **3-5.** *Educational Retreat 2012.* South Seas Island Resort, Sanibel, Fla. Hosted by: Southwest Florida Optometric Association Inc. CE hours: 12. Contact Brad Middaugh, O.D., at (239) 481-7799 or swfoa@att.net. For more information, visit www.swfoa.com.

To list your meeting, contact:

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Right Between the Eyes

By Andrew S. Gurwood, O.D.

History

A 35-year-old black female presented following an emergency room visit for a chief complaint of pain around both eyes.

The patient was in considerable distress, but did not report altered vision. She had no known ocular history and reported no allergies to medications.

Diagnostic Data

Her best-uncorrected visual acuity was 20/20 O.U. at distance and near. External examination was normal, and there was no evidence of afferent pupillary defect.

The anterior segments of both eyes were normal underneath the swollen adnexa. Intraocular pressure measured 19mm Hg O.U. The dilated fundus findings were normal.



Our patient presented with pain around both eyes. What is your diagnosis?

Your Diagnosis

How would you approach this case? Does this patient require any additional tests? What is your diagnosis? How would you manage this patient? What's the likely

prognosis?

To find out, visit www.revoptom.com. Click on the cover icon for this month's issue, and then click "Diagnostic Quiz" under the table of contents. ■

Retina Quiz Answers (from page 81): 1) c; 2) a; 3) c; 4) a.

Next Month in the Mag

Our February issue covers the latest research in pharmaceuticals.

Topics include:

- *Optometric Study Center: Topical and Oral Prescribing for Pain Management* (earn 2 CE credits)
- *Cost vs. Efficacy of Generic and Substitution Pharmaceuticals*

Also in February:

- *Are SiHi Lenses Living up to Their Promise?*
- *Pseudoexfoliative Glaucoma Management*
- *Special Diagnostic Equipment for a Pediatric Population*
- *Counseling, Educating and Managing the New Presbyope*

And...

- Don't miss our annual Ophthalmic Product Guide.

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Review of Optometry welcomes questions and comments. E-mail Amy Hellem, editor-in-chief, ahellem@jobson.com, with "Letter to the Editor" as the subject line.

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