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THE TOP 10 TO TRACK

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Hydrogel and overnight orthokeratology (OK) contact lens (CL) wear can increase tear film osmolarity, a new study finds. Researchers observed osmolarity values in non-CL wearers and those who have worn hydrogel and overnight OK lenses for at least three years, and found osmolarity was within normal limits in all groups, although it was significantly lower in non-CL wearers compared with hydrogel and OK lens wearers. Osmolarity returns to baseline one month after discontinuation of CL wear.


Researchers found day one post-op assessment with swept-source OCT provided better visualization of the macula than spectral-domain (SD) OCT in patients treated with pars plana vitrectomy and gas tamponade. For patients with rhegmatogenous retinal detachment, SD-OCT provided good visualization on day one in 35.3% of eyes compared with 73.5% of eyes using SS-OCT’s line scan protocol. In eyes with macular holes, the rates were 59.3% with SD-OCT and 88.9% with SS-OCT’s line scan protocol.


Clinicians should endeavor to refer patients with orbital-floor trap-door blowout fractures with incarcerated tissue for surgery within eight days, according to new research. The study found ocular motility outcomes were significantly better for patients who underwent repair surgery within eight days of injury than those after eight days.


Clinicians have long known intracranial pressure (ICP) plays a critical role in idiopathic intracranial hypertension, and “there has also been debate about its potential role in other disease states such as glaucoma,” says Nate Lighthizer, OD, an associate professor and assistant dean of Clinical Care Services at Oklahoma College of Optometry. “There is current thinking now that perhaps glaucoma is a two-pressure disease: the balance between intraocular pressure and intracranial pressure. So better understanding the role ICP plays in many disease states may be a critical element in the future.”

Unfortunately, the standard test for diagnosing elevated ICP, lumbar puncture (LP), is invasive and painful. Researchers have sought noninvasive methods for dynamically monitoring ICP, and think ultrasonography may be the key. Recently, researchers found measuring optic nerve sheath diameter with ultrasonography may be a noninvasive method of monitoring ICP.

Researchers in China used ultrasonography to measure the optic nerve sheath diameter (ONSD) in 60 patients admitted for lumbar puncture. After LP, the patients were split into two groups, those with elevated ICP between 200mm H2O and 300mm H2O and those above 300mm H2O. The investigators noted a strong correlation between the ONSD and ICP values on admission, and a strong correlation between the change in ONSD and ICP values one month later. Post-treatment, the elevated ICP and dilated ONSD had returned to normal, the researchers found.

Other studies have highlighted ultrasonography’s utility in (continued on page 5)
FDA Hits Opternative with Notice

The Food and Drug Administration (FDA) has advised online refraction company Opternative that its services constitute a medical device and, as such, require “marketing clearance,” according to a letter the agency issued in October, but didn’t publicize until March. The letter indicates that the company, which was sending marketing e-mails as of March 22, has been operating in violation of the Federal Food, Drug, and Cosmetic Act. In the letter, the FDA “requests that Opternative, Inc. immediately cease activities that result in the misbranding or adulteration of the On-Line Opternative Eye Examination Mobile Medical App device, such as the commercial distribution of the device through your online website.”

“We responded promptly to FDA’s warning letter from October 2017, and we are working diligently to voluntarily comply with all regulatory requirements,” says a representative from Opternative. “We continue to communicate with the FDA on a regular basis to work through the regulatory medical device clearance process with our outside experts.”

The FDA’s letter also says that the agency determined that the app “is a device because it is intended for use in the diagnosis of disease or other conditions or in the cure, mitigation, treatment, or prevention of disease, or to affect the structure or any function of the body.” It also outlines how Opternative can submit for premarket approval and “whether the product may be legally marketed.”

Clinical Acumen Boosts AI Accuracy

Although artificial intelligence (AI), and the machine learning algorithm that drives it, shows promise as a screening tool for ocular pathology such as diabetic retinopathy (DR), it’s not perfect. But researchers at Google found using a small set of DR cases judged by ophthalmologists and retina specialists can improve the algorithm’s accuracy.1

The researchers had the clinicians and the algorithm grade retinal fundus images from DR screening programs and then chose the consensus of the retina specialists as the reference standard. After adding the reference cases “as a tuning dataset,” the investigators looked at the area under the curve (AUC), sensitivity and specificity data between the manual grading and the algorithm. With the new data, the algorithm improved its AUC from 0.934 to 0.986 when screening moderate or worse DR, a boost that enabled the AI system to perform similarly to or even exceed US board-certified ophthalmologists and retinal specialists, according to study author Lily Peng, MD, PhD.1,2

Although reference cases are key to “teach” any deep learning machine to properly screen for DR, creating those standards is time-consuming. The researchers were happy to find using just a small subset of carefully adjudicated cases could make a huge difference in the algorithm’s performance.

measuring elevated ICP, and this study “adds further evidence for the sensitivity and specificity of ultrasonography for this purpose,” according an editor’s note from Andrew G. Lee, MD, of Houston Methodist Eye Associates in Houston, Texas.

Dr. Lee already uses the diagnostic tool in his practice to: differentiate difficult cases of pseudopapilledema vs. papilledema, follow patients with papilledema with residual post-treatment disc changes, evaluate those whose papilledema may no longer manifest ophthalmoscopically and patients with cerebrospinal fluid shunts and follow patients who refuse or cannot undergo a standard LP for measuring ICP.2

Because of the strong correlation between ONSD and ICP values, and because the ICP decreased as the dilated ONSDs decreased, the researchers believe “ultrasonographic ONSD measurements may be a useful, noninvasive tool for dynamically evaluating ICP.”2

“It is exciting to see that other measurements, such as ultrasound assessments of optic nerve sheath diameter, potentially offer easier, less invasive ways to gain an understanding of a patient’s ICP,” says Dr. Lighthizer. “Certainly, future research will only help to clarify this even more.”

Clinicians in the United Kingdom recently reported positive outcomes after treating two patients with severe age-related macular degeneration (AMD) by implanting an engineered retinal pigment epithelium (RPE) patch made of a fully differentiated, human embryonic stem cell (hESC)–derived RPE monolayer on a coated, synthetic basement membrane.1

“Until now, stem cell research for retinal disorders has primarily involved the implantation of a suspension of retinal pigment epithelium cells,” says Richard Trevino, OD, director of Residency Programs at the Rosenberg School of Optometry, University of the Incarnate Word. “The use of a patch is believed to improve the viability of the implanted cells, and the results of this study seem to support that notion.”

The researchers documented the survival of the implant with biomicroscopy and optical coherence tomography, and noted the patients gained significant visual acuity over 12 months of follow up.1

“The researchers have regulatory approval to perform this surgery on 10 patients, but they have chosen to report on the 12-month follow-up of their first two cases because the results have been so favorable,” says Dr. Trevino. “Both subjects experienced a dramatic improvement in vision (five to six lines on the ETDRS chart) following the procedure despite neither patient receiving anti-VEGF treatment.”

The Phase 1 trial was designed to first investigate the safety of the treatment, and results showed no tumors caused by residual undifferentiated embryonic stem cells spreading from the patch. Serious complications following the procedure were rare, the most serious being a retinal detachment associated with proliferative vitreoretinopathy (PVR), according to Dr. Trevino. “The researchers admit that the implanted RPE cells may have contributed to the development of PVR. Only by performing the procedure on more patients will the true risk be assessed.”

The researchers have a long way to go before proving the patch’s full safety and efficacy, however. For one, “there is no control group, so it is unclear how the vision recovery experienced following this intervention compares to the vision recovery that would occur following standard therapy for this disorder (anti-VEGF therapy),” Dr. Trevino adds. “But the prospect of having another treatment for this disorder that has the potential to improve vision is very exciting.”


OCT Reveals Ocular Changes in Astronauts
Researchers recently found that astronauts have disc edema-like changes in eye structure after returning to Earth from the International Space Station (ISS).

The study looked at morphological changes in the optic nerve head and surrounding tissues in 15 astronauts after they completed a roughly six-month mission aboard the ISS. The results were compared with 43 healthy control patients with no history of exposure to microgravity in space. After analyzing optical coherence tomography (OCT) data for the astronauts collected before and after the mission, the researchers found three major changes in eye structure.

Before the flight, the astronauts presented with recessed Bruch’s membrane openings compared with the healthy controls. After the mission, their membrane openings were deepened. Additionally, the astronauts showed a noteworthy increase of total retinal thickness near the optic nerve head rim margin, and the total number of eyes with choroidal folds increased.

While determining an exact cause of these changes was not within the study’s scope, the results do point to long-term exposure to microgravity and space travel as a possibility. Beyond that, the researchers believe the algorithms and methods used to determine structural changes could be helpful in both future space travel studies and those here on Earth.

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Scientists Discover a New ‘Organ,’ Label it “the Interstitium”

Using probe-based confocal laser endomicroscopy (pCLE), researchers can now view living tissue at the microscopic level—leading to the recent discovery of fluid-filled spaces in the body’s connective tissue. In a recently published report, investigators are calling the interconnected, fluid-filled spaces supported by a lattice of thick collagen “bundles” a new ‘organ,’ although further research and a consensus is necessary before the label can become official.1

Previous research used standard microscopic slides to explore this tissue layer, which, when viewed on the slides, appeared to be “densely-packed barrier-like walls of collagen.”2 pCLE reveals a failing of this traditional scientific method, as the preparation process collapsed the fluid-filled spaces and hid them from examination for decades, the researchers said in the report. By viewing the live tissue, Neil Theise, MD, a professor of pathology at New York University Langone School of Medicine, and colleagues found not a dense structure, but an “open, fluid-filled highway,” that transports “interstitial” fluid, including lymph and drains into the lymphatic system.1

Because the purported new ‘organ’ is in connective tissues all over the body, the researchers hypothesize several functions—and possible dysfunctions. For one, they note the interstitium may act as a “shock absorber” for other organs subject to “cycles of compression and distension.”2 And because it seems integral to lymph movement throughout the body, it may also be “a potential conduit for movement of injurious agents, pro-fibrogenic signaling molecules, and tumor cells,” according to the study. “This raises the possibility that direct sampling of the interstitial fluid could be a diagnostic tool.”

This finding, while potentially ground-breaking, raises more questions than it answers. However, it “allows us ask all kinds of questions we didn’t even know to ask beforehand,” said Michael Nathanson, MD, chief of the digestive diseases section at Yale University School of Medicine, who was not involved in the work but observed the network of dark fibers in a 2011 study of his own.1


Rare Corneal Dystrophy Gene ID’ed

Genome sequencing has lead researchers to identify the root cause of posterior polymorphous corneal dystrophy (PPCD), a rare autosomal-dominant form of corneal dystrophy that affects the corneal endothelium, according to a new study published in the March edition of the American Journal of Human Genetics.1 Investigators from University College London’s Institute of Ophthalmology and Moorfields Eye Hospital London have nailed down the precise variation to the DNA—located on a gene called GRHL2—that leads to dysfunction in the endothelial barrier and, ultimately, PPCD.1

Using data from a large family of Czech origin, the researchers mapped a locus for an autosomal-dominant corneal endothelial dystrophy. The whole-genome sequencing identified a unique variant that causes the gene to be expressed inappropriately in corneal endothelial cells. “We are delighted that the results from this study led to the discovery of a new genetic cause of PPCD. This will be the foundation for further studies to understand even more about the biological processes leading to corneal dystrophies and to developing new treatments,” Neil Meemaduma, a research manager with Fight for Sight—which assisted in funding the research—said in a statement on the organization’s website.2


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

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

Please see brief summary of Prescribing Information on adjacent page.

References:

* Fingertip Formulary data 2017

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

Lotemax (loteprednol etabonate ophthalmic gel) 0.5%
Rx only
Initial Rx Approval: 1998

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

DOSEAGE AND ADMINISTRATION
Invert closed bottle and shake once to fill tip before instilling drops.
Apply one to two drops of LOTEMAX into the conjunctival sac of the affected eye four times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

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

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

Cataracts
Use of corticosteroids may result in posterior subcapsular cataract formation.

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

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

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

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

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

ADVERSE REACTIONS
Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera.
The most common adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%), and foreign body sensation (2%).

USE IN SPECIFIC POPULATIONS
Pregnancy
Teratogenic Effects
Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (35 times the maximum daily clinical dose), a dose which caused no maternal toxicity. The no-observed-effect-level (NOEL) for these effects was 0.5 mg/kg/day (6 times the maximum daily clinical dose). Oral treatment of rats during organogenesis resulted in teratogenicity (absent innominate artery at ≥5 mg/kg/day doses, and cleft palate and umbilical hernia at ≥50 mg/kg/day) and embryotoxicity (increased post-implantation losses at 100 mg/kg/day and decreased fetal body weight and skeletal ossification with ≥50 mg/kg/day). Treatment of rats with 0.5 mg/kg/day (6 times the maximum clinical dose) during organogenesis did not result in any reproductive toxicity. Loteprednol etabonate was maternally toxic (significantly reduced body weight gain during treatment) when administered to pregnant rats during organogenesis at doses of ≥5 mg/kg/day.
Oral exposure of female rats to 50 mg/kg/day of loteprednol etabonate from the start of the fetal period through the end of lactation, a maternally toxic treatment regimen (significantly decreased body weight gain), gave rise to decreased growth and survival, and retarded development in the offspring during lactation; the NOEL for these effects was 5 mg/kg/day. Loteprednol etabonate had no effect on the duration of gestation or parturition when administered orally to pregnant rats at doses up to 50 mg/kg/day during the fetal period.

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

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

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

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

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

PATIENT COUNSELING INFORMATION
Administration
Invert closed bottle and shake once to fill tip before instilling drops.

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

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

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

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Proceed With Caution: Low Vision and Driving
Here’s how you can navigate the complex interplay between DMV standards and the needs of your patients who are visually impaired. By Mark E. Wilkinson, OD, and Khadija S. Shahid, OD, MPH

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Sizing Up Anti-inflammatories in Dry Eye Disease
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ANDREW S. GURWOOD, OD
The February 2018 cover story, “How the Diploma Deluge is Reshaping Optometry,” presented comments from educators and data from the Association of Schools and Colleges in Optometry (ASCO) to look at the impact of optometry’s expanded educational footprint. The feature generated criticism, praise and suggestions, some of which is addressed in the following letters and our reply.

Behind the Drive
Kudos for your well researched and appreciated article. I believe one cannot have a “deluge” of graduates without producing a surplus of optometrists, and that this surplus was self-induced and is counterproductive to patient care. This surplus was first predicted in a 1995 Rand study and again by an Abt. Associates study in 2000.1,2 But both underestimated future surpluses because graduates have since increased about 40%.

In 2011, the Lewin Group—with AOA-appointed advisors—was commissioned to survey how optometrists practiced and to estimate the future supply of, and demand for, eye care. Its 2012 survey found a 32% optometry surplus and its supply/demand model in 2014 predicted greater future optometry surpluses but increasing ophthalmology shortages.3 However, Lewin’s findings stated, “There would be an adequate supply of eye care providers in the future,” which was egregiously misleading. Lewin buried the optometry surplus within the surplus of “eye care providers” and characterized it as “an adequate supply of eye care providers.”

In 2014, Lewin next developed optometry and ophthalmology supply/demand projections and found future surpluses of optometrists and shortages of ophthalmologists. These initial projections assumed no surplus of optometrists existed in 2012 despite the findings of its own national survey of a 32% “excess capacity.”

The group then constructed a complex model that did include the 2012 optometry “excess capacity,” but increased future eye care demands due to the Affordable Care Act, the growing diabetes rate and child health insurance plans. In this “unified eye care market,” optometrists and ophthalmologists were assumed interchangeable. The final model’s assumptions claimed:

1. No increase in future ophthalmologists or their productivity.
2. Optometry “excess capacities” will “fill” shortages of ophthalmologists.
3. Optometrists and ophthalmologists have identical scopes of care and considered interchangeable generic “eye care providers.”
4. A rate of 1.36 optometrists provide the care equivalent of one ophthalmologist.

These were, of course, implausible assumptions.

The chief impetus for the “diploma deluge” were Bureau of Labor Statistics reports claiming high demand for optometrists that led the media to report optometry was “hot.” But the primary source for those forecasts were rosy projections supplied by the AOA leadership and bitterly resisted, over the years, by some staff officers.

Today’s annual graduation rate of 1,900 will eventually produce a workforce of 76,000 practicing optometrists, a density of optometrists per 1,000 nearly twice today’s. It has been too easy, for too long, for schools to meet Accreditation Council on Optometric Education (ACOE) accreditation standards, which are far less robust than medical and dental schools due to a lack of quantitative standards for required student contacts by types/numbers during training.

—Kenneth J. Myers, PhD, OD President, American Board of Certification in Medical Optometry


The Whole Truth
Your recent article identifies a subject worthy of discussion by the profession in looking to plan for the future. Unfortunately, the article appears to be something written more for a deadline than for a serious consideration of all the aspects. The effect of mandatory board passage for graduation and its effect on graduation rate was not even mentioned, nor was the recalibration of the Optometric Admission Test (OAT) during the years across which the author compares scores. While footnotes are used to imply some vigorous research, all the data is not given. For classes that matriculated in
2017, the University of the Incarnate Word Rosenberg School of Optometry had an entering class GPA that was higher than four of the six oldest institutions listed.

The entire article must be viewed with suspicion as it shows the Arizona College of Optometry to have been established in 2017 and yet provides its pass rates on licensing examinations. Once you realize the magnitude of that error you begin to question everything else the article asserts.

—Timothy A. Wingert, OD
Dean, University of the Incarnate Word Rosenberg School of Optometry

Keeping Us Honest

We thank the above readers and others who reached out to comment on the article. Several readers pointed out that Midwestern University’s Arizona College of Optometry’s first class was in 2009, not 2017. We regret the error. The online version has been corrected.

Second, Table 3 of our article compared the OAT scores of matriculating students from 2008 with those from 2017. Some readers pointed out, correctly, that the scoring was modified in 2009. The test is graded on a scale of 200 to 400. In 2009, adjustments were made to reduce the average score—which had crept up to 320—to 300. According to Association of Schools and Colleges of Optometry President David Damari, OD, the changes included “changing the way raw scores (number of items answered correctly) were converted to the standard scores reported.” He went on to explain that, “As the test ages—so to speak—the average score goes up a little bit at a time, for various reasons, especially in reading comprehension.”

Every year, administrators review the test to determine whether it’s still a predictive indicator for schools. Before the 2009 test, they decided it was not. However, the adjustment was “not really a change in the test, it’s a change in how many items the student needs to get correct in order to get to 300,” Dr. Damari explains. “It’s an adjustment, not a major rescaling.” There’s an ebb and flow to all testing, he suggests. Once a test starts to mature, average scores go up and that’s how administrators know it’s time to recalibrate the test. In fact, educators are looking again to adjust the scoring. Dr. Damari notes that “2008 was just before one calibration and 2017 is probably going to end up being right before another recalibration. If anything, that’s an incredibly fair comparison.”

The article also makes use of recently released statistics from the National Board of Examiners in Optometry revealing the percentage of graduates who pass the boards. However, we did not discuss a caveat to those numbers; that some schools require students to pass part one or parts one and two of the boards (nobody requires passing part three) before it allows them to graduate. Although it does impact what that ultimate board pass rate is, this really raises a philosophical question. If optometry schools exist to educate future optometrists, should they do all they can to ensure their students’ success—and does “doing all they can” include guaranteeing they can pass the boards?

Look at Southern College of Optometry (SCO), which started requiring the national boards in 1990, according to Lisa Wade, OD, director at SCO’s Hayes Center for Practice Excellence. It suffered from low pass rates before that and its reputation was negatively impacted. When the school started requiring board passage, that changed drastically; in fact, it influenced other schools in the South to embrace requiring board passage. A rising tide lifts all boats.

Others wonder whether requiring students to pass a test that the institution itself does not compose itself amounts to a violation of the institution’s autonomy. After all, the school issues a degree, not a license.

As our story noted, the numbers don’t tell all. Optometry schools’ standards clearly exist on a spectrum and, although they can be measured in a variety of ways, they can never really be ranked. There will always be some incongruity or unique circumstance. Although we can quantify performance and use data to predict success, we cannot quantify human beings and, ultimately, that’s what optometric education must focus on: training actual human beings from a variety of backgrounds, to become skilled, dedicated, passionate doctors.

If it’s to thrive, optometric education must serve two masters, one from the idealistic world of academia and one from the sober, hard-nosed world of capitalism. And while doing that may require schools to compromise from time to time, the educational system’s influence over the future of optometry is not absolute. The real future is in the hands of the students themselves, who—we hope our article demonstrated—strive to grow the profession while preserving the legacy of their predecessors.

—Bill Kekevian
Senior Editor,
Review of Optometry
Is hydrogel good enough for most 1-day lens wearers?

Dr. Rosinski: I strongly believe that silicone hydrogel 1-day lenses provide better long-term eye health for my patients than hydrogel 1-day lenses. Furthermore, 91% of my colleagues agree according to recent research. This is in stark contrast to how many 1-day patients they fit in silicone hydrogel—they fit only 30% of 1-day patients in silicone hydrogel.

This begs the question: Why don’t doctors routinely fit 1-day wearers in the same material they prefer for frequent replacement wear? Unfortunately, the answer to this question remains a mystery, particularly in light of startling new research, which demonstrates that doctors’ lens prescribing patterns lag far behind their beliefs about what’s best for patients.

We spoke with three optometrists for insight on this disconnect between beliefs and behavior. In the interviews that follow, these 1-day silicone hydrogel advocates explain why they are committed to this material for 1-day fits and share advice on how to always recommend the lens you trust the most.

Dr. Frogozo: I agree with 92% of ECPs who say silicone hydrogel 1-day lenses are the best choice to safeguard patients’ eye health related to contact lens wear. This same study shows that 92% likewise believe that silicone hydrogel 1-day lenses provide the best benefits to their patients. In terms of my own personal beliefs and how that guides how I practice, I’m concerned that the hydrogel wearer will develop hypoxia and eventually drop out of lens wear. If the answer is no, then why would you deprive the 1-day wearer of this same opportunity?

What meaningful advantages does silicone hydrogel have in a 1-day lens?

Dr. Frogozo: I agree with 92% of ECPs who say silicone hydrogel 1-day lenses are the best choice to safeguard patients’ eye health related to contact lens wear. This same study shows that 92% likewise believe that silicone hydrogel 1-day lenses provide the best benefits to their patients. In terms of my own personal beliefs and how that guides how I practice, I’m concerned that the hydrogel wearer will develop hypoxia and eventually drop out of lens wear. It’s a familiar story that we’ve seen play out when hydrogel was the go-to lens in frequent replacement.

Dr. Huisman: Doctors want health, but patients care about comfort. Silicone hydrogel 1-day lenses offer both.
Silicone hydrogel 1-day lenses are great for my younger patients because they’re so easy to insert and remove—plus they’re extremely comfortable. My patients aren’t rushing home from work so they can take out their lenses. That’s a meaningful, practical advantage.

Dr. Rosinski: The number one advantage is ocular health. When the clariti 1 day family of lenses was introduced, it was a no-brainer for me to switch my 1-day patients. Silicone hydrogel creates a highly “breathable” lens that promotes whiter, brighter** eyes.

How do you get patients on board with your decision to switch to a silicone hydrogel material—especially if they seem happy with their current hydrogels?

Dr. Huisman: Patients are confident in your recommendation when you cite a specific reason for change. I educate them on the benefits and advantages of silicone hydrogel generally and for their case specifically. Quite often, my hydrogel patients have signs of neovascularization or hyperemia, so I take a photo of this and show it to the patient, explaining that this is the reason change is needed. In the event that the patient has no signs and is very happy with the current lenses, I shift the conversation focus around preventative care.

Dr. Frogozo: My practice is primarily referral-based, which means patients walk in wearing many different types of lenses. Despite this, I fit my sphere patients almost exclusively in 1-day silicone hydrogel lenses. And, as the toric parameters expand, I’m fitting more and more astigmatics in 1-day silicone hydrogel as well. Patients rarely object to my recommendation. I proactively educate my patients on the need for oxygen in lens wear and advise them that silicone hydrogel is healthiest.

Does the lens trial play a significant role in convincing 1-day wearers about the benefits of silicone hydrogel?

Dr. Frogozo: In my practice, the conversation is usually more powerful than the trial. A strong recommendation from a doctor is all most patients need to hear. If you believe silicone hydrogel is a superior material, the patient will likely believe it too. However, if you have a patient who is already wearing a 1-day lens in a hydrogel material and you want to switch to silicone hydrogel, the lens trial can be a tipping point. In these cases, I tell the patient that their current lenses are based on older technology, so I’m offering them an opportunity to trial something newer and healthier.

Dr. Huisman: I agree. Trials are great but the conversation has to precede it. I don’t want patients to think I’m making a change for change’s sake. Eighty-two percent of ECPs believe that silicone hydrogel should be the standard of care for 1-day contact lens patients and 87% say silicone hydrogel material should be the first choice of material for daily disposable lenses. When patients hear that from us, they’re more likely to approach the lens trial with enthusiasm.

Dr. Rosinski: Triaing silicone hydrogel 1-day lenses also helps strengthen our relationships with patients. First, we make the recommendation and educate the patient on the benefits. Next, they take the lens for a test drive. Finally, when they return satisfied with the comfort and vision of their new lenses, they’re more confident than ever in our knowledge and our commitment to providing the best possible care.

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* With higher oxygen permeability than hydrogel materials, silicone hydrogel contact lenses minimize or eliminate hypoxia-related signs and symptoms during lens wear.

** Data on file; clariti® 1 day offers whiter eyes than 1-DAY ACUVUE® MOIST®.
**Just Say Yes to (Some) Drugs**

Don’t shy away from prescribing the right medications when your patients need them. **By Paul M. Karpecki, OD, Chief Clinical Editor**

Optometrists manage more anterior segment disease than anyone, and it’s critical that we have the pharma knowledge to treat these patients appropriately. This month’s annual pharmaceutical issue provides detailed advice to elevate your practice so you can give patients much-needed relieve and recovery. I’d like to preface this article series with a few thoughts from my perspective as both a clinician and an educator.

**Antibiotics**

Just because we haven’t seen a new antibiotic in a few years doesn’t mean we should get complacent in drug selection. Drug resistance, while steady, is incredibly high.1 The most recent ARMOR study—a yearly review of over 3,000 ocular isolates in the United States—found methicillin-resistant *Staphylococcus* is resistant to 30% to 40% of antibiotics; for coagulase-negative *Staph.* it’s nearly 50%. Because we don’t typically culture conjunctivitis and it takes so long to get a culture for an infectious keratitis, we have to be knowledgeable and judicious when treating empirically.1 Most bacteria are susceptible to newer agents such as Besivance (besifloxacin, Bausch + Lomb).1

**Dry Eye Drugs**

Although artificial tears continue to advance with a number of new and increasingly effective options, they still play a palliative role, with little value in addressing inflammation. Both Restasis (cyclosporine, Allergan) and Xiidra (lifitegrast, Shire) have made significant contributions to treating the underlying inflammation of dry eye disease.2 Xiidra may bring symptom improvement in as little as two weeks, and some studies of Restasis show improvement at one month.3

**Topical Steroids**

Don’t fear these drugs. For superficial punctate keratitis (SPK), few treatments are more effective.4 There are risks to withholding steroids when managing inflammatory diseases such as corneal neovascularization, synechiae, persistent SPK, progressive dry eye, corneal haze and scarring.

At the same time, don’t be cavalier with steroids. Although the only absolute contraindication is epithelial herpetic disease, there are times when you should apply caution in their use. For example, be wary of steroids when treating an early infectious keratitis (especially without a confirmed diagnosis), an abrasion or when using a bandage contact lens. There still may be a role for steroids in such cases, but exercise greater caution. Always check intraocular pressure within three to five weeks for any patient on a corticosteroid. To help ensure the patient returns for the check, I often provide no refills on steroid-containing drops in new patients.

**Systemic Medications**

An optometrist without access to systemic meds is in a difficult position to effectively care for patients. Many cases necessitate systemic therapy, and in today’s healthcare system, a referral before treatment leaves the patient vulnerable to significant morbidity. Soft tissue infections such as dacryocystitis, hordeola, canaliculitis and preseptal cellulitis are just one category that comes to mind (orbital cellulitis requires referral to an emergency room for intravenous antibiotics).

Systemic medications are also required to fully control chronic conditions such as ocular rosacea, multiple or repeat hordeola and chalazia, and chronic ocular surface disease. Severe allergies, dermatological disorders and immune responses may require a short course of oral prednisone. One cannot manage most cases of ocular herpes simplex virus without oral antivirals, and even over-the-counter oral nonsteriodals can greatly aid a patient with a corneal abrasion.

It’s nearly impossible to effectively manage ocular disease without occasionally having to reach into the bag for an oral medication. Give this hard-won legal right the recognition—and use—it deserves. ■

**Note:** Dr. Karpecki is a consultant for many companies discussed here.

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The Extinction of the Ink Pen

…and other things. What’s in your pile of office junk you never use anymore?

By Montgomery Vickers, OD

Now that we are all techno-post-pseudo-Millennials, I cannot find an ink pen anywhere. That’s right, people from the South call it an ink pen. If you do not call it an ink pen, you are either a transplant from the North or pretentious. Either way is fine by me.

I do have a couple of ink pens that survived the Great Ink Pen Purge created by the Feds in an attempt to protect patient records by making it easier for any basement-dwelling hacker get all of them with the stroke of a key. Before, they had to break into my office and carry them out, which is way more effort than any hacker would expend.

There are so many other things that have become extinct in the new frontier of optometry:

Pen lights. There may be a few holdouts who test every pupil every time with a penlight, but most of us know if anything’s messed up by the time we hit them with a slit lamp.

PD ruler. Why bother? ODs are always griping about not being paid for giving patients PDs. Folks, anything on the patient’s chart is their information, not yours. If you like to fight over releasing PDs, just quit measuring them. You can’t give them what you don’t have.

Trifocals. Now and then a patient wanders in who loves his version of “start, step and stumble,” but most aren’t very excited to have any lines on their glasses, much less two.

Double segs. Another victim of the tech age where mechanics and electricians plug a gizmo in to reset everything instead of actually doing anything real. Have you noticed how soft their hands are these days?

Quarterly soft contact lenses. What? You still have some patients disposing of their lenses once every 90 days? That’s just two 90 packs of dailies lasting 22 years.

Bandage contact lenses. You remember, the ones that were actually studied and approved by the FDA for this use only. I’ll bet I know what you really use.

Paper Snellen charts. Ok, these are not extinct, but 100% of them are at the pediatrician’s office where your patient’s kid “just had an eye exam.” To be fair, they don’t need to see the pediatrician for a physical either because, well, they seemed healthy enough when we saw them.

Last-minute cancellations. I just stuck this in here because I knew you’d laugh.

Shirt and tie. The last time I saw this in the office was me passing by a mirror. The staff and the patients absolutely loved it, so try it.

Being on time. Now that we have 200 separate pretesting instruments, we might see our 9am by 9pm, but at least we’ll know the exact wavelength color of their irides and how long they slept.

Confirmation calls. When patients had one phone in the house, two rings garnered an answer. Now everyone has a phone glued to their ear, but no one answers.

Lunchtime office hours. I blame myself for this one. I started closing the office for lunch in 2006. This attracted patients like bears to honey, and they all came to pick up stuff as soon as I locked the door.

Elevator music. Once a staple of every reception area, the soothing orchestral versions of the Beatles’ White Album has given way to big TVs explaining progressive spectacle lenses to people wearing ear buds listening to orchestral versions of the Beatles’ White Album. It’s OK, time marches on. The next generation will look back fondly on extinct stuff, too. Like ODs, if we don’t watch out.
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A patient called me Sunday evening after being on the losing end of a bar brawl Friday night. When seen in the ER on Saturday, a CT scan revealed an orbital floor fracture. He was told to see an eye doctor. How do you examine this swollen eye and how do you know when surgery is in order?

“First of all, don’t panic,” says James Milite, MD, an oculoplastic surgeon from Omni Eye Services in Iselin, NJ. In many cases, the impact of the injury will make the eye look worse than it is. “Take a step back and take your time doing a thorough exam,” he says.

A comprehensive history is your next step. You’ll want to know how the injury happened and what struck the eye. “Ask if there was any loss of consciousness at the scene of the incident; also find out if there have been any flashes, floaters or double vision. Check their lateral gaze and downgaze, ability to open and close the mouth, and look for evidence of decreased sensitivity in the distribution of the maxillary division of the trigeminal nerve,” says Dr. Milite.

After ruling out head trauma and foreign bodies, conduct your typical eye exam—acuities, confrontation fields, pupils and a careful anterior and posterior segment exam. “Make sure the retinal periphery is OK, and look out for corneal abrasions, hyphema, iridodialysis, uveitis, cataract, vitreous hemorrhage and retinal detachment,” adds Dr. Milite. Also, examine the orbit. “This can be the trickiest part due to the eye swelling,” he says. “Observe if the globe is sunken or displaced downward away from the midline. This might indicate a fracture of the cheek bone or cheek complex,” he says. Left untreated, it could permanently pull the lower lid down or flatten the malar region.

**To Refer or Not to Refer?**

After examination, you should have a sense of whether or not you have a surgical candidate on your hands, says Dr. Milite. “You’re looking for either or both of the following: entrapment causing restriction of extraocular muscles (EOMs) and diplopia in functional fields, and enophthalmos greater than 2mm.” However, the degree of periorbital edema often makes assessment of globe position and motility difficult.

“It’s also important to look out for signs of proptosis of the eye,” adds Dr. Milite. “This is an indicator of a retrobulbar hemorrhage.” Though uncommon, it necessitates an urgent referral to an oculoplastic specialist. All these patients require CT imaging if not already obtained prior to your exam.

Typically, if the primary injury is an orbital floor fracture, you won’t need to send for emergency surgery. However, there are a few extenuating circumstances. Young kids, for example, can more easily present with entrapment and require immediate surgery. “I usually see this in kids under age 13,” adds Dr. Milite, “as their bones are pliable and don’t crack—they bend and snap back, causing tight EOM entrapments.” This can lead to nausea, vomiting and a decrease in blood pressure.

Since the fracture snaps closed almost like a trap door, it might not seem significant on the scan, but these signs represent a potentially urgent case and require a rapid referral to an oculoplastic surgeon. Failure to refer could result in irreparable damage to the tightly entrapped muscle.

When choosing to treat these cases with antibiotics or steroids, a few simple rules will suffice.

“Antibiotics are given due to theoretical risk of infection, so a five-day course of prophylactic antibiotic can’t hurt,” says Dr. Milite. “If you have a patient with a moderate-size fracture and excessive swelling, an oral steroid will cut the window down for edema resolution to unmask the development of enophthalmos and help determine the need for repair.”

Consult a specialist if in doubt. In this case, referral resulted in surgery later that week. “Describe the fracture, explain motility issues and record everything,” Dr. Milite says. “If it’s a surgical candidate, ask what you can do to prepare the patient before sending them along.”

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**Taking the Floor on Fractures**

When a mishap puts an orbital floor fracture in your chair, know how to look past the gore and devise a plan. **Edited by Paul C. Ajamian, OD**

A 25-year-old male with orbital floor fracture raises questions about the need for reconstructive surgery.
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A Significant Proposal

Want better clinical outcomes? Demand better study design, experts say.

Lies, they say, come in three varieties: lies, damn lies and statistics. Cynical though it may sound, that rings true for anyone whose profession revolves around numbers. Medicine, of course, is rife with statistics: the incidence and prevalence of diseases, the efficacy of therapies, optimal dosing regimens, adverse events, risk factors, survival rates—all are expressed in numeric terms, sometimes to a hundredth of a decimal point. Even quality of life, by its very name a qualitative measure, gets quantified in health care.

Do doctors rely too much on the statistics in medical literature? The people in charge of medicine and the people in charge of statistics think so. An editorial in the Journal of the American Medical Association, echoing the American Statistical Association, argues that probabilities (expressed by the P value) “are misinterpreted, overtrusted and misused.”1

Actually, it’s fairer to say that the problem isn’t so much reliance on statistics but rather the way in which we ascribe validity to medical ‘facts’ that are expressed numerically. The two groups seek to tighten that up by advocating for “lowering the routine P value threshold for claiming statistical significance from 0.05 to 0.005 for new discoveries.” That means the likelihood of a random (rather than causative) association in a study would drop from 5% to 0.5%, raising the bar for what gets called significant. This would “shift about one-third of the statistically significant results of past biomedical literature to the category of just ‘suggestive,’” the editorial states.

Poof, one-third of your ‘facts’ just went up in smoke.

It’s a scary proposition, but may be for the best. Such a move would cut a lot of noise out of the conversation. Too many studies using P values in the 0.05 range are quoted and promoted as gospel, in eye care and other disciplines. “Most claims supported with P values slightly below 0.05 are probably false (i.e., the claimed associations and treatment effects do not exist),” the editorial states. “Even among those claims that are true, few are worth acting on in medicine and health care.”

And yet, many of those claims are the lifeblood of medical practice today. Health care would be better off, says the editorial, “with fewer, larger, and more carefully conceived and designed studies with sufficient power to pass these more demanding thresholds.”

Of course, this would be no panacea. Those with a vested interest, most notably for-profit entities more interested in finding a marketing angle rather than the purity of truth, could just move the goalposts. “Selected study end points may become even less clinically relevant because it is easier to reach lower P values with weak surrogate end points than with hard clinical outcomes,” JAMA warns.

It’s not easy to think of scientific validity as a tunable instrument: turn the dial up and certainty goes down. But this proposal reminds us that our facts are only as ‘real’ as our methods—and motives—demand.

1. Ioannidis JP. The proposal to power P value thresholds to .005. JAMA. March 22, 2018 [Epub ahead of print]
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No Sneaking Around this Code

Modifier -59 should be a last resort. Here’s how to use it wisely.

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

Consider this scenario: A patient comes in for a glaucoma workup after being identified as a suspect during last week’s annual exam. During this follow-up office visit (9921X) you want to do: optical coherence tomography (OCT) optic nerve (92133), fundus photography (92250), pachymetry (76514) and threshold visual fields (92083).

The Temptation

You know fundus photography and OCT of the optic nerve are not allowed on the same date of service. However, you have heard certain modifiers, such as -59, can “override” these rules on the claim form so you can get paid for both services. So what should you do? Let’s take a look.

Where to Start

The rules that govern situations such as this one are the National Correct Coding Initiative (NCCI) edits, which are found in CMS’s NCCI Policy Manual For Medicare Services, published yearly and updated quarterly. These edits control the pairing of procedures on the same date of service.¹

Understanding these code pairing rules and their context is crucial, especially when applying a modifier that will override a claim pair denial. One of the most abused modifiers is -59.

CMS Definition

To properly use a modifier, you must know both the definition and its proper application. Modifier -59 is used to identify procedures/services, other than E/M services, that are not normally reported together, but are appropriate under the circumstances. Documentation must support a different session, different procedure or surgery, different site or organ system, separate incision/ excision, separate lesion or separate injury (or area of injury if extensive) not ordinarily encountered or performed on the same day by the same individual. Modifier -59 can be used appropriately in these situations (remember, the eye is considered a single anatomic structure and site):²,³

1. “Modifier -59 is used appropriately for different anatomic sites during the same encounter only when procedures which are not ordinarily performed or encountered on the same day are performed on different organs, or different anatomic regions, or in limited situations on different, non-contiguous lesions in different anatomic regions of the same organ.”³

2. “Another common use of modifier -59 is for surgical procedures, non-surgical therapeutic procedures or diagnostic procedures that are performed during different patient encounters on the same day and that cannot be described by one of the more specific NCCI-associated modifiers—i.e., 24, 25, 27, 57, 58, 78, 79 or 91. As noted in the CPT definition, modifier -59 should only be used if no other modifier more appropriately describes the relationship of the two procedure codes.”³

Limitations

Modifier -59 should not be appended to an E/M service. To report a separate E/M service with a non-E/M service performed on the same date, use modifier -25.⁴

You should never use modifier -59 to bypass a PTP edit unless you meet and document the proper criteria required by any NCCI-associated modifier. The use of modifier -59 does not require a different diagnosis for each HCPCS/CPT coded procedure; conversely, different diagnoses are not adequate criteria for use of modifier -59. The HCPCS/CPT codes remain bundled unless the procedures are performed at different anatomic sites or separate patient encounters.⁴

Misuse

Modifier -59 is used inappropriately if the basis for its use is simply that the narrative description of the two codes is different.

One of the common misuses of modifier -59 is related to the portion of the definition allowing its use to describe a “different procedure or surgery.” The code descriptors of the two codes of a code pair edit usually represent different procedures, even though they may be overlapping. The edit indicates that the two procedures should not
be reported together if performed at the same anatomic site and same patient encounter, as those procedures would not be considered “separate and distinct.”

However, if the two procedures are performed at separate anatomic sites or at separate patient encounters on the same date of service, modifier -59 may be appended to indicate that they are different procedures on that date of service.

-59 in Your Office
In ophthalmic practice, the most common combination with which modifier -59 is improperly used is CPT codes 92250 and 92133/92134. The NCCI manual specifically states: “Fundus photography (92250) and scanning ophthalmic computerized diagnostic imaging (e.g., 92133, 92134) are generally mutually exclusive of one another in that a provider would use one technique or the other to evaluate fundal disease.

As with most rules, a few exceptions exist, and there are a limited number of clinical conditions where both techniques are medically reasonable and necessary on the ipsilateral eye. In these situations, both CPT codes may be reported appending modifier -59 to CPT code 92250."

Those limited number of clinical conditions must meet the definition of medical necessity, i.e., patient harm could be a result if the two tests aren’t performed on the same date of service. This does not mean there is a wholesale acceptance of this code combination with modifier -59 just because the doctor thinks it’s appropriate or because it is convenient for either the doctor or the patient.

For our case example, using modifier -59 on the fundus photography code might get you paid, but it is not appropriate. You must first establish proper medical necessity for each procedure that you need to perform. Remember, modifier -59 is a last resort and should be used sparingly in clinical practice.

Send questions and comments to rescodingconnection@gmail.com.

Focus on Refraction

Fresnel Prism to the Rescue

Finding the perfect prism can take time and patience, but once it fits, it sticks.
By Marc B. Taub, OD, MS, and Paul Harris, OD

When we are called to see patients at a rehabilitation facility, and occasionally when patients are scheduled for vision therapy evaluations at Southern College of Optometry, we truly do not know what we are going to encounter. Brain injury cases range from visual field and acuity loss to visual inattention to vague complaints that something simply “feels off.” Of course, some patients are in such a state of decreased mental and physical capacity that they cannot respond in a traditional manner. Those are the most challenging, but can reap huge rewards for the patients in the long run.

Perhaps the most common complaint we encounter is double vision. This occurs for a multitude of reasons, including muscle and nerve injury, and can be quite severe. In these situations, the extent may vary depending on the gaze and head position. In the short term, we often use occlusion—either binasal or spot—to eliminate the double vision and evaluate the visual system weeks later as the visual, and cognitive and control systems come back online. For comitant diplopia, we often investigate the use of prism to attempt to restore single vision. The two cases presented here demonstrate how the use of temporary Fresnel prism can get your patients on the road to recovery quickly and effectively.

Case One

One morning, a 44-year-old male reported that, several days prior, he was pulling into a parking spot at work, and when backing in, he turned his head to get a better view and suddenly felt different. It turns out, he had a major aneurysm and did something to the blood vessels and supply to his brainstem. Ever since, he had double vision. When he presented, he was wearing an eye patch. Luckily, he was still operating at a high cognitive level, a crucial aspect when considering the use of Fresnel prism.

Visual acuity, confrontation fields, pupils and eye movements were all normal. His primary complaint was the double vision, which he described as “vertical.” Upon visual inspection, the eyes appeared aligned. Cover testing at distance and near revealed a six prism diopter right hypertropia. The magnitude was not changed by altering his head posture or gaze. This was confirmed subjectively and objectively via cover test with loose prism. Not all patients suffering from brain injury double vision can obtain single vision so soon after, so we considered this a good opportunity to use temporary prism. However, the higher the prism strength, the more the visual acuity is degraded. Some patients are not willing or are unable to adjust to the degraded acuity. As his acuity without glasses was excellent, tried frames with plano lenses we had handy.

These frames, fit binocularly with Fresnel prism, can be used to restore single vision to patients such as the one in our first case.
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Focus on

Refraction

The use of prism follows a pattern we have presented over and over in this column: less is typically more. Over our careers, we have both found that patients need less prism for fusion than measured on the cover test or even as measured with standard von Graefe or other methods. Giving less prism than measured also serves a therapeutic role in leaving degrees of freedom in which the system can operate.

With two base down over the right eye and two base up over the left eye, the patient reported seeing single vision. We informed him that these were full-time glasses and confirmed with the rehabilitation team that there were no visual restrictions. We checked back several days later and the patient was still smiling. The rehab team reported good progress on balance and hand-eye coordination activities. He will follow up with the vision rehab service at the college in several weeks.

You may be wondering why we chose to split the prism. With a patient who sees well, even though he comprehends the potential for degraded vision, we tend to split the prism to even out the acuity decrease. Essentially, this brings down both eyes equally instead of penalizing one; lessening the visual acuity in one eye would throw off the binocular system we are working so hard to rehabilitate. If one eye has decreased acuity for whatever reason, choosing not to split the prism may be a better option. In that instance, we tend to put the full amount of the prism or the bulk of it in front of the eye with the worse visual acuity or was turned all the time.

Case Two

Immediately following the previous patient, a 63-year-old male who suffered a head injury after a fall presented to the clinic. The patient was in good spirits and was joking around with me, which is always a good sign. Unlike the patient described in case one, he was not wearing a patch, indicating that either the magnitude of the diplopia was especially high or fairly low. If the magnitude of the diplopia is large, one of the images may be out of the patient’s line of sight. On the other hand, if the magnitude of the diplopia is small, there is potential that the patient is indeed fusing some of the time. In this case, it was fortunately the latter. As with the first case, the patient was refractively normal. The double vision was intermittent, occurring 50% of the time without a recognizable pattern on the part of the patient or therapists. Cover testing showed a three diopter left hyperphoria.

We grabbed our plano spectacles and Fresnel powers of one and two prism diopters from the kit. Subjectively, the patient reported good fusion with the lower power but felt more comfortable with the higher one. We allowed him to sit with the prism on and questioned him regarding the acuity difference. Since he was thrilled to not see double, he was not fazed by the slight acuity difference. We ended up with two prism diopters base down in the right eye on the plano spectacles and the patient was happy.

The therapists were ecstatic at the improvement in skills several days later. He too will be re-evaluated in several days. We are hoping to either reduce or eliminate the prism if symptoms abated.

Fresnel prisms can aid your patient care regardless of whether or not you treat patients with stroke or traumatic brain injury.

If you are considering grinding in prism, the temporary prism can help confirm the amount needed. In some cases, the use of the prism has a therapeutic effect and the amount of prism can be reduced over time. This is especially true of patients in vision therapy.

For some reason, optometrists seem afraid of prism. With the use of Fresnel prism, this fear should be eliminated.

These frames are similar to the ones on page 28, but are only fit monocularly with Fresnel prism. The other lens is plano.
Sleeping in contact lenses that aren’t approved for such use has been associated with discomfort, as well as with more serious eye health problems. Even so, a survey of contact lens wearers found that 64% of those who sleep in their lenses for at least a week at a time are not wearing extended/overnight use lenses. A separate study found that about one-third of contact lens patients report wearing their lenses to bed.

In our practice, many patients want extended-wear contact lenses because of lifestyle demands, such as new moms, shift workers and on-call physicians and nurses. One of my patients—a law student—spends endless hours studying in the school library and sleeps for a few hours at a time, taking naps when his schedule allows. He recently complained of lens discomfort and frequent redness, and his history revealed that he often wore 2-week replacement lenses continuously and overnight for a week at a time (for which his lenses were not approved). I explained that sleeping in lenses like his could cause the discomfort he was describing, and that it also increased his risk of ulcerative keratitis, a serious condition that can lead to loss of vision, which is usually caused by a microbial infection. I switched him to AIR OPTIX® NIGHT & DAY® AQUA contact lenses because, unlike some overnight lenses that are available, I find that AIR OPTIX® NIGHT & DAY® AQUA contact lenses provide continuous comfort, and are specifically designed for continuous wear, even while napping and sleeping. The lenses are FDA approved for daily wear and up to 30 nights of continuous wear.* AIR OPTIX® NIGHT & DAY® AQUA contact lenses are made from lotrafilcon A, a lens material with an established safety profile including a low rate of microbial keratitis. In a study of more than 6,000 lotrafilcon A wearers, only 0.18% of patients were diagnosed with presumed microbial keratitis, and less than 0.04% with presumed microbial keratitis and a loss of visual acuity.

My patients’ eye health is important to me and my practice, but I also want to make sure that overnight lens wearers remain comfortable throughout the full recommended wearing period. AIR OPTIX® NIGHT & DAY® AQUA contact lenses feature SmartShield® Technology, a proprietary surface technology that protects against the inherent hydrophobic properties of silicone.* SmartShield® Technology acts as a protective barrier, limiting exposure of silicone on the surface of AIR OPTIX® contact lenses, and thereby helping to maintain excellent surface wettability and deposit resistance. With this technology, my patients can experience clear vision and consistent comfort from the first day they put on their lenses, waking up to these benefits for up to 30 days thereafter.

Since the health of my patients is very important to me, I’m concerned that so many may be inappropriately wearing their contact lenses overnight. As their eye doctor, my job is to educate them and provide the best lenses to meet their needs. Thanks to the many benefits of AIR OPTIX® NIGHT & DAY® AQUA contact lenses, I can provide comfort to my overnight lens-wearing patients, ensuring that they see, look and feel their best.

References

*Extended wear for up to 30 continuous nights, as prescribed by an eye care practitioner
D iabetic macular edema (DME) contributes significantly to vision loss in diabetes patients and is the most common cause of vision loss among young adults. Previously, these patients were subjected to focal or grid laser photocoagulation. Although this method was effective in reducing vision loss by 50%, it was ineffective in restoring lost vision. All patients who participated in the original Early Treatment Diabetic Retinopathy Study (ETDRS) had visual acuity of 20/40 or worse.1

Fortunately, management of DME has improved over the last decade and has increased the potential of visual restoration with the approval of anti-VEGF compounds Lucentis (ranibizumab, Genentech) and Eylea (aflibercept, Regeneron), and the off-label use of Avastin (bevacizumab, Genentech). Given the role inflammation plays in the development of DME, steroids have also become an effective option in DME management. Ozurdex (dexamethasone, Allergan) is a time-release intravitreal implant that emits a low dose of dexamethasone over 90 to 120 days. Another implant, Iluvien (fluocinolone acetate, Alimera Sciences), the most recent therapy approved to treat DME, has an efficacy of up to 36 months. Focal or grid macular laser is also still used for clinically significant macular edema.

The ways we detect and define DME have evolved, too. Classification of DME is now based more on optical coherence tomography (OCT) images than funduscopy. The world of ophthalmology and retina in general is moving away from the classification of clinically significant macular edema (CSME), which primarily relied on funduscopy findings, and more towards spectral-domain OCT (SD-OCT) findings and retinal thickness. Now, DME is broken down into “center involved” (CI-DME) and “non-center involved” (NCI-DME) cases based on SD-OCT subfields.

These advances have given retina specialists and comanaging optometrists more options for management of DME—but also more room for doubt and debate regarding whether, when and how to intervene.

A Tale of Two Patients

By Dr. Haynie

Case 1. A 51-year-old white female presented for an evaluation with a six-month history of blurred vision in the right eye. Her medical history includes hypertension and 12 years of Type 2 diabetes. Her medication history included metformin and glyburide for diabetes, and lisinopril for hypertension. Her best-corrected visual acuity measured 20/40 in the right eye and 20/20 in the left eye. The anterior segment exam was unremarkable, and she was phakic. Dilated examination revealed moderate nonproliferative diabetic retinopathy (NPDR) with multiple dot-blot hemorrhages and central macular thickening. SD-OCT confirmed CI-DME (Figure 1).

She was treated with serial anti-VEGF injections, variously using bevacizumab and aflibercept. She responded well, achieving resolution of the edema and a recovery in vision to 20/20 with stable OCT images (Figure 2).

This case may seem straightforward, and the retinal referral was considered both necessary and unremarkable. Now let’s look at another case of DME and discuss how the referral and management may differ.

Hold ’em or Fold ’em?

Better treatment options for DME give us more flexibility, but also more responsibility. We need to use anti-VEGF prudently—if at all. By Diana Shechtman, OD, and Jay M. Haynie, OD

Figs. 1 and 2. This patient's center-involved DME (left) responded well to anti-VEGF (right).
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Case 2. This 68-year-old Native American female was referred for an evaluation of diabetic retinopathy and macular edema. She reported that her vision had been stable with no recent symptoms. Her medical history included Type 2 diabetes of 12 years’ duration, hypertension and cardiac arrhythmia. Medications included glargine and metformin for diabetes, and amlodipine, metoprolol and clopidogrel for hypertension. Entering best-corrected visual acuity measured 20/25 in each eye. Anterior segment evaluation revealed grade 2 nuclear cataracts in each eye. Dilated fundus examination revealed mild NPDR in each eye with a circinate ring of lipid in the temporal macula of the right eye (Figure 3). SD-OCT imaging revealed NCI-DME with a normal foveal contour and thickness.

After a discussion of the available treatment options, including serial anti-VEGF intravitreal injections, she elected to observe rather than treat. After three months, the NCI-DME resolved and her vision was maintained at 20/25 in the right eye (Figures 4 and 5).

How Our Practice Compares
By Dr. Shechtman

Given that I practice in a retina clinic in South Florida, we are inundated with cases similar to the ones described above. The question of whether to treat or observe has become increasingly controversial. DME is dynamic and, as Dr. Haynie points out in case 2, may have the potential for resolution without intervention. Hence, it is critical to educate the patient about the importance of tight glycemic control.

Yet, many cases will progress and, if untreated, result in decreased visual acuity. Thus, all five surgeons at my practice would agree with the management of case 1. In the presence of decreased visual acuity (i.e., <20/25) and CI-DME, anti-VEGF therapy is initiated at our practice. Patients are often treated for about three months. If further assistance is necessary, we’ll consider a steroid implant such as Ozurdex or Iluvien. In our practice, many cases defined as CSME may still benefit from focal laser. Yet, the management of each case is assessed on an individual basis.

Studies such as the Diabetic Retinopathy Clinical Research Network’s Protocol V may in fact help us to better assess the efficacy of anti-VEGF therapy in cases where the visual acuity is 20/25 or better. This study compares three approaches to management in patients with center-involved DME but good visual acuity: (1) prompt use of laser plus deferred anti-VEGF, (2) observation plus deferred anti-VEGF and (3) prompt use of anti-VEGF therapy.

Researchers hope the results will reveal how long anti-VEGF therapy can be deferred and the impact of observation or laser treatment in such cases. There is still debate as to the prompt use of anti-VEGF: does the benefit justify accepting the known risks, and what is the optimal number of injections needed to maintain vision? Hopefully, Protocol V’s results will shed some light.


Fig. 3. Circinate lipid ring in case 2.

Figs. 4 and 5. The macular edema (left) resolved without therapy at three months (right).
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Dry eye, as we understand it today, is entirely different than it was 20—or even 10—years ago. Of course, the disease itself has not changed, but our understanding of it has. As a result, we can now offer our patients comfort, relief, improved ocular health, better vision and a quality of life that we struggled in vain to attain just a few years ago.

Thanks to tremendous amounts of clinical research and the compilation of that research in the recently published Tear Film and Ocular Surface Society (TFOS) Dry Eye Workshop (DEWS) II reports, anyone practicing optometry can make this specialty as fundamental to primary care as a dilated fundus exam. As you’ll discover in this continuing education program, you don’t need expensive equipment to provide high-quality, comprehensive ocular surface care. New technology makes it easier in higher-volume settings and in clinics that want to build a strong dry eye specialty presence, but by no means is this necessary. However, a current understanding of dry eye is essential. This is what TFOS DEWS II offers.

The following discussion breaks down these extensive reports into an easy-to-follow, five-step process. As you will learn, a contemporary approach to dry eye involves early detection and proactive intervention to slow the cascade of disease. This is in stark contrast to the old approach, when we would watch-and-wait for identifiable...
signs of moderate to late-stage disease. Our current approach to more advanced cases is also very different today than it was a few years ago, because we now have a better understanding of effective strategies to quell disease and safeguard the ocular surface, lids and meibomian glands from further compromise. All of this is good news for patients and great news for the doctors on the frontlines who encounter this complex disease all day, every day.

**STEP ONE: TRIAGE**

*Dr. Karpecki:* TFOS DEWS II lays out a very structured and methodical way of getting to the bottom of dry eye disease. From a practical perspective, in most offices, this would begin when the patient first checks in at your office. The recommendation of the Diagnostic Committee is to begin with a set of triaging questions. Do you find this helpful in terms of practice flow and identifying dry eye?

*Dr. O’Dell:* I find it very helpful. The recommended triaging questions save a lot of time, error and oversight.

*Dr. Karpecki:* Which questions do you find most useful?

*Dr. O’Dell:* I find, “How long have your symptoms lasted and was there any triggering event?” the most beneficial. Most times, a dry eye patient has a gradual onset of symptoms. They can’t say that everything went wrong on Sept 28th. But when you have a patient who can pick the exact day, you’re onto something you may never have otherwise discovered. Sometimes, you’ll find that patients you thought had dry eye in fact have recurrent corneal erosion or epithelial basement membrane dystrophy. The other big time-saver is, “Are the symptoms or any redness much worse in one eye than the other?” Having this information before you step into the exam room is incredibly valuable.

### THE BIRTH OF A NEW DEFINITION FOR DRY EYE

The revised definition of dry eye that was created by TFOS in the TFOS DEWS II report states: “Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.”

This definition is different from the original TFOS DEWS definition in meaningful ways. It was important to this subcommittee that the definition has international relevance. A lot of time was spent talking about individual words and how they would translate in different languages.

Next, important terms were identified. For example, it’s important that dry eye is recognized as a disease because this suggests that there must also be diagnostic and management criteria, whereas a syndrome might be viewed as simply a nuisance. “Multifactorial” is another important term that puts dry eye into clearer perspective. However, the words the committee focused on most were “loss of homeostasis.” This is new to the definition. It was included because we know that there are things about dry eye that we can’t yet grasp, but the term “loss of homeostasis” is an umbrella term that accounts for these unknowns. As such, this new definition may not need to be changed for quite some time.

The new definition also recognizes that symptoms are important but don’t need to be specifically defined, since they present in many different ways and affect comfort and/or vision to varying degrees.

Finally, the latter part of the definition reflects our current knowledge of the etiological pathways, but leaves an opening for what we might learn in the future. For example, neurosensory abnormalities were included in the new definition as a direct result of a preponderance of new research in that area.

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### Panel Participation in Tear Film and Ocular Surface Society (TFOS)

**Dry Eye Workshop II (DEWS II) Report**

- **Dr. Karpecki:** TFOS Diagnostic Methodology Subcommittee member and TFOS Global Ambassador
- **Dr. Jones:** TFOS Treatment and Management Subcommittee Chair and Steering Committee member
- **Dr. Nichols:** Co-chair of the TFOS Definition and Classification Subcommittee and Steering Committee member
- **Dr. O’Dell:** TFOS Public Awareness and Education Subcommittee member and TFOS Global Ambassador

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**REVIE W O F O P T O M E T RY 3 7 A P R I L 2 0 1 8**
**Modernize Your Methods of Dry Eye Care**

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**STEP TWO: RISK FACTOR ANALYSIS**


- How severe is the eye discomfort?
- Do you have any mouth dryness or swollen glands?
- How long have your symptoms lasted and was there any triggering event?
- Is your vision affected and does it clear on blinking?
- Are the symptoms or any redness much worse in one eye than the other?
- Do the eyes itch, appear swollen or crusty, or have they given off any discharge?
- Do you wear contact lenses?
- Have you been diagnosed with any general health conditions (including recent respiratory infections) or are you taking any medications?

**TFOS DEWS II TRIAGING QUESTIONS**

1. How severe is the eye discomfort?
2. Do you have any mouth dryness or swollen glands?
3. How long have your symptoms lasted and was there any triggering event?
4. Is your vision affected and does it clear on blinking?
5. Are the symptoms or any redness much worse in one eye than the other?
6. Do the eyes itch, appear swollen or crusty, or have they given off any discharge?
7. Do you wear contact lenses?
8. Have you been diagnosed with any general health conditions (including recent respiratory infections) or are you taking any medications?

**Dr. Karpecki:** Moving on to medications, what role do glaucoma drops play in terms of being a risk factor for dry eye disease?

**Dr. O’Dell:** Topical glaucoma therapy creates ocular surface disease, so we need to screen these patients, whether or not they’re complaining about their drops burning or end-of-the-day discomfort. Many patients require multiple medications to control their disease, which increases their dry eye symptoms exponentially. Fortunately, we are starting to see a paradigm shift in the way we manage glaucoma, with the introduction of alternative preservatives, preservative-free options and first-line surgical options including selective laser trabecuoplasty (SLT) and minimally invasive glaucoma surgery (MIGS).

**Dr. Karpecki:** Are you seeing dry eye at an earlier age?

**Dr. O’Dell:** Absolutely. Future research needs to take a closer look at what’s happening in the under-40 crowd. Everyone is staring at screens, including young children. Consider the effects of this over time.

**Dr. Karpecki:** Indeed, we’re starting to see those effects even now. We’re all familiar with the original Beaver Dam research, but when researchers looked at the participants’ offspring in the Beaver Dam Offspring Study (BOSS), they discovered that the incidence of dry eye in the patients between age 21 and 34 was about 13%, which is about the same incidence aren’t going to work well if you’ve got a patient whose ocular surface is damaged. We need to address the ocular surface before we start making other changes in contact lens wearers. Looking for meibomian gland dysfunction and Demodex is a great place to start in any symptomatic contact lens wearer. Always look for those two things before changing anything else. Then, if you still need to change the lens, the previous TFOS report on contact lens discomfort offers adequate evidence to support switching to a daily disposable modality.*

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as their parents. That's a pretty high number. Does it strike you as unrealistic?

Dr. Jones: It doesn’t surprise me at all. In our clinics at the University, the number of patients who report symptoms has definitely increased because of this digital world—even when patients are not wearing their lenses. Digital device use reduces blink rate, which increases evaporation, which in turn is leading to an increase in symptoms. The take-home message here is that you need to routinely ask patients of all ages whether they are suffering from symptoms of dry eye. This includes the whole under-40 crowd, including kids.

Dr. Nichols: It’s also important to look at the young people in your practice and think about prevention, because if they’re showing signs in their 20s, imagine what they’re going to be living with when they are 50, 60 or 70 years old. We need to try and halt progression and prevent dry eye from emerging in the first place.

Dr. Jones: Many kids also are at much greater risk due to acne medications, such as isotretinoin derivatives. This is commonly prescribed and can cause brutal dry eyes, dry mouth and dry skin.

Dr. O’Dell: Acne medications aren’t the only culprits. Some of the acne face washes have been linked to keratitis. There’s still a lot to learn about the chemicals in cosmetics including soaps, shampoos, lotions and face wash. Our patients are being exposed to these products every day without knowing the residual effects to the ocular surface.

Dr. Karpecki: Why specifically are we still seeing a predilection after age 35 towards women?

Dr. O’Dell: I learned so much from the TFOS DEWS II Sex, Gender, and Hormones Committee. First, to clarify terms, “sex” refers to your biological makeup, and “gender” relates to what the environment does to influence your behavior. In both male and female patients, the level of hormones and androgens in every part of the lacrimal system—from meibomian glands to the lacrimal gland to the accessory glands—is unique based on sex. In post-menopausal patients especially, androgens play an enormous role.

**STEP THREE: DIAGNOSTIC TESTS**

Dr. Karpecki: Step three is diagnostic testing, which includes a symptomology screening questionnaire plus at least one of three diagnostic tests to measure homeostasis. Do you rely heavily on a particular dry eye questionnaire?

Dr. Nichols: We tend to use the Ocular Surface Disease Index (OSDI) because it provides a great benchmark, and has excellent reliability and validity, and good sensitivity and specificity. If you choose to ask more questions yourself, you don’t need a survey, but if you are pressed for time, as most doctors are, the detailed surveys allow you to see other patients while you collect critical information.

Dr. Jones: Asking about symptomology using a questionnaire is very helpful for diagnosis. It’s also great for management because it gives you a benchmark, so when you do begin therapy you can reliably measure any shift that occurs.

Dr. Nichols: And these two are selected because they’ve been validated or have shown a shift with treatments. And so, you’re right. If you don’t see any change at all, maybe you need to approach your management structure a bit differently.

Dr. Karpecki: That’s an excellent point. Like OSDI, the Dry Eye Questionnaire-5 (DEQ-5) and the SPEED questionnaire also offer a score, which helps in diagnosis and later in terms of management so you know whether or not your treatment plan is working.

Dr. Karpecki: The list of dry eye symptoms is long. It includes burning, stinging, transient blur, dryness, photophobia, epiphora, blurred vision, contact lens intolerance, injection, increased blink rate, foreign body sensation and grittiness. Among these, is there any area that you’re starting to look more closely at in recent years?

Dr. O’Dell: I’m taking a more serious look at increased blink rate because it leads to a vicious cycle of inflammation that’s hard to break, even with today’s topical therapies. In some cases, we’ve had to use botulinum toxin injections to slow the blinking. But even
this is tricky because if you slow it too much you can cause even more dry eye. I often first notice blink rate issues at the slit lamp when patients appear to be overly photophobic during the exam.

Dr. Nichols: I agree. I also elicit information on blink problems by asking patients whether they stop and close their eyes during a work day or at the end of the day. Many patients—especially contact lens wearers—say their eyes are tired and they need to blink a lot to try and clear the vision. The visual parts of the symptomatology are central to how I gather meaningful information about dry eye. These patients generally think there's something wrong with their prescription when, in fact, it has more to do with blinking too much. The body is trying to maintain adequate tear breakup on an unstable tear film.

Dr. Karpecki: After screening, the three tests identified in the TFOS DEWS II report for measuring homeostasis are non-invasive tear breakup time, osmolarity or ocular surface staining testing. Which of these do you perform?

Dr. Nichols: The guidelines point out that you only need to perform one of these, but we often do all of them.

**STEP FOUR: SUBTYPE CLASSIFICATION**

Dr. Karpecki: After your screening and symptomatology review, the TFOS DEWS II guidelines call for subtype classification testing. In other words, is the dry eye primarily evaporative or is it mostly aqueous deficient? It’s often mixed, but the new guidelines suggest looking at that scale and determining the severity of each subtype to figure out where the patient is on the dry eye spectrum. What is the benefit of going through this classification exercise? Does it offer a better explanation about why signs and symptoms don’t always correlate?

Dr. Nichols: This is where I think we stopped short in previous definitions. DEWS II offers a new classification scheme that makes it clear that dry eye isn’t either aqueous deficient or evaporative. It is often a combination of both, occurring on a sliding scale, which makes it necessary to treat both.

Dr. Karpecki: What is your testing protocol?

Dr. Nichols: You have to look at the meibomian glands and tear volume. There are many high- and low-tech ways to test for evaporative vs. aqueous deficient dry eye. Taking a thorough look and taking your findings seriously are what matters. For example, if you see only one isolated blocked gland in a contact lens patient, don’t ignore it. Treat it because this patient is on a road to a place they don’t want to go. They already have disease.

Dr. Jones: It is worthwhile reading the entire TFOS DEWS II report, but if you had to read just one part, it would be the diagnostic report. It dissects every single dry eye test that could or should be done in succinct detail. When you see it put together like this, you realize that this isn’t rocket science. Every single one of us can do this using readily accessible tools.

Classification of Dry Eye Disease*

STEP FIVE: TREATMENT

Dr. Karpecki: Once you know what you’re trying to combat, treatment becomes much more straightforward. But have we reached the point where we can pick a single treatment based on our diagnostics and know it’s going to be the silver bullet?

Dr. Jones: One of the biggest challenges for the Management and Therapy Committee was grouping the incredibly diverse list of therapies and suggesting when to use them. To give you an idea of how much research there is on this topic, the Management and Therapy Committee report has 1,002 references. What we found was that one treatment alone often doesn’t work. Instead, you may need to mix and match a few things. To simplify this process, the committee developed a four-step process, progressing from the simplest therapies to the more complex. (See TFOS DEWS II Staged Management and Treatment Recommendations).

Dr. Nichols: Indeed, the Management and Therapy section is not the cookbook that we may wish it could be, but it provides a level of evidence that puts us on the right path. One outcome that we hope for most as a result of this report is that it will be a call to action, inspiring more head-to-head studies that will help us select those silver bullets.

Dr. Karpecki: In the meantime, one thing we do know about dry eye is that inflammation needs to be addressed at every level.

Dr. Jones: That’s very true. By definition, dry eye has an inflammatory component. It’s worthwhile bearing in mind that many of our mildly symptomatic patients have low levels of inflammatory response that now we can effectively manage at this earlier stage, getting them out of the vicious cycle of the inflammatory response that precipitates further damage to the ocular surface, more tear film break-up time and more inflammation. You’ve got to get in there and break that cycle.

ACT SOONER RATHER THAN LATER

Dr. Karpecki: A recent study looked at patients preparing for cataract surgery. The patients who had osmolarity scores within normal limits were within a half diopter of intent, whereas 17% of those with hyperosmolarity would have missed their IOL calculation by more than a diopter. What is your protocol for cataract surgery candidates?

Dr. Jones: You should always aim to have a primed, pristine optical surface before referring a patient for cataract surgery. Especially if you have a patient who demonstrates an interest in a multifocal or other pre-

TFOS DEWS II STAGED MANAGEMENT & TREATMENT RECOMMENDATIONS

Step 1:
- Education regarding the condition, its management, treatment and prognosis
- Modification of local environment
- Education regarding potential dietary modifications (including oral essential fatty acid supplementation)
- Identification and potential modification/elimination of offending systemic and topical medications
- Ocular lubricants of various types (if MGD is present, consider lipid-containing supplements)
- Lid hygiene and warm compresses of various types

Step 2:
- If above options are inadequate, consider:
  - Non-preserved ocular lubricants to minimize preservative-induced toxicity
  - Tea tree oil for Demodex (if present)
  - Tear conservation
  - Punctal occlusion
  - Moisture chamber spectacles/goggles
  - Overnight treatments (such as ointment or moisture chamber devices)
  - In-office, physical heating and expression of the meibomian glands (including device-assisted therapies, such as LipiFlow [TearScience])
  - In-office intense pulsed-light therapy for MGD
  - Prescription drugs to manage DED
  - Topical antibiotic or antibiotic/steroid combination applied to the lid margins for anterior blepharitis (if present)
  - Topical corticosteroid (limited duration)
  - Topical secretagogues
  - Topical non-glucocorticoid immunomodulatory drugs (such as cyclosporine)
  - Topical LFA-1 antagonist drugs (such as lifitegrast)
  - Oral macrolide or tetracycline antibiotics

Step 3:
- If above options are inadequate, consider:
  - Oral secretagogues
  - Autologous/allogeneic serum eye drops
  - Therapeutic contact lens options
  - Soft bandage lenses
  - Rigid scleral lenses

Step 4:
- If above options are inadequate, consider:
  - Topical corticosteroid for longer duration
  - Amniotic membrane grafts
  - Surgical punctal occlusion
  - Other surgical approaches (e.g., tarsorrhaphy, salivary gland transplantation)


To obtain two hours of continuing education credit, complete the exam by recording the best answer to each self-assessment question online at: https://www.reviewofoptometry.com/c/ce/frontline-ocular-surface-disease-care-march18. Or, mail the Examination Answer Sheet on the next page to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. A minimum score of 70% is required to obtain a certification of completion. The fee for this course is free.

1. TFOS DEWS II refers to dry eye as a ____________.
   a. Syndrome
   b. Disease
   c. Nuisance
   d. None of the above

2. The TFOS DEWS II revised definition of dry eye disease includes the following: “Dry eye is a multifactorial disease of the ocular surface characterized by a loss of ____________, and/or ____________ of the tear film and accompanied by ____________ symptoms.”
   a. Equilibrium
   b. Homeoeostasis
   c. Balance
   d. Equanimity

3. The following is a true statement about the TFOS DEWS II definition of dry eye?
   a. Symptoms must be specifically defined
   b. Symptoms are unimportant
   c. Symptoms affect comfort and/or vision to varying degrees
   d. None of the above

4. The Beaver Dam study found that the incidence of dry eye in the patients between age 21 and 34 was about ____________.
   a. 4
   b. 11

5. In what demographic population should you routinely ask patients whether they are suffering from symptoms of dry eye?
   a. Individuals under 40 years of age
   b. Women over 40
   c. Men over 40
   d. All of the above

6. What is a true statement when classifying patients into types of dry eye disease?
   a. Patients can’t be diagnosed with both aqueous deficient dry eye and evaporative dry eye simultaneously
   b. Many patients have some element of both aqueous deficient dry eye and evaporative dry eye
   c. Patients with aqueous deficient dry eye always exhibit more signs and symptoms than those with evaporative dry eye
   d. Patients with evaporative dry eye exhibit more signs and symptoms than those with aqueous deficient dry eye

7. What aspect of glaucoma might contribute to increasing the risk for ocular surface disease?
   a. Higher intracocular pressure
   b. Visual field loss
   c. Topical therapies that use preservatives
   d. MIGS procedures

8. Though the TFOS DEWS II report left symptoms open, what common symptoms of dry eye do patients often present with?
   a. Increased blink rate
   b. Blurring
   c. Dropping lids
   d. A and B

9. The TFOS DEWS II Diagnostic Committee set forth the need to first begin asking patients questions to help an eye care practitioner make an assessment about whether a patient might have dry eye disease and the type.
   a. Trick
   b. Traising
   c. Undercover
   d. Rapid-fire

10. The TFOS DEWS II report recommended which of the following diagnostic tests as homestasis markers?
    a. Noninvasive tear break-up time
    b. Osmolarity
    c. Ocular surface staining
    d. All of the above

11. The TFOS DEWS II report suggested that, to classify subtypes of dry eye disease into evaporative and aqueous deficiency, it is important to investigate abnormal lipids as well as possible ____________.
    a. Ocular hypertension
    b. Corneal abrasions
    c. Meibomian gland dysfunction
    d. Retinal tears

12. Expressing the meibomian glands and assessing tear volume can especially help with what aspect of dry eye diagnosis?
    a. Differentiating signs from symptoms
    b. Differentiating symptoms from signs
    c. Differentiating the type of dry eye
    d. Determining the severity of dry eye

13. Which of the following triaging questions is recommended?
    a. Do you have any mouth dryness or swollen glands?
    b. How long have your symptoms lasted and was there any triggering event?
    c. Are the symptoms or any redness much worse in one eye than the other?
    d. All of the above

14. What commonly prescribed medication puts kids at...nally IOL, you have to make sure that the ocular surface is healthy. It makes a big difference.

Dr. O’Dell: If I have a patient that I’m sending out for cataract surgery, I always have them come back for a dry eye evaluation with me before they go to the cataract surgeon so I can start therapy several weeks before it’s time for surgery.

Dr. Karpecki: Dry eye disease is a chronic, progressive disease that affects millions of people, yet too often it is approached as a mere nuisance, as evidenced by the fact that so few people are receiving medical treatment.5,11 It’s encouraging that there appears to be a growing commitment to action, at least before surgery.

Dr. Nichols: That’s true, but the next step needs to be acting sooner rather than later. Our changing environment and increased dependence on digital technology only adds to this growing problem, making intervention and prevention that much more critical to sustained quality of life as patients age.
CE TEST

risk of developing dry eye disease?

a. Amoxicillin
b. Azithromycin
c. Isotretinoin derivatives
d. Bupropion

15. What was one of the biggest challenges for the TFOS DEWS II Management and Therapy Committee in preparing recommendations for the report?

a. Large number of management and therapy options available
b. Lack of management and therapy options available
c. Unwillingness of doctors to utilize available management and therapy options available
d. Lack of patient access to management and therapy options available

16. How many steps did the Management and Therapy Committee come up with as eye care practitioners progress through from the simplest therapies through to the more complex?

a. 3
b. 4
c. 5
d. 6

17. What is a true statement about the four therapeutic steps?

a. The steps are unchangeable
b. The steps should not be customized to the patient
c. They’re not mutually exclusive
d. They are mutually exclusive

18. Step 1 includes which of the following recommendations:

a. Education on dry eye, and environmental and dietary modifications
b. Identification and potential modification of offending medications
c. Various types of ocular lubricants, lid hygiene and warm compresses
 d. All the above

19. Step 2 prescribes which possible strategies if Step 1 approaches are inadequate:

a. Non-preserved ocular lubricants, tea tree oil for Demodex (if present) and tear conservation (punctal occlusion and moisture chamber spectacles/goggles)
b. Overnight treatments (such as ointment or moisture chamber spectacles/goggles)
 c. In-office physical heating and expression of the meibomian glands, as well as intense pulsed light therapy and laser devices;
d. Ibuprofen

20. What strategies are not mentioned in Step 3 for dry eye that is nonresponsive to therapy?

a. Amniotic membrane treatment and sceral lenses
b. Intracocular lenses
c. LASEK procedures
d. B and C

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Examination Answer Sheet
Valid for credit through December 31, 2018

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Frontline Ocular Surface Disease Care

Directions: Select one answer for each question in the exam and completely darken the appropriate circle. A minimum score of 70% is required to earn credit.

Mail to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY, 10001.

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There is an eight- to 10-week processing time for this exam.

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10. State _______ _______ _______ _______

11. ZIP _______ _______ _______ _______

12. Telephone #: _______ _______ _______ _______

13. Fax #: _______ _______ _______ _______

By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by any fraudulent or improper means.

Signature _______________ Date _______________

Lesson #116224 RO-UAB-0318
**Systemic Therapy**

**Know Your Systemic Meds:**

The Top 10 to Track

Here’s what you need to know about the ocular effects of the heavy-hitters.

*By Megan Hunter, OD, and Michelle Marciniak, OD*

While systemic medications are often necessary for the patient’s long-term health, ocular side effects, as minor as dry eye and as serious as macular toxicity, can challenge the treatment process, often leading to modification or even discontinuation of the medication.

Optometrists must be prepared to manage and comanage patients who present with concurrent medication use and ocular concerns. Here, we discuss some of the commonly prescribed systemic medications with serious ocular side effects, and what to look out for.

**Hydroxychloroquine**

In the United States, Plaquenil (hydroxychloroquine, Sanofi-Aventis) is commonly used to treat rheumatic conditions such as rheumatoid arthritis, systemic lupus erythematosus and Sjögren’s syndrome. Plaquenil use has the potential risk of retinal toxicity. Overall, the incidence is low; however, risk depends on the length of therapy and cumulative dosage. After five years of use, the risk of toxicity is <1%, but increases to 2% after 10 years and nearly 20% after 20 years. The underlying pathophysiology of the toxicity is not well understood, although researchers do know that Plaquenil affects the metabolism of retinal cells, including the photoreceptors. Since the toxicity predominately affects the macula, light absorption, cone metabolism or both may play a role. Hydroxychloroquine binds to melanin, which concentrates in the retinal pigment epithelium and prolongs the effects, even after discontinuation of the medication. Tamoxifen use and renal disease can also result in increased risk.

Optometrists must screen patients taking Plaquenil for early functional and structural changes associated with toxicity, because once a clinically evident bull’s...
eye maculopathy is present, the damage cannot be reversed.\(^3\) The current American Academy of Ophthalmology (AAO) recommendations advise screening with objective test for functional retinal damage—such as spectral-domain (SD) optical coherence tomography (OCT), fundus autofluorescence or multifocal electroretinogram (ERG)—in addition to a dilated fundus exam and 10-2 visual fields.\(^1\) Clinicians should use wider visual field test patterns (24-2 or 30-2) for patients of Asian descent, as research shows this patient population has early damage in a more peripheral, rather than paracentral, pattern.\(^1\)

High-resolution OCT can detect localized thinning in the parafoveal region, and loss of the inner/outer segment line, known as the ellipsoid zone, on SD-OCT is considered one of the earliest signs of potential toxicity. Fundus autofluorescence abnormalities have been reported prior to visual field loss and early photoreceptor damage will show an increase in autofluorescence due to outer segment debris accumulation.\(^2\) Multifocal ERG produces local responses across the posterior pole and will show paracentral ERG depression in early Plaquenil toxicity.

The medication dosage is also an important factor in monitoring for toxicity. Plaquenil is typically prescribed as 200mg or 400mg daily. Clinicians should use real weight rather than ideal weight when calculating dosage because ideal weight can cause an overdose in thin individuals.\(^1\) The target dosage is usually less than or equal to 5mg/kg of real weight. Because the cumulative dose poses the highest risk of retinal toxicity, the AAO’s recommendations call for a baseline examination at the initiation of therapy and then annual screening after five years of treatment for low risk patients.\(^1\) More frequent screening is recommended for high-risk patients, including those on a higher dose and those with renal or liver disease.\(^3\)

**Topiramate**

This sulfonamide monosaccharide is an effective anti-seizure medication that enhances the action of gamma-aminobutyric acid, decreases the action of glutamate and is associated with NAION, seen here, has been linked to the use of PDE 5 inhibitors. The medication dosage is also an important factor in monitoring for toxicity. Plaquenil is typically prescribed as 200mg or 400mg daily. Clinicians should use real weight rather than ideal weight when calculating dosage because ideal weight can cause an overdose in thin individuals.\(^1\) The target dosage is usually less than or equal to 5mg/kg of real weight. Because the cumulative dose poses the highest risk of retinal toxicity, the AAO’s recommendations call for a baseline examination at the initiation of therapy and then annual screening after five years of treatment for low risk patients.\(^1\) More frequent screening is recommended for high-risk patients, including those on a higher dose and those with renal or liver disease.\(^3\)
and blocks sodium channels. It is also a common treatment option for migraine and any number of other conditions such as bipolar, post-traumatic stress and obsessive-compulsive disorders.4

ODs must carefully monitor patients taking topiramate, as it can cause ciliary body effusion, which relaxes the zonules and allows lens thickening, resulting in a myopic shift of as much as -9.00D.5,6 It can also cause anterior rotation of the ciliary processes, possibly inducing appositional acute angle-closure glaucoma.7,8 Topiramate-induced angle-closure typically occurs within the first two weeks of starting the medication or within hours of doubling the dose.6

The incidence of myopia and acute angle-closure with topiramate use is a well-recognized adverse reaction, but the true incidence is generally thought to be low. A 2004 study cited only 86 cases of topiramate-associated acute angle closure and 17 cases of acute bilateral myopia in the literature at the time.9 Since 2004, fewer than 50 additional case reports have been published.10

B-scan ultrasound, ultrasound biomicroscopy and anterior segment OCT can reveal the anatomic changes that create this reaction.

Most ocular issues associated with topiramate resolve after discontinuing the medication. Oral hypotensives can help control intraocular pressure, and cycloplegics can help retract the ciliary processes.

Phosphodiesterase Inhibitors
The active ingredient in erectile dysfunction drugs, phosphodiesterase 5 (PDE 5) inhibitors block the predominant metabolizer of cyclic guanosine monophosphate that stimulates the smooth muscle relaxation necessary for erection.

The most common ocular complaint with PDE 5 inhibitors is a bluish tinge or haze to vision along with increased light sensitivity. Other ocular side effects include decreased color vision, blurred or transitory decreased vision with central haze, changes in light perception, transient ERG changes, conjunctival hyperemia, ocular pain, mydriasis (may be an emotional effect) and retinal vascular occlusions and subconjunctival hemorrhages, which may be secondary to exertion.

Ischemic optic neuropathy (ION) is the most concerning potential side effect. Although infrequent (3% of cases) in those taking low doses (25mg to 50mg), incidence can reach 11% in those taking 100mg and 50% at 200mg.11 Symptoms begin 15 to 30 minutes after taking the medication and peak at 60 minutes. Research suggests ION has a temporal association with PDE 5 inhibitors, but this remains controversial.12 For one, many patients taking these medications also have risk factors for ION, and the vascular risk factors for nonarteritic anterior ION (NAION) often overlap with vascular risk factors for erectile dysfunction.

Fingolimod
Gilenya (fingolimod, Novartis), the first oral treatment for relapsing-remitting multiple sclerosis (MS), is effective in reducing the number of relapses and improving overall disability progression over long-term follow up.13 Fingolimod is an immunomodulating agent that binds to sphingosine-1-phosphate (S1P) receptors on the lymphocytes and prevents lymphocyte release from the lymph nodes, reducing lymphocyte migration into the central nervous system.14 This spares the system from attack by these myelin-reactive lymphocytes.

As sphingolipids are the third most numerous lipid in the retina, fingolimod-associated macular edema (FAME) is a possible side effect, reported in two pivotal studies that investigated the drug’s safety and efficacy.13,15 S1P receptors in retinal blood vessel endothelial cells are responsible for maintaining cell adhesion complexes, so when fingolimod downregulates the S1P receptors, it also downregulates adhesion complexes and increases retinal vascular permeability, resulting in macular edema.16 In one study, 0.5% of patients receiving 0.5mg of daily fingolimod and 1.0%
of those receiving 1.25mg of daily fingolimod developed macular edema.\textsuperscript{15}

Most patients who develop FAME do so within four months of starting treatment, and it resolves after the drug is discontinued.\textsuperscript{16} Clinicians should screen patients taking fingolimod with a complete eye examination and macular OCT at baseline and again in three to four months. Clinicians should exercise caution when treating diabetic patients due to the preexisting blood-retinal barrier compromise.

\textbf{Isotretinoin}

An analog of vitamin A, this is used to treat severe, recalcitrant nodular acne, psoriasis and disorders of keratinization. It alters skin lipid composition, reduces sebaceous gland size and decreases sebum production, in addition to having anti-inflammatory properties.\textsuperscript{17}

Isotretinoin-induced meibomian gland dysfunction leads to eye dryness, ocular surface irritation, decreased contact lens tolerance, photophobia, blepharoconjunctivitis, decreased vision and even an increase in hordeolum and chalazion frequency.\textsuperscript{18} Studies link isotretinoin to the development of secondary increased intracranial hypertension due to increased resistance to cerebrospinal fluid outflow.

Patients will often present with headaches (typically postural), blurred vision, subjective visual obscurations and tinnitus. Increased intracranial pressure can cause bilateral optic nerve head edema, which can last for weeks after discontinuing the medication and can be vision-threatening if left undetected. While a serious complication, increased intracranial pressure is rare.

This patient being anti-coagulated with warfarin presented with subconjunctival hemorrhage.
Tamoxifen
This oral anti-estrogen medication is used to treat advanced metastatic breast cancer and as an adjunctive endocrine therapy following resection in early disease. Endocrine therapy works to combat steroid hormone assisted tumor growth in hormonally dependent breast cancer cells. Tamoxifen competes with estradiol for receptors in breast, uterine and vaginal tissues as well as in tumors containing high concentrations of estrogen receptors.19

Ocular toxicity from tamoxifen presents as a crystalline retinopathy with yellow-white refractile bodies confined to the plexiform and nerve fiber layers, typically found in the macular and paramacular areas. Sometimes, it can be associated with macular edema and a whorl-like superficial keratopathy.20 Ocular toxicity is typically associated with high doses in excess of 120mg daily; it is not common with the standard daily dose of 20mg.21,22

SD-OCT imaging helps to detect subtle maculopathy associated with tamoxifen use. Low dose usage, for example, can produce pseudo-cystic cavities in the macula, which do not leak on fluorescein angiography and do not increase retinal thickness.22

Research suggests Müller cell dysfunction contributes to tamoxifen ocular toxicity because the medication inhibits the glutamate-aspartate transporter present in Müller cells.23 Excess glutamate, in turn, may cause Müller cell dysfunction and neuronal apoptosis, creating cavitory spaces.24 One study found that full-thickness macular holes were five times more likely in patients taking tamoxifen, possibly caused by the foveal cavities.25 All patients on tamoxifen should have a baseline and yearly dilated eye examination with SD-OCT of the macula.

Ethambutol
This medication is most commonly used as part of the lengthy quadruple antibiotic regimen for the treatment of tuberculosis (TB) and in the multi-drug therapy for mycobacterium avid complex lung disease.26 It is a metal chelator, which prevents cell wall synthesis in mycobacteria by inhibiting arabinosyl transferase.27

Ethambutol is known to cause optic neuropathy characterized by bilateral central vision loss, color vision loss, central or cecocentral visual field scotomas and eventual optic atrophy. The exact mechanism is unknown, but researchers theorize it is due to either decreased mitochondrial copper, which is necessary for oxidative phosphorylation, or the chelation of zinc, which inhibits lysosomal activation.28,29

Toxicity is dose related with an incidence of 18% with more than 35mg/kg/day, 5% to 6% with doses of 25mg/kg/day, 3% with 20mg/kg/day and less than 1% at less than or equal to 15 mg/kg/day.30

Between 30% and 64% of patients will show some visual recovery if the optic neuropathy is detected early and the medication is discontinued.27 Patients should have a baseline exam with vision screening, color vision, Amsler grid and visual field testing, in addition to a dilated fundus exam and nerve fiber layer OCT. They should be re-examined monthly while on the ethambutol therapy.

Tamsulosin
Flomax (tamsulosin, Boehringer Ingelheim Pharmaceuticals) is a selective α1A-adrenergic receptor antagonist used to treat benign prostate hyperplasia by relaxing smooth muscles in the neck of the bladder and in the prostatic urethra. These receptors are also found on the iris dilator muscle, leading to ocular effects of the medication. Selective α1A antagonists are known to cause intraoperative floppy iris syndrome (IFIS) during cataract extraction and a likelihood of iris prolapse through the surgical wound.31 The exact mechanism of IFIS is unknown, although one study suggests chronic blockade of the α1A-adrenergic receptors can decrease smooth muscle tone and cause diffuse atrophy of the iris dilator.32 The researchers found histologic evidence of decreased iris dilator muscle thickness in cadaver eyes with a history of tamsulosin use compared with
Surgeons and comanaging ODs must be aware that IFIS may still occur several years after discontinuing the drug, and stopping tamsulosin preoperatively is of uncertain benefit.

**Atrial Fibrillation Agents**

Several vascular medications come with significant ocular effects ODs must be on the lookout for:

**Amiodarone** is an effective anti-arrhythmic medication for ventricular tachycardia and fibrillation and atrial fibrillation. It blocks calcium, potassium and sodium channels in cardiac tissue, which prolongs the cardiac action potential and refractory period. It is also a weak beta blocker and vasodilator.

Ocular side effects of amiodarone include corneal verticillata or whorl-like keratopathy, which occur in 70% to 100% of patients. This keratopathy is related to the dose and duration of treatment, generally appears after at least one month of therapy and will resolve upon discontinuation of the medication. Amiodarone can also cause anterior lenticular subcapsular opacities in 50% to 60% of patients. Both the corneal and lenticular deposits are typically asymptomatic.

Amiodarone has also been linked to optic neuropathy, with a clinical presentation similar to NAION. Amiodarone-induced optic neuropathy should be a diagnosis of exclusion, considering most patients taking amiodarone have vascular risk factors already increasing their NAION risk. One report found an incidence of amiodarone-induced optic neuropathy at roughly 2% compared with a general incidence of 0.3%. It has an insidious onset, milder degree of vision loss, longer duration of disc edema and is more commonly bilateral than NAION. Amiodarone has a long half-life, averaging 58 days, but up to 142 days, and accumulates in the lysosomes of multiple tissues, including the optic nerve, so optic disc swelling can persist for months after discontinuing the medication.

**Digoxin** inhibits the cell membrane Na+/K+-ATPase, creating an increase in intracellular calcium and sodium and extracellular potassium. This leads to stronger myocardial contraction and excitability and a decrease in the conduction and depolarization velocity in the atrioventricular node, which resets the rhythm of the heart rate. Digoxin is a second-line treatment for atrial fibrillation and in severe symptomatic systolic heart failure.

Approximately 95% of patients in the toxic range will report ocular symptoms, the most common being xanthopsia, or yellowing vision. Cyanopsia (blue vision) and chloropsia (green vision) have also been reported. Other ocular symptoms include flashing,
scintillating or moving lights or spots, blurred, hazy, misty or snowy vision and difficulty reading. Clinical ocular finding include decreased visual acuity, central scotoma, color vision defects and delayed b-wave time and decreased b-wave amplitude on ERG. While still under debate, most presume ocular symptoms are related to Na+/K+ ATPase inhibition.38
Symptomatic patients should be referred back to their ophthalmologist for monitoring.
Anticoagulants are used to help prevent clotting in a wide variety of systemic disorders. The ocular and systemic side effects occur with varying frequencies depending on the specific medication. Platelet inhibitors such as clopidogrel, dipyridamole and aspirin are effective but with a lower incidence of side effects.
Warfarin is a potent oral anticoagulant that poses a high risk of bleeding disorders. It inhibits vitamin K-dependent gamma-carboxylation of coagulation factors II, VII, IX and X. Warfarin can be affected by other medications, as well as a patient's dietary intake of vitamin K. Because of its narrow therapeutic window, warfarin requires frequent monitoring via laboratory testing.41
Direct-acting oral anticoagulants (DOACs) are used to treat patients with venous thromboembolism and nonvalvular atrial fibrillation.42 Research shows DOACs are as effective as warfarin in preventing stroke due to atrial fibrillation while also having a lower overall bleeding risk, especially intracranial bleeding. In addition, they are less affected by diet and medication interactions.43
Atrial fibrillation agents all carry the risk of increased bleeding. The most worrisome is the potential for intracranial hemorrhage. Ocular side effects of anticoagulants include subconjunctival hemorrhage, retinal hemorrhage, orbital hemorrhage causing mass effect and exacerbation of bleeding from other ocular conditions such as proliferative diabetic retinopathy and exudative macular degeneration.
Bisphosphonates
These are widely used in the treatment of osteoporosis, Paget’s disease, metastatic bone disease, multiple myeloma and hypercalcemia of malignancy. Bisphosphonates are rapidly cleared from circulation and are absorbed into bone mineral surfaces, where they slow osteoclasts to allow osteoblasts to rebuild bone.44
The most common ocular side effect of bisphosphonates is a self-limiting conjunctivitis.45 Uveitis and scleritis, two serious possible ocular side effects, might not resolve unless the medication is discontinued.46 One study found scleritis and uveitis were rare in bisphosphonate users and that 43% of the patients who developed either condition had a systemic condition already associated with uveitis or scleritis.47 Researchers can only speculate that bisphosphonates use might be a precipitating factor for ocular inflammation in patients already prone to these conditions.44 Patients who develop serious ocular inflammation need oral and topical anti-inflammatories and often require discontinuation of bisphosphonates for resolution.
Ocular side effects from systemic medications can be serious and sight threatening. It is crucial for ODs to catalogue each patient’s systemic medications, discuss the possible side effects and monitor appropriately. Often additional testing is warranted to screen for effects and detect early toxicity.
Dr. Hunter is an assistant professor of ophthalmology at Loyola University Medical Center in Maywood, IL. She is the current national chair of the American Academy of Optometry’s admittance committee.
Dr. Marciniak works at the Jesse Brown VAMC, teaches at Illinois College of Optometry and works in private practice. She is a member of the Advanced Competency in Medical Optometry committee.
7. Maddalena MA. Transient myopia associated with acute glaucoma and retinal edema. JAMA


Due to their complexity and temperament, ocular infections can be a pain to treat. Finding the right antibiotic requires a thorough understanding of the most likely causative organisms, resistance patterns, patient-specific data and pharmacologic information that may impact the patient’s response to the drug. And we have to do it all while the clock is ticking—the earlier treatment begins, the better the prognosis. However, sometimes, less is more. Exercising discretion in situations where the infection is most likely to be caused by a virus, such as in non-purulent infectious conjunctivitis, has been proven to decrease the spread of resistant organisms.1,2

Into this thicket walks the optometrist, who must navigate carefully and make many decisions to move forward safely.

**Chiefs of Staph.**
The first key question is whether to use an oral or a topical agent. Oral antibiotics are accepted as the standard of care when treating infections
Launch your adoption of amniotic membrane with the Jump Start Kit. Call Katena for details!

Amniotic membrane is considered an appropriate end stage treatment option to address conditions outlined in the DEWS II step 4 guidelines for severe dry eye.*

*Physicians may choose to use amniotic membrane at their discretion.


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such as hordeolum, preseptal cellulitis and dacryocystitis.\(^3,4\) Compared with topical antibiotics, orals produce significant levels in the bloodstream, which provide superior penetration of the lacrimal apparatus and surrounding tissues. For this reason, these infections are more effectively treated with an oral agent.\(^3\)

Most corneal and conjunctival infections are best managed with a topical fluoroquinolone, perhaps with an oral agent added for more coverage if circumstances warrant.

Common organisms associated with hordeolum, dacryocystitis and preseptal cellulitis include the gram-positive organisms *Staphylococcus aureus*, *Staphylococcus epidermidis* and *Streptococcus pneumonia* (Table 1). But they also include gram-negative organisms such as *Haemophilus influenza* and occasionally *Pseudomonas aeruginosa*.\(^5-7\)

When choosing an oral antibiotic to treat these relatively common ocular infections, inquire about the presence of allergy, pregnancy, breastfeeding, comorbidities, drug interactions, prior antibiotic use and ability to pay out of pocket for the more costly medications.

**MRSA Matters**

Since methicillin-resistant *Staphylococcus aureus* (MRSA) is a common colonizer on skin, clinical consideration should be given to determine whether a patient needs antibiotic coverage for this organism. This is typically determined by local prevalence and risk factors.\(^5-7\)

While sulfamethoxazole-trimethoprim (SMX-TMP), doxycycline and clindamycin may not be an OD’s typical “go to” drugs when treating hordeolum, preseptal cellulitis and dacryocystitis, they are important to consider, especially when managing community-acquired (as opposed to hospital-acquired) resistant *Staph. aureus* MRSA infections (Table 2).\(^33\)

In the past, practitioners relied on intravenous vancomycin to treat MRSA infections, but today we are fortunate to have SMX-TMP, doxycycline and clindamycin in our MRSA arsenal. Doxycycline is generally considered a “last-line” in the trio of agents that cover MRSA, mainly because of tolerability issues. When a patient fails traditional therapy that covers *Staph. aureus*, or when a patient is colonized with MRSA, it’s a good time to consider a potential MRSA infection. Susceptibility to MRSA is also heightened in young patients, prisoners and athletes.\(^29,30,33\)

**Beta-Lactams**

These antibiotics are widely used to treat upper respiratory tract infections and ocular infections. (Table 3).

*Amoxicillin and amox + clavulanate.* These agents are considered amino-penicillins or extended spectrum penicillins because the amino side-chain extends its coverage to include more gram-negative organisms such as *Haemophilus influenza* and the Enterobacteriaceae family, such as *Escherichia coli*, when compared with penicillin VK.

Additionally, these agents have good coverage against sensitive *Staph. aureus* and even *Streptococcus pneumoniae* when given in high doses. Keep in mind that coagulase-negative *Staph*. species (e.g., *Staph. epidermidis*) show a decent amount of resistance to these agents and may not be a reasonable choice in a patient infected with this organism. Neither MRSA nor *Pseudomonas aeruginosa* is susceptible to these agents.\(^9,10\)

Mechanistically, these agents

<table>
<thead>
<tr>
<th>Table 1. Likely Organisms Responsible for Lid/Adnexal Infections</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hordeolum</strong></td>
</tr>
<tr>
<td><strong>Preseptal Cellulitis</strong></td>
</tr>
<tr>
<td><strong>Dacryocystitis</strong></td>
</tr>
</tbody>
</table>

The resolution of this patient's dacryocystitis and preseptal cellulitis was achieved using antibiotics.
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inhibit bacterial cell wall synthesis by plugging into penicillin-binding proteins. This results in bacterial cell death. The addition of the beta-lactamase enzyme inhibitor clavulanate provides increased protection for amoxicillin against hydrolysis when it’s exposed to infections caused by organisms that produce beta-lactamase, including Hae-mophilus influenzae and Moraxella catarrhalis.

While traditional penicillin has impaired gastrointestinal absorption when given with food, amoxicillin is relatively stable. Additionally, amoxicillin plus clavulanate has improved gastrointestinal tolerability when given with food and is generally recommended that patients take it with a meal. While both amoxicillin and amoxicillin plus clavulanate are both fairly well-tolerated, the latter is more likely to cause nausea and vomiting. Drug interactions with these two agents are miniscule, and the regimen is generally considered safe to take while pregnant.9,11

**Cephalexin.** A first-generation cephalosporin, this has gram-positive coverage similar to amoxicillin, but is considered more stable against hydrolysis by beta-lactamase-producing organisms. MRSA and Pseudomonas aeruginosa are not susceptible to this agent. Side effects with cephalexin are well-tolerated and include miniscule drug interactions. In general, cephalexin has also been considered safe in pregnancy.9,10

**Allergy ambiguities.** Decades of clinical research have allowed us to fine-tune our understanding of penicillin allergies and the potential cross-reaction between a penicillin allergy and other beta-lactam antibiotics (Table 4). While some sources report cross-reactions between penicillins and cephalosporins occurring as often as 10% of the time, other research suggests that this is an exaggeration. Nevertheless, the mere “potential” of this cross-reaction has created a culture in which physicians tend to avoid beta-lactam agents and overuse the broader spectrum agents such as macrolides and fluoroquinolones.

When evaluating a patient who claims to possess a “penicillin allergy,” practitioners should investigate the specific nature of the allergic reaction to the drug. Researchers estimate that 90% of patients who claim to be allergic to a penicillin are not actually allergic.9,10,12-14 Typically, the cephalosporins, possessing similar side chains, increase the likelihood of a cross-reaction. For example, cephalexin is more likely to cross-react with a patient who has penicillin allergy compared with a drug lacking the side chain (e.g., cefuroxime axetil). If a patient is listed as having an allergy to a cephalosporin, it’s wise to consider that claim to be true. Also, a cross-reaction from a cephalosporin to a penicillin is rare.12-13

**Meet the Macrolides**

The macrolide class of agents includes erythromycin, clarithromycin and azithromycin. Erythromycin is incredibly well-known to cause nausea and vomiting and is typically taken three to four times per day. For obvious reasons, this agent is rarely used in eye care.

Clarithromycin, on the other hand, causes a moderate amount of nausea and vomiting and is dosed BID. As it shows no real benefit over azithromycin, it is used less. The third agent in this class, azithromycin, proves fairly well-tolerated and is reported as having the fewest drug interactions. It has become the drug of choice for infections requiring a macrolide treatment. It is taken twice daily on the first day and then once for another four days. Due to the

---

**Table 2. Dosing Regimens for Community-Acquired MRSA**

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfamethoxazole+Trimethoprim DS</td>
<td>800mg/160mg; take one tab BID x 10 days</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>300mg to 600mg TID x 10 days</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>100mg BID x 10 days</td>
</tr>
</tbody>
</table>

* higher dose = increased S. pneumoniae coverage.

**Table 3. Dosing Regimens for Beta-Lactam Agents**

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>500mg BID-TID x 10 days</td>
</tr>
<tr>
<td></td>
<td>875mg BID x 10 days*</td>
</tr>
<tr>
<td>Amoxicillin+Clavulanate</td>
<td>500mg/125mg BID-TID x 10 days</td>
</tr>
<tr>
<td></td>
<td>875mg/125mg TID x 10 days*</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>500mg BID x 10 days</td>
</tr>
</tbody>
</table>
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post-antibiotic effect, five days of therapy treat bacteria systemically for approximately 72 hours past the last dose.18

When Side Effects Set In
Pharmacologically, once we move away from inhibitors of bacterial cell wall synthesis (beta-lactams), we begin to see more side effects from antibiotics because they have an effect on bacterial cells and mammalian cells that are rapidly dividing. Examples of these rapidly dividing cells include those found in the skin, gut and bone marrow.16,17

Mechanistically, the macrolides are protein synthesis inhibitors and bind to the 50S ribosomal subunits in susceptible organisms. *Staph. aureus* and *Strep. pneumoniae* are generally less sensitive to azithromycin as compared with the beta-lactam agents.

On the contrary, *Haemophilus influenzae* is generally more sensitive to azithromycin than to the beta-lactam agents. While azithromycin is generally well tolerated, with occasional mild gastrointestinal complaints, it may cause QT prolongation (a heart arrhythmia) in susceptible patients. It has miniscule drug interactions and is generally considered safe in pregnancy.16-18

Face the Fluoroquinolones
The fluoroquinolone group of systemic antibiotics includes levofloxacin, ciprofloxacin, moxifloxacin and gemifloxacin, colloquially known as the “respiratory fluoroquinolones,” for their excellent *Strep. pneumoniae* coverage. The ophthalmic fluoroquinolones are also divided into two categories. The earlier-generation topical agents include ciprofloxacin and ofloxacin. The newer “fourth-generation” topical fluoroquinolones include besifloxacin, moxifloxacin, levofloxacin and gatifloxacin.22-26

Ciprofloxacin. This earlier-generation agent is another frequently used oral fluoroquinolone. While it no longer provides reliable *Strep. pneumoniae* coverage, it does have relatively good efficacy against *Pseudomonas aeruginosa.* Both ciprofloxacin and levofloxacin have decent coverage against *Haemophilus influenzae* but no appreciable MRSA coverage.

Levofloxacin. Due to the differences in spectrum of coverage between ciprofloxacin and levofloxacin, ocular infections are generally treated with levofloxacin if and when a fluoroquinolone is necessary. If a pseudomonal infection is suspected or confirmed, consider ciprofloxacin instead. While levofloxacin may have sufficient pseudomonal coverage, it should be reserved for gram-positive infections to decrease potential resistance issues.11,19-26

Gemifloxacin and moxifloxacin. As members of the “respiratory fluoroquinolone” group, gemifloxacin and moxifloxacin have bacterial coverage similar to levofloxacin. Occasionally, a *Strep. pneumoniae* isolate may be more sensitive to one agent than another in this group; however, they are generally considered fairly equivalent from an empiric treatment perspective. In some situations levofloxacin and moxifloxacin may have slightly broader coverage against highly resistant *Strep.* species. While less well known, both gemifloxacin and levofloxacin are generally considered interchangeable with levofloxacin in the management of gram-positive infections. Levofloxacin has slightly improved gram-negative activity.20,21,27,28

These drugs are bacterial DNA synthesis inhibitors. It’s generally accepted that fluoroquinolones have chelation drug interactions, which result in a decrease in systemic absorption when the drug is given with di- and trivalent cations (e.g., multivitamins, calcium, iron). As such, any dietary supplements that are cations should be given either two hours prior or four hours after the fluoroquinolone.19-26

Adverse effects associated with fluoroquinolone antibiotics are perhaps their most notable drawback. They are generally contraindicated in pregnancy, breastfeeding and in children younger than 18 years old, due to the corresponding risk of
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Effects for these agents as well.20,21 Lrokes (and QT prolongation from the respiratory fluoroquinolones) are listed as potential side effects for these agents as well.20,21

Additionally, the risk of tendon rupture in the shoulder and Achilles has been well documented; it seems more likely to occur in elderly patients and in those taking systemic corticosteroids.31 While practitioners once suspected a correlation between fluoroquinolone use and the presence of a red eyelid,32 this notion has debunked this.36 Additional research and clinical anecdotes will further determine the severity of this risk.35,33

The expanding role of the optometrist requires a multi-dimensional perspective on the treatment of infections that typically require systemic therapy. Additionally, staying current on resistance patterns, drug interactions and side effects is critical to one’s ability to choose the appropriate agent as well as one that will best encourage patient compliance.

Dr. Offerdahl-McGowan is an assistant professor of biomedicine at Salus University.

Dr. Caldwell is an oculist consultant in Duncansville and Johnstown, PA, and a past president of the Pennsylvania Optometric Association.

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Dry eye disease (DED) is a multifactorial disorder of the ocular surface that severely impacts vision and quality of life. Risk factors that contribute to DED include age, gender, hormones, autoimmune disorders, local environment, use of video displays, contact lens wear and exposure to medications/preservatives, all potentially leading to secretory or evaporative DED, or both. Inflammation is another key risk factor. The Tear Film and Ocular Surface Society’s Dry Eye Workshop II definition recognizes the impact of inflammation, saying, “Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.”

With this more finely honed understanding of DED in mind, optometrists must make controlling inflammation a priority. This article will review the optometrist’s role in diagnosing inflammation in the clinical setting, including options to best treat inflammatory dry eye disease with the tools currently available.

Identification and Classification
Inflammation on the ocular surface can be overt or covert in its presence. Conjunctival hyperemia, or redness, is a hallmark of ocular inflammation that can be objectively evaluated by anterior segment photography or with the use of grading scales, such as the Efron Grading Scales. Vital dyes are also valuable in the detection of ocular surface damage often seen alongside inflammation. The use of sodium fluorescein or lissamine green vital dyes are particularly helpful in identifying and visualizing the devitalized cells on the conjunctiva.

Tear chemistry can also be associated with ocular surface inflammation. Using a system designed to measure tear osmolarity (e.g., TearLab Osmolarity System, or I-Pen [I-Med Pharma]) is one way to quantify inflammation. For patients without inflammation, their hyperosmolarity will read less than...
With this quantification, physicians can engage their patients by providing pretreatment and post-treatment results. Research shows matrix metalloproteinases, specifically matrix metalloproteinase-9 (MMP-9), is a byproduct of inflammation and cell insult and, now that an MMP-9 detection test (e.g., InflammaDry Detector, Quidel) is available, we can count that as yet another biomarker of inflammation.

The clinical symptoms of inflammation include end-of-day burning, stinging, dry, irritated, scratchy, watery and itchy eyes. If a patient presents with these clinical signs and symptoms, therapeutic intervention is indicated. As soon as patients can be categorized as mild-to-moderate—by either exhibiting corneal staining signs, decreased tear film break-up time or complaining of DED symptoms—anti-inflammatory medications are justified.

**The Right Stuff**

When deciding which anti-inflammatory to use, keep in mind each drug’s mechanisms of action.

Corticosteroids act on various inflammatory responses, including intercellular adhesion molecule-1 (ICAM-1)-mediated cell adhesion, cytokines/chemokines/MMPs expression and induction of lymphocyte apoptosis. Simply put, corticosteroids have a broad-spectrum mechanism of action when it comes to controlling inflammation. However, their long-term use in ocular conditions is not recommended because of steroid-related side-effects such as increased intraocular pressure and cataract formation.

Thankfully, not all corticosteroids are created equal. A “soft” steroid such as loteprednol or fluorometholone could be used for four to six weeks without having to worry about the long-term side effects. Sometimes I will also pulse the soft steroid BID to TID dosage for one week on and two weeks off, then repeat as needed with close clinical follow ups.

Cyclosporine (CsA) is another anti-inflammatory medication used to combat inflammation associated with DED. In a large multicenter study, 0.05% to 0.1% CsA treatment significantly reduced HLA-DR expression and to a lesser extent expression of other inflammatory and apoptotic markers in patients with moderate-to-severe DED. Topical CsA increase goblet cell density and conjunctival production of immunomodulatory transforming growth factor beta-2 (TGF-β2). Topical cyclosporine 0.05%, Allergan) received approval from the FDA to increase tear production in patients whose tear production is presumed to be suppressed due to ocular inflammation associated with keratoconjunctivitis sicca. Because of the mechanism of action of CsA, the anti-inflammatory effects usually won’t take place until after at least four to six weeks of starting therapy. This won’t be the best medication to use if you have a patient with acute inflammation. Also because of the immunomodulator properties of CsA, it is a good option to treat autoimmune disease associated dry eyes, such as Sjogren’s syndrome.

Lifitegrast is an anti-inflammatory drug that was approved by the FDA in 2016 for the treatment of the signs and symptoms of DED. Xiidra (topical lifitegrast 5%, Shire) is the first dry eye medication that targets integrin signaling as a lymphocyte function-associated antigen-1 (LFA-1) antagonist that blocks coupling of LFA-1 and ICAM-1. LFA-1 is found on the surface of the T-cell. By blocking the interaction between LFA-1 and ICAM-1, it is thought that one of the inflammatory cycle is prevented. In the OPUS-1 clinical study, there was a significant improvement in signs such as inferior corneal staining, but not in symptoms. The subsequent OPUS-2 study established significant improvement in the eye dryness score, and this was confirmed in OPUS-3. Symptomatic relief occurred as early as two weeks after initiating treatment for patients with moderate DED.

After determining the severity of the inflammation in a DED patient, whether by having a positive response to the InflammaDry test, or by simply grading the clinical signs; the optometrist can then decide if a
Topical steroid is needed to quickly decrease the overall inflammation along with either Restasis or Xiidra. If the inflammation is particularly severe, you might consider using a soft steroid with both Restasis and Xiidra together, as their mechanisms of action are different. However, if the inflammation isn’t so severe it requires a topical steroid, lifitegrast (for faster symptom relief) and CsA (if the patient has autoimmune disease associated DED) without the steroid is acceptable.

Complementary Treatments
Controlling inflammation is a vital step in reducing DED symptoms, but optometrists can also treat mechanical issues that can be (but aren’t always) the root cause of dry eye. For instance, patients with meibomian gland dysfunction have obstructed glands and may be able to find relief once they are unclogged. A conservative approach involves using heat therapy in the form of a warm compress that is directly applied to the lids to try to reduce the viscosity of the meibum within. A more aggressive approach involves in-office treatments, such as Lipiflow (Tearscience), a thermal pulsation device that simultaneously heats the meibum and pulsates to liberate the melted meibum out of the glands.

Diet is another aspect in which we can directly affect the level of inflammation in the eye. Oral supplementation with omega-3 polyunsaturated fatty acids, eicosapentaenoic acid or docosahexaenoic acid found in fish oil all decrease the production of pro-inflammatory mediators, such as prostaglandin E2, and cytokines. Gamma-linolenic acid (GLA) is also an omega-6 with anti-inflammatory properties. HydroEye (Science Based Health) provides the necessary GLA formulation through black currant seed oil.

Another contributor to inflammation is blepharitis from the lids. The etiology of the condition is not fully understood, but low-grade bacterial infection (primarily *Staphylococcus*), Demodex mites, environmental factors and certain systemic disease have all been implicated as potential contributors.

The Combo Approach
Topical antibiotic-corticosteroid combination products are useful for decreasing the bacterial load as well as managing the inflammation. Concerns of antibiotic resistance and long-term exposure to the corticosteroids comes to mind when using these type of medications. After a short course of a topical antibiotic-corticosteroid combo medication, you may want to switch the patient to a type of hypochlorous acid (HOCl) cleanser such as Avenova (NovaBay) or Acucyn (Sonoma Pharmaceuticals) to keep the bacterial levels at bay. HOCl is a natural product made by neutrophils to kill microorganisms. Oral doxycycline is another tool that can be used to control inflammation via MMP suppression. A maintenance dose of 50mg BID can be prescribed for up to three months to try to control the inflammation systemically.

Topical steroids should not be used long-term without frequent monitoring because of the potential side effects. They can be used initially to control the acute stage of inflammation and then tapered off. CsA in the form of Restasis is safe to use long term but requires contin-
ued use to achieve its anti-inflammatory properties and also can take up to two months before noticing any improvement in DED symptoms. Xiidra is also safe to use long term and can work on the current inflammatory response for faster resolution.

Dr. Dang is an optometrist practicing in a multispecialty eye clinic in Bakersfield, California. His clinical practice covers a broad spectrum of ocular care with a unique clinical focus on ocular surface disease and dry eye.

PROTOCOLS AND PITFALLS IN TOPICAL STEROID USE

It's always a balancing act between benefit and side effects. Here's how to keep patients safe while treating with steroids. By Aaron Bronner, OD, and Walter O. Whitley, OD, MBA

Topical corticosteroids are the most frequently prescribed class of ophthalmic agents by optometrists across the United States. In 2013, topical corticosteroids (including steroid/antibiotic combination agents) accounted for a little more than 50% of ophthalmic prescriptions written by ODs. Steroids were prescribed three times more frequently than topical antibiotics, topical antivirals and topical nonsteroidal anti-inflammatory drugs (NSAIDs) combined. And it’s no wonder we like these drugs, given their mechanism of action. Because essentially all nucleated cells in the body express receptors for these molecules, corticosteroids have a sweeping effect across many tissues.3

Regardless of dosing strategy (systemic or local), the therapeutic goal of corticosteroids is to limit the immune response via the phospholipid A2 pathway. Because of their wide range of target cells, corticosteroids have the broadest, but least specific, effect on inflammation of any immune modulatory agent.4 When dosed orally for systemic therapy, these agents reduce differentiation and maturation of immune cells within primary immune tissue, as well reduce the expression of pro-inflammatory cytokines and chemokines peripherally.4 When dosed topically, steroids don’t influence immune-cell maturation, but the effect on cytokines, chemokines and other pro-inflammatory molecules—an extremely robust mechanism of action—is preserved.4

By reducing the production of this legion of inflammatory mediators, locally dosed corticosteroids reduce several actions, including: vascular permeability to immune cells, the recruitment of other immune cells to that site, tissue breakdown, histamine release and subsequent edema, angiogenesis and fibroblast activation (which results in scar tissue), among other consequences.4 With these broad effects and their trickle-down clinical benefits, treating everything from ocular surface disease to uveitis, it’s easy to see why corticosteroids are such a favorite of the profession. However, although steroids are quite effective at addressing inflammation, they can cause complications. Common side effects

Release Date: March 2, 2018
Expiration Date: March 2, 2021
Goal Statement: Topical corticosteroids can be a useful treatment strategy for everything from ocular surface disease to uveitis—but they can cause complications, and clinicians must account for their side effects. To better prepare clinicians to prescribe these agents safely, this article reviews the known side effects of ophthalmic corticosteroids, appropriate use and dosing-based differences among target tissues.
Faculty/Editorial Board: Aaron Bronner, OD, and Walter Whitley, OD, MBA

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of oral prednisone include increased intraocular pressure (IOP), cataract formation, mood changes and increased blood sugar levels. These are not associated directly with the desired mechanism of these agents and are truly unintended; with increased IOP possibly leading to irreparable vision loss via glaucoma, it’s particularly worrisome.

Another negative effect of steroid use is not an unexpected adverse event, but a predictable effect based on the mechanism at play. Because the primary effect of corticosteroids is limiting the immune response, their use may result in worsening of any condition where the immune response is needed (i.e., an infectious process). Unfortunately, although this effect can potentially lead to catastrophic vision loss, it is often under-recognized by the profession.

To better prepare clinicians to prescribe these agents safely, this article reviews the known side effects of ophthalmic corticosteroids, particularly within the cornea, their appropriate use and dosing-based differences among target tissues.

**Intraocular Pressure**

Elevation in IOP is the most frequently observed side effect of ophthalmic steroid use, occurring in 18% to 36% of patients.5 Regardless of the specific steroid being used, this response is more likely to occur with prolonged dosing and has been observed in higher frequency in glaucoma suspects and patients.5 Research suggests the mechanism likely involves the alteration of the trabecular meshwork’s (TM) outflow efficacy via increased deposition of extracellular matrix within the TM.7 This is usually transient, though recalcitrant steroid-induced ocular hypertension is possible.

Ophthalmic steroid preparations come in varying strengths with equally varying risks of IOP elevation. For example, loteprednol 0.5%, rimexolone 1% and fluorometholone 0.1% are associated with a lower risk of steroid response, a lower peak pressure when a response occurs and a slower time to peak compared with dexamethasone phosphate 0.1% or prednisolone acetate 1%.8,10

The mechanism by which these steroids achieve their increased safety differs slightly between medications. In fluorometholone 0.1% and rimexolone 1%, lipophility plays a large part in reducing IOP spikes, as the medications don’t penetrate the cornea due to biphasic nature, thereby lowering the potential for spikes.11,12 Phosphate preparations, as hydrophilic solutions, penetrate the epithelium poorly, which may be ideal for ocular surface conditions.11,12

Hydrophobic alcohol-based and acetate suspensions, however, should theoretically be more adept at penetrating all layers of the cornea. Loteprednol achieves its enhanced safety, in part, from its reduced half-life in the anterior chamber as an ester-steroid, which reduces adverse drug reactions. The newest topical steroid, difluprednate 0.05%, seems to have the highest anti-inflammatory potency.13 This comes at a cost, however, as research shows the medication can cause the most extreme and most rapid IOP responses.14

**Cataract Development**

The literature shows significant variability in cataract formation in patients on systemic steroids, as between 6.5% and 38.7% of patients on them develop cataracts.15 Further research reveals varying associations with cataract and inhaled steroid compared with perinatal dosing.16 Whether topical, oral or inhaled, steroids are commonly associated with posterior subcapsular cataracts.16 Surprisingly, however, there is little to no peer-reviewed information regarding rates of cataract development with ophthalmic corticosteroids overall, let alone among specific agents.

Because of loteprednol’s soft drug design, research suggests it has a reduced potential to cause cataracts; however, this claim seems to be based on one retrospective review of 159 patients, none of whom developed cataracts while using loteprednol 0.2% for more than a year.16

Although the differences in the potential to cause cataracts among individual agents are still unknown, we do know that chronic topical steroid use, at least in cohorts with chronic intraocular inflammation, is associated with cataract formation—with greater dose frequency associated more frequently than lesser dosages.17 Practitioners can reasonably predict that long-term topical steroid use follows the same trends seen in IOP response among specific agents: those that don’t penetrate into the anterior chamber well and those that have a short half-life (fluorometholone, rimexolone and loteprednol) can be expected to produce cataracts at a lower rate than more potent steroids (prednisolone, dexamethasone and difluprednate).
Selection and Dosing
When dosing corticosteroids for inflammation of the ocular surface, select a poorly penetrating agent (fluorometholone or rimexolone) or soft drug corticosteroid such as loteprednol to reduce risk of complications.

When inflammation is in the anterior chamber or deeper cornea, the selected agent should balance with the degree of anti-inflammatory effect necessary. For mild to moderate inflammation, lotsprednol is a good option, and more severe cases may do well with prednisolone acetate 1%. In particularly severe cases of corneal or anterior chamber inflammation, the only topical medication we’ve had success with is difluprednate; otherwise, you’re on to sub-Tenon’s dosing or systemics.

Excess steroid use can be nearly as harmful as chronic inflammation and is of particular concern with long-term management of inflammation as seen in chronic keratitis (as seen occasionally with herpes zoster ophthalmicus), prevention of corneal graft rejection and cases of chronic uveitis. When deep penetration is unnecessary but chronic therapy is expected, a more superficially acting steroid is appropriate, and in all cases the lowest dose that fully controls inflammation should be used.

With more substantial steroids and the increased potential for steroid complications over an abbreviated time frame, the follow-up interval should be appropriately reduced.

Immune Response and Tissue Function
Beyond the capacity for creating IOP spikes and cataracts, steroid use comes with tissue-specific precautions. Optometrists typically prescribe corticosteroids for three primary anatomic zones: the ocular surface (including lids, conjunctiva, episclera and corneal epithelium), the non-superficial corneal layers and the anterior chamber. Steroid use for each of these zones carries a risk for the development of cataracts and glaucoma, depending on the specific agent and duration of treatment. The risks also differ based on the consequences of potentiating infection.

Conjunctiva. True conjunctival infections, with the exception of hyper-acute conjunctivitis, can generally be treated with corticosteroids with near impunity and without fearing induction of vision-threatening escalation of the disease. The reason for this has to do with the tissue function, its role in vision and the normal immune response.

A mucous membrane, the conjunctiva is essentially a watershed between the external and internal environments. As with all mucous membranes, one of the conjunctiva’s primary functions is to act as a barrier. The most effective barrier tissue of the body is the skin. Non-nucleated, keratinized tissue, which makes up the most external layer of the skin, creates a robust physical barrier to microbes and is able to repel most infections. In fact, it is nearly impossible for a virus to infect healthy, uncompromised skin.18

As a mucous membrane without the benefit of keratinized tissue, the conjunctiva is at a disadvantage relative to the skin at preventing infection; therefore, infectious conjunctivitis is more likely than infectious dermatitis. These conjunctival infections, however, are almost always self-contained and have minimal long-term consequences because what the conjunctiva lacks in keratinized tissue it makes up for with immediate access to the immune response. The conjunctiva is host to secondary lymphoid tissue (conjunctiva-associated lymphoid tissue), has a rich vascular supply and, further, is not directly responsible for visual function—allowing some tissue damage without impact on function.19

Due to the conjunctiva’s immediate access to the immune response, topical steroid use may reduce the degree of the conjunctival immune response without eliminating it entirely. Steroids can be a useful adjunct in the therapeutic and palliative management of both inflammatory and infectious conjunctivitis by reducing unnecessary inflammation that can lead to pain, conjunctival fibrosis, destruction of goblet cells and symblepharon formation.

Clinicians must remember that infectious conjunctivitis should be preferentially treated with anti-infective medications. Still, it may be difficult to differentiate infectious from non-infectious, and given the access to local immune response, treatment of a localized infectious conjunctivitis with steroid will not lead to profound tissue destruction, unlike that which may occur within the cornea. Thus, combination drops (paired antibiotic/corticosteroid) are safe and effective in most cases.

Likewise, anterior uveitis stemming from disseminated systemic infections such as tuberculosis, syphilis, Lyme disease and leprosy may be treated with topical corticosteroids without risk of exacerbating the underlying disease process. Of course, these patients all need systemic anti-infectives to cure their disease, but topical steroids are safe, may enhance treatment by reducing inflammatory sequela and may reveal an infectious etiology if you see an incomplete clinical response to anti-inflammatory treatment.

Although the uveitis generated by these etiologies often exhibits an incomplete response to topical corticosteroids, topical steroid use is always appropriate front-line therapy for cases of anterior uveitis.20

The same cannot be said for systemic corticosteroid agents may

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be indicated on a case-by-case basis for infectious disease management (shingles treatment is often supplemented with oral prednisone, and some severe leprosy-related immune reactions necessitate their use), they should not be used for all cases. They have been known to exacerbate the underlying disease and possibly even lead to death.21

When using topical agents to control a severe uveitis, clinicians should not shy away from strong initial dosing. Anecdotally, treating uncontrolled anterior uveitis with prednisolone acetate 1% QID often meets with predictably minimal success. In cases of moderate to severe acute anterior uveitis, clinicians should prescribe frequent, often hourly, dosing of a strong steroid such as difluprednate, which has been shown to be non-inferior to prednisolone acetate for anterior uveitis when dosed half as frequently, and only taper gradually once inflammation is totally controlled.22

**Cornea.** Corticosteroids are often necessary to facilitate a good, prompt resolution of corneal pathology; however, when used incorrectly they may result in catastrophic vision loss. The specific implications and risks associated with steroid use in the cornea revolve around the cornea’s function and immune response.

The cornea has two primary functions: an optical interface that transmits and refracts light and a barrier to the external environment. However, maintenance of one sets up challenges for the other. The external cornea is part of the ocular surface mucous membrane; however, unlike most other mucous membrane-derived tissue, the cornea has no local lymphoid tissue, few native immune cells and no local vascular supply. Presence of any of these at high levels would enhance corneal barrier function through increasing access to the immune response, but would result in a loss of corneal clarity and reduced optical function. However, the absence of these features from the central cornea limits the cornea’s ability to function as a barrier. If the cornea develops an infection, the immune system is initially unable to help, which allows for early unfettered proliferation of the microbe.

Once an immune response is mounted, corneal optical function is sacrificed to preserve the barrier function, as resultant inflammation often contains the infection at the expense of a corneal scar. Because of the inherent antagonism of the cornea’s barrier and optical functions, which are precariously balanced in a healthy eye and are thrown out of balance in an infected cornea, the use of corticosteroid in infected conreas has significant possible ramifications.

A now somewhat frequently encountered misconception is that the Steroid for Corneal Ulcers Trial (SCUT) shows no benefit or harm in the adjunctive use of corticosteroids for corneal ulcers; therefore, some think we can use steroids for corneal pathology with impunity.23 This is a dangerous clinical mindset and a misinterpretation of the SCUT study and its findings. The study does not show there was no benefit or harm in the use of corticosteroids for corneal ulcers. It shows, instead, no benefit or harm with light use of corticosteroid (prednisolone phosphate 1% QID for one week, BID for one week, then QD for one week) in the management of bacterial keratitis when attempts were made to sterilize the cornea prior to institution of steroid.23 All patients in this study were placed on moxifloxacin hourly for two days prior to being grouped
into steroid plus antibiotic group or antibiotic plus placebo groups. Therefore, conservative steroid use as an adjunct therapy only follows SCUT protocol when attempts to sterilize the cornea are made prior to initiation of steroid use—a critical distinction.

Due to the lack of immune response held by the central cornea, further suppression of it with corticosteroids prior to ensuring an effective antimicrobial sterilization (as assessed with a positive response to therapy) may result in spread of the ulcer and treatment failure. In addition, while the SCUT study was inconclusive on conservative use of corticosteroid in bacterial keratitis following an attempt at sterilization, we know from other retrospective reviews that steroid use with corneal ulcers increases the likelihood of progression towards outcomes requiring surgical management. More than 50% of patients from one series on infectious keratitis that went on to require keratoplasty had been on previous steroid (compared with 18% of the ulcers that did not require surgery). Likewise, nearly 25% of patients in another study on eyes requiring enucleation or evisceration for infectious keratitis had been on topical corticosteroids. These outcomes are critical to guard against; thus, the cavalier use of corticosteroids must be avoided when dealing with corneal pathology.

While most eye care providers know to avoid corticosteroid use with infectious keratitis except in an extremely conservative manner, the same is not always true with combination therapies, often prescribed for “mystery keratitides.” These have the most potential to do harm because of the flawed assumption that the antibiotic agent in the combo drop covers against infection while the anti-inflammatory covers for inflammation. In the central and paracentral cornea, the absent native immunity may not allow low-dose antibiotics to cover for the deleterious effects of the steroid when faced with an infectious challenge. Tissue concentrations of the antibiotic in these agents may be insufficient to slow colonization of the offending microbe, and the immune suppressing effect of the steroid may allow further spread by reducing immune cell migration and clearance of the infection. These possibilities are particularly concerning given that, in this setting, any worsening of infection may lead to a permanent worsening in vision.

It is crucial to know the corneal pathology you are dealing with prior to initiating combination or steroid therapy for any keratitis. Anecdotally, a good rule of thumb is to avoid using a steroid for any isolated ulcerative process of the central and paracentral cornea. If you have an infiltrate and an epithelial defect, it’s likely you are dealing with an infectious process.

The possible exception to this rule is a lesion in the far corneal periphery, as these are typically hypersensitivity/immune-mediated reactions, which will respond well to combination drops. In the rare event a far peripheral ulcer is infectious—which is uncommon near the limbus—proximity to the immune response will generally prevent dramatic escalation until initiating more appropriate therapy. Additionally, scarring in the far periphery is much less likely to cause reduced vision.

Though we aren’t promoting cavalier use of steroid on peripheral corneal pathology, the immunology and function of the peripheral cornea allows for more forgiveness in outcomes, should your treatment be incorrect. As with all corneal pathology, follow-up should be short when introducing a steroid to identify any worsening. For non-peripheral keratitis, stromal inflammation that is small, non-ulcerated and multifocal (i.e., contact lens-associated red eye, Thygeson’s and adenoviral infection) generally responds robustly and safely to corticosteroid.

Though inappropriate use of steroid on bacterial, protozoan or fungal keratitis may lead to poor visual outcomes, exacerbation of viral keratitis is probably the most frequently described negative corneal sequela of corticosteroid use in optometry. It is certainly true that treating herpes simplex virus (HSV) infections (clinically, this refers to the dendritic spectrum of disease, as well as the rare HSV-derived necrotizing stromal keratitis) with a steroid will worsen the infectious episode and increase superficial scarring.

The pathognomonic finding of a dendrite is a telltale sign that a steroid is inappropriate; however, it can be more challenging to differentiate dendritic lesions near the limbus.
from hypersensitivity reactions, often leading to the somewhat tongue-in-cheek “steroid provocative test.”

For other forms of herpetic keratitis, such as stromal keratitis and endotheliitis, topical steroids are an important part of the therapy, though their use should generally be paired with an antiviral to reduce the risk of reactivation.

For any significant unilateral vascularization, especially with a nonulcerated stromal keratitis, you are probably dealing with an immune-mediated process, likely HSV immune stromal keratitis, and may safely pair a corticosteroid with oral or topical antivirals. The same treatment holds in cases of unilateral acute corneal edema without stromal infiltration or dramatically elevated IOP, which is generally HSV endotheliitis. A sight-threatening infection of the cornea resulting in this clinical constellation is rare.

Laterality is also helpful in distinguishing what keratitides are “steroid safe.” Though it is possible that bilateral corneal inflammation may be infectious in origin, it’s unlikely—approximately 97% of HSV keratitis is unilateral and, barring exceptional risk factors such as bilateral corneal surgery, nearly all cases of microbial keratitis are unilateral.25 Although the same rules of avoidance hold—infiltrated ulcerations of the mid-periphery, paracentral or central cornea should avoid steroid—most other bilateral keratitides are safely treated with a steroid. In our experience, the most common sources of bilateral corneal inflammation are Staphylococcus hypersensitivities, rosacea keratitis, Thygeson’s, contact lens reactions and postviral epidemic keratoconjunctivitis, all of which respond well to corticosteroid or combination drops.

Because inappropriate steroid use on the cornea can have a devastating impact, follow up should be reduced when adding a steroid to evaluate for a negative response. All patients also should be counseled to call the office if the condition worsens.

Without a doubt, corticosteroids have earned their place among the class of ophthalmic agents most frequently prescribed by optometrists. Their ability to aid in the management of almost all forms of ocular inflammation, even those inflammations derived from infectious processes, means their indications for use are without peer. This familiarity, however, can breed complacency, which can lead to vision loss. For all ophthalmic steroid use, monitoring chronic use for adverse effects, particularly increased IOP, is critical. And when prescribing them, be sure to anticipate possible tissue-specific complications and adjust your follow-up schedule appropriately.

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

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OPTOMETRIC STUDY CENTER


10. Alonso-Casilla JA. Comparative study of the intraocular pressure effects of fluorometholone 0.1% versus dexamethasone 0.1%. Br J Ophthalmol. 1988;72(10):661-3.


You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form and return it with the $35 fee to: Jobson Medical Information, Dept.: Optometric CE, 440 9th Avenue, 14th Floor, New York, NY 10001. To be eligible, please return the card within a year of publication.

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


6. With increased IOP, up to what percentage of patients are steroid responders? a. 6%. b. 16%. c. 36%. d. 46%.


8. “Soft steroids” achieve increased safety due to the following mechanisms, except: a. Decreased penetration. b. Reduced half-life. c. Decreased concentration. d. Increased half-life.

Osc Quiz

follows a period of antibiotic only.
b. There is no benefit but substantial harm to adding corticosteroids for corneal ulcer treatment following a period of antibiotic only.
c. There is no benefit or harm to adding corticosteroids for corneal ulcer treatment prior to initiating antibiotic therapy.
d. Steroids are contraindicated for use in corneal ulcers.

16. Peripheral corneal infiltrates are most likely due to ________ reactions.
a. Hypersensitivity
b. Allergic
 c. Infectious
d. Viral
17. Clinical findings for herpes simplex immune stromal keratitis include all of the following except:
a. Dendrite
b. Stromal edema
c. Keratic precipitates
d. High IOP
18. What is the most common cause of vision loss after penetrating keratoplasty?
19. Microbial keratitis may be more likely by up to _______ times in patients with a PK compared with normal extended wear soft contact lens users.
a. 25
b. 35
c. 45
d. 55
20. All of the following are forms of lamellar grafting except:
b. Descemet’s membrane endothelial keratoplasty.
c. Descemet’s stripping endothelial keratoplasty.
d. Penetrating keratoplasty.

Examination Answer Sheet

Protocols and Pitfalls in Topical Steroid Use
Valid for credit through March 2, 2021

Online: This exam can be taken online at www.reviewofoptometry.com/ce. Upon passing the exam, you can view your results immediately and download a real-time CE certificate. You can also view your test history at any time from the website.

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Processing: There is a four-week processing time for this exam.

Answers to CE exam:

1. a. 2. a. 3. c. 4. a. 5. a. 6. a. 7. c. 8. d. 9. c. 10. a. 11. a. 12. a. 13. a. 14. a. 15. a. 16. a. 17. a. 18. a. 19. a. 20. a.

Post-activity evaluation questions:

1. Rate how well the activity supported your achievement of these learning objectives:
   1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree
   1. Improve my understanding of the known side effects of topical steroids. 2 3 4 5
   2. Become familiar with the appropriate protocols for topical steroid use in optometric practice. 2 3 4 5
   3. Increase my understanding of the dosing-based differences among target tissues. 2 3 4 5
   4. Better understand the effects of topical steroids on the conjunctiva. 2 3 4 5
   5. Increase my knowledge of steroid use for corneal pathology. 2 3 4 5
   6. Improve my ability to properly prescribe topical steroids for ocular conditions. 2 3 4 5

2. Rate the quality of the material provided:
   1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent
   1. The presentation was clear and effective. 4 5
   2. The content was balanced and free of bias. 4 5
   3. The content was evidence-based. 4 5
   4. The presentation was clean and effective. 4 5
   5. The content was well-organized and easy to follow. 4 5

3. Additional comments on this course:

Please retain a copy for your records. Please print clearly.

First Name ________________________________
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E-Mail ________________________________
The following is your: ☐ Home Address  ☐ Business Address
Business Name ________________________________
Address ________________________________
City ________________________________ State ________________________________
ZIP ________________________________
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Fax # ________________________________

By submitting this answer sheet, I certify that I have read the lesson in its entirety and completed the self-assessment exam personally based on the material presented. I have not obtained the answers to this exam by any fraudulent or improper means.

Signature ________________________________ Date ________________
Lesson 116173 ________________________________

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Driving with both visual acuity (VA) and visual field (VF) loss has been a hot topic in the field for decades. Although the literature routinely acknowledges that driving is a privilege, not a right, loss of driving privileges can have devastating consequences such as increased social isolation, decreased quality of life and depression. Such high stakes can make the subject of driving tough to bring up during the examination of individuals with visual impairments. However, ODs have a duty to properly assess and counsel patients with congenital or acquired visual impairments who would like to acquire driving privileges or who will become non-drivers because of their vision loss.

Here, we discuss the complexities of the current vision and licensure standards, how they affect patients with visual loss and what ODs can do to properly evaluate and counsel patients who are visually impaired and want to drive.

State Standards

One 1991 study found uniform standards did not exist in the United States for VA, VF or the use of bioptic telescopes—which is still true today. Currently, two vision standards exist for driving licensure. Some states enforce a rejection standard: an individual will automatically be rejected for licensure if they do not meet the minimum vision standards for VF or VA. Most commonly, those standards are VA in one or both eyes of 20/40 or better and a binocular field of view of 140 degrees or more. However, significant variances occur; for example, Wisconsin only requires a 40-degree field of view, and a number of states have no VF specification.

The alternative to the rejection standard is the vision screening standard. Rather than being automatically rejected if they do not meet the minimum standards, individuals may be granted driving privileges after further evaluation of all factors, including a behind-the-wheel test. For example, Iowa, allows for individual review, via a behind-the-wheel test, for individuals with VAs better than 20/200, a VF greater than 140 degrees or both.

The Aging Driver

Today, more than five million Americans age 65 and older are afflicted with a form of dementia, such as Alzheimer’s disease. By 2050, this number is expected to grow to 13.8 million as the baby-boom population ages. Data indicates that 50% of persons with Alzheimer’s disease continue to drive up to three years after they have been diagnosed with the disease, and that these individuals also have worse driving performance and are more likely to cause traffic accidents. Clinicians must be prepared to care for these patients and properly counsel them—and their loved ones—on safe driving.


Proceed With Caution: Low Vision and Driving

Here’s how you can navigate the complex interplay between DMV standards and the needs of your patients who are visually impaired.

By Mark E. Wilkinson, OD, and Khadija S. Shahid, OD, MPH
In addition to vision standards, two different licensure standards exist for driving, depending on the state. According to some states, as long as the individual’s VA and VFs are adequate enough to allow them licensure, they can continue to drive until that license expires, regardless of how poor their acuity or VFs become during the licensing cycle. Drivers in those states may have a false sense of security, despite changes in their vision, when they have a license that does not expire for several years.

In other states, individuals whose VA or VFs drop below the state’s licensure standards are no longer legal to drive from that time forward, regardless of the licensing cycle. This is the same standard used for commercial licensure, where the driver needs to know they meet the vision standard every time they get behind the wheel of a commercial vehicle. Currently, only a few states, such as Illinois and Pennsylvania, use this standard for driving.

Above and beyond these vision and licensure standards, states have different protocols for restricted driving privileges, including: daylight-only, no driving when headlights are required, reduced speed, local area driving or a restricted distance from home and no highway driving. States also vary on whether the use of a bioptic telescope is allowed for licensure, as well as what VA requirements must be met, both with the bioptic telescope and the carrier lens.

All of these variations mean an individual could be licensed in some states and not even considered for driving in others. Clinicians should be familiar with their state’s vision standards for driving to properly educate their patients who are visually impaired. Unfortunately, it can be difficult to know for sure what any given state’s driving laws entail, and you must check each DMV/DOT website or call to clarify.

**Case 1**

A 38-year-old female presented with a history of reduced vision for the past 20 years secondary to Stargardt disease. Her best-corrected visual acuity (BCVA) was 20/160-2 OD, 20/160-1 OS and 20/125-2 OU. She had small bilateral central scotomas consistent with her central acuity. A discretionary review request was made to the Iowa DOT, and she received unrestricted driving privileges following several behind-the-wheel tests that took place during the day and at night. She has maintained full driving privileges, without incident, for the past 17 years.

The relative central scotomas in this patient’s eyes, caused by Stargardt disease, did not prevent her from driving safely.
Roadblocks
Given today’s driving environment, the vision screening standards used by state departments of motor vehicles/departments of transportation (DMV/DOT) may not be satisfactory in assessing drivers’ functional vision (Cases 1 and 2). 1-10,15

Although VA has been widely used in driving regulations for decades, it is a poor predictor of performance for several reasons: 8,9

- The correlation of VA alone to accidents is less than 1%. 10
- The minimum standard of 20/40 is based on an American Medical Association recommendation dating back to 1937. 16

Research shows individuals with normal sight have a functional VA of less than 20/40 when driving at night at speeds greater than 55mph with high beams on and at speeds greater than 35mph with low beams on. 17,18

- The effective lateral field of view when driving with headlights is only 35 to 45 degrees. 19
- A driver’s license has no restrictions concerning driving during periods of dense fog, heavy rain or snow—times when even a driver with normal VA will have their vision reduced to this presumed unacceptable level.

Vision Testing for Driving
High-contrast VA testing under photopic conditions continues to be the standard for licensure. However, additional visual function tests not currently used in the United States for driving licensure may be helpful when evaluating functional abilities behind the wheel. 8-10

**Full-field, non-threshold visual field testing.** Testing that includes at least the I4e and a V4e isopter is valuable information, even in states that do not specify VF requirements. Alternately, the SSA Kinetic testing strategy on the Humphrey field analyzer (Zeiss) could be used with the standard testing protocol as well as a V4e equivalent isopter.

**Contrast sensitivity.** This is an important test for assessing an individual’s fitness to drive because research shows it is predictive of driving outcomes for individuals older than 65 with normal vision. 8

A common cause of contrast sensitivity loss is cataracts, and drivers with cataracts are 2.5 times more likely to have an at-fault accident than those without cataracts. 8,20-24

Additionally, individuals with cataracts are four times more likely to report difficulties with driving compared with individuals without cataracts. 8,20-23 Some individuals with binocular cataracts continue to experience driving difficulties, even after monocular cataract surgery. 23

Research shows improved contrast sensitivity after cataract surgery is more valuable than improved visual acuity when assessing driving difficulties due to vision. 10 Finally, one study found that contrast sensitivity of less than 1.25 log units was the only independent predictor of crash involvement for individuals with cataracts in the previous five years. 23

**Useful field of view (UFOV).** Unlike conventional visual field measures, UFOV assesses higher-order visual processing skills such as selective and divided attention and visual processing speed under increasingly complex visual displays. Thus, it more closely approximates the complexity of driving as a visual task.

As we age, our ability to process visual information slows. While not a linear progression, this slowing makes driving in complex environments more difficult. UFOV testing can be valuable in determining
which at-risk drivers with normal or near-normal visual acuity are no longer able to process visual information in a timely manner to allow for safe driving (Case 3).

However, research shows visual processing training over several hours using UFOV testing with preset criteria for success can improve not only UFOV test performance, but also the driving performance of persons older than 55. Preset criteria of 10 training sessions over five weeks resulted in an approximately 50% lower rate of at-fault motor vehicle crashes (MVCs) during the subsequent six years compared with control group.23,28

Clinical Steps
The American Medical Association’s (AMA) Physician’s Guide to Assessing and Counseling of Older Drivers recommends that clinicians assess all risk factors when evaluating drivers older than 65, including vision, cognition and motor skills.24 For example, a clock drawing test is an easy test of cognition, and UFOV is an excellent tool to use to assess cognition as it relates to fitness to drive.29 If concerns exist in any of these areas, it recommends a referral to the state DMV/DOT for a formal driving assessment.

When examining patients of driving age, clinicians should ask the following questions, regardless of the patient’s current level of visual functioning. Your patient’s answers may surprise you:

- Do you drive an automobile? If so, are you driving at night or only during the day? Do you drive only close to home, or are you driving both in town and on the highway? (For example, they may be driving at night, when they are only visually qualified to drive during the day. If you don’t ask, you won’t know to tell the patient they are not visually qualified to drive at night according to state law.)
- Do vision problems cause you to be fearful when driving?
- During the past six months, have you made any driving errors? (e.g., an at-fault motor vehicle accident related to the patient not seeing someone or not judging distance correctly.)
- Is your mobility affected by your vision? (If your patient is struggling to find their way to the exam chair or has difficulty getting through the doorway, it is hard to imagine that they can safely operate a motor vehicle.)

Patients come to us because of the quality of care we provide, which includes giving honest information about their vision and ocular health. As optometry’s scope of practice continues to expand, we share the burden of not just the accurate and timely diagnosis of ocular disease, but also the effect that disease may have on visual function. If your patient develops visual or cognitive changes that could affect their ability

Adapted and Semi-Autonomous Cars
In 2016, a total of 37,461 deaths and two million injuries were from automobile accidents, a 5.6% increase from 2015.1 With these staggering numbers, it’s no wonder so much attention has been focused on semi-autonomous safety systems and their potential to reduce accident rates. But this technology could also have a significant impact on our low vision patients. It may one day allow individuals with visual acuity and visual field loss the ability to continue driving, just as those with physical handicaps, including loss of limbs, currently have the ability to operate an adapted motor vehicle and be licensed to drive.

Low Vision

to safely operate a motor vehicle, it is your professional responsibility, and legal duty, to share that information with your patient.

Assistive Technology
Currently, in 43 states, the acquisition of a bioptic telescope—a hands-free, spectacle mounted device—is the only way an individual with visual acuity less than 20/70 can attempt to qualify for driving privileges. However, research has yet to provide definitive data on the risks and benefits associated with this assistive technology, and the topic is surrounded by controversy. One report of 300 bioptic drivers found the biggest challenge with reduced vision (binocular acuity less than or equal to 20/200) was reading street signs—they had no problems seeing other traffic, people or animals when driving. In the past, practitioners felt that when a bioptic telescope is fit on one eye and appropriate training is performed, the user can maintain peripheral awareness with their fellow eye when viewing through the telescope for wayfinding such as reading street signs.

While bioptic telescopes are only used for a small percentage of driving time (1% to 10%) for wayfinding tasks such as spotting street signs, they reduce the user’s visual field and can contribute to inattention blindness. As with cell phone use while driving, when using a bioptic telescope, the driver must attend to two tasks at once, which is difficult because of the time lag associated with switching from one activity to another. One study found as drivers switch repeatedly between tasks such as using the radio or information system, the time cost adds up, increasing inattention blindness. Some believe the distraction created by a bioptic telescope outweighs the visual benefits, but more research is needed to clarify whether driving with a bioptic telescope makes individuals who are visually impaired safer drivers than those who do not use a device.

Today’s audible GPS devices provide these patients a readily available and significantly cheaper option compared with a bioptic telescope. A GPS device can allow the driver to focus more attention on the road and the traffic around them and less time attempting to read street signs.

Based on our clinical experience evaluating hundreds of patients with vision loss in the 20/71 to 20/199 range, many continue to drive safely for decades in Iowa, where bioptic telescopes are not permitted to gain licensure. Today, all of our patients with impaired vision use a GPS system in lieu of their bioptic telescope.

Of course, if a person feels a bioptic helps them drive more safely, they should be allowed to use it. Clinicians should carefully discuss the pros and cons with patients who are visually impaired, and counsel them appropriately on their options when it comes to assistive technologies behind the wheel.

In general, a person’s fitness to drive cannot be determined by their age, VA or VF alone. The functional manifestations of various ocular conditions and an individual’s ability to compensate for any visual impairment varies widely. We must use our knowledge and tools to assess competency to drive or refer to a driver rehabilitation specialist for additional assessment.

We are responsible for helping our patients understand when their vision falls below the state’s standards and how that will affect their driving privileges. At the same time, we need to serve as advocates for those with reduced VA or reduced VFs who have the compensatory skills to continue driving safely, despite those reductions. Finally, we need to advocate for standardized VA and VF requirements on a national level, so that individuals

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Case 3
A 56-year-old male presented with a chief complaint of increasing difficulties over the past three months with both clarity and tracking when reading. He noted that he was losing his place frequently as he read from line to line and column to column. In addition, his wife said she refused to ride with him because his driving was “scary.” His BCVA was 20/20 OD/OS, and his ocular health evaluation and VFs were unremarkable. He was referred to a neuro-ophthalmologist for a positron emission tomography scan, which revealed the left occipital and parietal occipital lobes had more atrophy than the right, out of proportion to the patient’s age. He was diagnosed with the visual variant of Alzheimer’s disease (VVAD), and his urologist questioned whether he was safe to continue driving. UFOV testing found he had nine times slower visual processing speed, five times slower divided visual attention skills and severely delayed selected visual attention skills—thus, he was at a high risk for an automobile accident.

Based on these findings, he was advised to retire from all driving immediately.

The clock drawing test, as seen here, has an 86% sensitivity and a 96% specificity for assessing dementia.
who are visually impaired have the same ability to demonstrate they can safely operate a motor vehicle, regardless of their home state. ■

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Dr. Shahid is a clinical assistant professor in the Department of Ophthalmology and Visual Sciences at the University of Iowa’s Carver College of Medicine, where she provides vision rehabilitation and primary eye care.


4. Wilkinson ME. When is it appropriate to stop driving due to vision impairment? Aging and Vision. 2003;1(2).

Driving Legals

As clinicians, we have the duty to warn, which is a legal rationale designed to provide a means of protecting the patient from an unreasonable risk of harm.” Duty to warn states that failure to warn a patient of conditions that create a risk of injury will be upheld as a cause of action against eye care providers when it can be shown that the failure to warn is the proximate cause of an injury. All of this jargon means that if an individual with a visual impairment has a motor vehicle accident related to their reduced vision, they can hold their eye care provider responsible for the accident. In this case, the patient can argue that they had insufficient warning of their impair-ment, and because of their impairment, their operation of a motor vehicle or other machinery resulted in an injury. Thus, you should warn patients whose vision no longer legally qualifies them to operate a motor vehicle to abstain from driving and note this in the patient’s record.


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Registration cost: $595

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FACULTY

Paul Karpecki, OD, FAAO
Program Chair

Douglas Devries, OD

Mark Dunbar, OD, FAAO

Murray Fingeret, OD, FAAO

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**Separate registration required. Review of Optometry® partners with Salus University for those ODs who are licensed in states that require university credit. See event website for details.

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**Complimentary Workshops**  
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- **Blepharitis/MGD Workshop (1:00pm-3:00pm)**  
  Speaker: Douglas Devries, OD
- **Retinal Disease Workshop (1:00pm-3:00pm)**  
  Speaker: Mark Dunbar, OD
- **Dry Eye Disease Workshop (3:00pm-5:00pm)**  
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I occasionally see patients with classic Thygeson’s superficial punctate keratopathy. I find these cases to be difficult to treat. What are today’s best management options for these cases? Also, is there anything new regarding the disease’s etiology?

“Thygeson’s superficial punctate keratitis (TSPK) was first introduced by Phillips Thygeson in the mid-twentieth century,” says James Aquavella, MD, professor of ophthalmology at the University of Rochester Flaum Eye Institute. “Characteristically, it produces symptoms of photophobia, tearing and foreign body sensation.”

According to Dr. Aquavella, the disease’s slit lamp appearance is almost pathognomonic, typically with five to 10 intraepithelial satellite lesions in the central cornea. “The lesions are discrete, not confluent, limited in number to the central cornea, and the vision is usually only minimally impaired,” he says. “There is usually bilateral involvement, although the extent may vary from left to right.”

TSPK usually doesn’t cause staining, but “on occasion you will find an overlying epithelial disruption,” says Dr. Aquavella. “There is also minimal discharge and minimal or absent conjunctival injection.”

The disease is known for frequent bouts of recurrence. While this can make management frustrating, it also makes diagnosis easier when combined with the fact that TSPK is chronic and often bilateral, Dr. Aquavella says, because it separates the disease from similar conditions such as epidemic keratoconjunctivitis (EKC). “Usually TSPK patients still experience vision ranging from good to normal, while the vision in EKC is usually compromised,” Dr. Aquavella says. “Also, unlike TSPK, EKC is not quickly reversed.”

Management

“In TSPK, pain relief is instantaneous with the application of a bandage contact lens,” says Dr. Aquavella. Here, it is standard to add a prophylactic antibiotic, he adds, but antibiotics are not indicated with other treatments.

Another approach is the use of a mild steroid such as fluorometholone, loteprednol or, on occasion, 1% methylprednisolone. According to Dr. Aquavella, these will quickly heal any TSPK lesions. From there, the steroids should be slowly tapered over about a month or so. “Perhaps start with tapering the steroid to BID and then, ultimately, once or twice a week,” says Dr. Aquavella. “If there is no pressure response, some patients will require an occasional extra drop to keep the condition in check.”

Other options that have proven effective are cyclosporine in olive oil and tacrolimus, Dr. Aquavella says.

Etiology

The jury is still out on an exact cause for TSPK. Although viral mechanisms such as herpes simplex virus and varicella zoster virus have been suggested in early, limited studies, more recent research has not been able to show viruses as the culprit.1-4 “When TSPK lesions clear there is no sign of viral involvement,” says Dr. Aquavella. “Also, post-viral conditions often linger for longer periods.”

According to Dr. Aquavella, autoimmune processes have been long suspected to play a role in TSPK, and research shows that autoimmune diseases such as Sjögren’s syndrome have a positive association with the disease.5 Still, a definitive underlying cause for TSPK remains unknown.

A 53-year-old Hispanic female presented with intermittent ocular irritation and itchiness in both eyes for the past year. She does not use any drops. In addition, she reported transient episodes of dimming vision, and sometimes her vision even gets dark for several seconds. This started six months ago and happens about once a week. She felt this was occurring in both eyes.

Her medical history was significant for well-controlled hypertension. She was on amlodipine, atenolol, enalapril and lisinopril. She also reported having had brain surgery in the early 1990s in Nicaragua for removal of a frontal lobe tumor. She felt like she fully recovered from this operation and did not have any lasting effects.

On examination, her entering visual acuity with glasses measured 20/20 OU at distance and near. Confrontation visual fields were full-to-careful finger counting in both eyes. Her ocular motility testing was normal, and the pupils were equally round and reactive without an afferent pupillary defect. The anterior segment was unremarkable. Intraocular pressures measured 19mm Hg OU.

Her dilated fundus exam showed changes (Figure 1). An OCT and visual field was also obtained and is also available for review (Figures 2).

Take the Retina Quiz
1. How would you describe the patient’s symptoms?
   a. Usual presbyopia changes.
   b. Transient ischemic attacks.
   c. Amaurosis fugax.
   d. Visual aura.

2. What is the significant finding seen in this patient?
   a. Advanced optic nerve cupping.
   b. Nerve fiber layer defects.
   c. Thrombin in the retinal vein.
   d. Cholesterol plaque in the retinal artery.

3. How would you classify these changes?
   a. Calcific.
   b. Thrombin.
   c. Cholesterol.
   d. Fibrin.

4. What is the correct diagnosis for this patient?
   a. Whitman plaque.
   b. Hollenhorst plaque.
   c. Branch retinal artery occlusion.
   d. Advanced glaucoma with nerve fiber layer defect.

5. How should this patient be managed?
   a. Observation.
   b. Immediate referral for carotid evaluation.
   c. Begin treatment with prostaglandin eye drops for IOP control.
   d. Visual field and OCT testing.

For answers, see page 98.

Diagnosis
Our patient is describing symptoms consistent with amaurosis fugax, which is a transient loss of vision in one eye. It is usually fleeting, lasting seconds to minutes and results from transient retinal ischemia. It is most commonly due to an embolism or plaque from the ipsilateral carotid artery, but can occur from a number of other causes associated with transient ischemic attacks.¹

If the plaque is small enough, it will continue to pass through the retinal circulation with zero to minimal noticeable effects on vision.
Larger plaques can cause a complete blockage of the artery, which in turn can lead to severe ischemia and permanent loss of vision. If this occurs at the level of the optic nerve or more posterior, a complete central retinal artery occlusion can occur. If the blockage occurs along one of the branches of the central retinal artery (CRAO), a branch retinal artery occlusion can occur. Though vision loss may occur with a branch retinal artery occlusion (BRAO), it is much less devastating than a central retinal artery occlusion.

So, what is going on with our patient? On the fundus exam of the right eye, an obvious plaque can be seen along the superior temporal artery. The presence of a plaque is a clear “marker” for possible carotid artery disease or heart disease. Is this what is causing our patient to have amaurosis fugax?

**Discussion**

Three main types of retinal arterial emboli exist; Hollenhorst plaques, calcific emboli and platelet-fibrin plaques.

Hollenhorst plaques, perhaps the most common, are highly refractile and often glisten when the light from the indirect lens shines on the plaque. Hollenhorst plaques represent cholesterol emboli that originate from atheromatous lesions in the ipsilateral carotid artery or from the aorta. They usually become lodged in the bifurcation of one of the branches of the central retinal artery. In most instances, patients are asymptomatic and are discovered as part of a routine exam.

Calcific plaques are not refractile and have a dull white appearance. They tend to be larger and can be associated with heart valve or aorta calcification.

Platelet-fibrin emboli also dull-white in appearance but are more elongated. They are also associated with carotid artery disease.

The plaque seen in our patient did have a refractile, glisteny appearance, so it is likely a Hollenhorst plaque; however, it is difficult to determine if our patient's symptoms are a result of the Hollenhorst plaques passing through her retinal circulation. What's more, even though we only see a single plaque, she had others. Most patients with Hollenhorst plaques are asymptomatic; clearly our patient has symptoms and this deserves serious attention.

Carotid artery disease is one of the major causes of stroke. Stroke itself is one of the most common causes of mortality and severe disability in adults. Patients who have had a stroke will often have preceding ischemic events such as transient ischemic attacks, amaurosis fugax, or both. For that reason, patients with these symptoms need a full cardiac work up, as well as carotid artery studies such as carotid duplex Doppler ultrasound. Hypercoagulable testing (antiphospholipid antibodies and homocysteine levels) and angiography may be helpful in certain cases.

Our patient was made aware of our findings and was referred to her primary care physician that same day. We sent her there with a letter explaining our findings and was told that if she was not able to see her primary care physician that day, she should go within the week and that, if she experienced any further symptoms, she should go to the local emergency room.

She was seen one month later for follow up of her symptoms as well as for a glaucoma work-up (IOP, visual fields and OCT). She had not experienced any more amaurosis. She had seen her primary care physician and carotid Doppler studies were performed and were normal. She scheduled an echocardiogram in a few weeks' time. Her OCT was normal but she did have a corresponding visual field defect in the same area of the Hollenhorst plaque.

She returned two years later, still doing fine and had not had any further symptoms.

---

It all began with an optometry resident treating a 34-year-old woman for a painful swelling of her eyelid of four days’ duration. The pain had been increasing, forcing her to seek treatment. She had a small, internal focal swelling of her right upper eyelid, which was painful to palpation. The patient reported no trauma, use of new cosmetics or other products and didn’t recall being bitten by any insect. She was not ill and had no fever. After a comprehensive analysis, a resident correctly diagnosed a hordeolum and after educating the patient, had recommended hot, moist compresses and digital massage along with over the counter analgesics. After dismissing the happy patient, the resident shared the case and asked the opinions of several colleagues. Much to her surprise, each person had a different approach to treating patients with hordeola and each assured her that their approach “worked every time,” leaving the young doctor more confused.

Hordeola are commonly encountered in clinical practice, and many clinicians have their own personal approach to managing this rather benign condition. A patient with a hordeolum will present with an acutely swollen and edematous upper or lower eyelid. Visual function will be normal, unless the swelling is so profound that it induces a mechanical ptosis or astigmatism. There may be an associated inflammation of the conjunctiva with possible mucopurulent discharge from the glands at the margin of the eyelid. The eyelid will be painful to palpation, sometimes extremely so. There may be an associated pustular pimple-like lesion at the epidermis or, more commonly, at the lid margin. Often, patients will present with a significant blepharitis concurrently. Hordeolum is one of the most common acquired lid lesions in children.1

Development
The sebaceous glands of the lids, the meibomian glands and glands of Zeis are the sites of origin of hordeola. Twenty to 30 meibomian glands are located in the upper lid and 10 to 20 in the lower lid and in the tarsal plate. Unlike the meibomian glands, the glands of Zeis are associated with the eyelash follicles. These glands produce the superficial lipid layer of tears.1

A hordeolum represents a bacterial infection of these glands of the eyelid with subsequent abscess formation. This will be associated with a tender, inflamed swelling at the lid margin, often pointing anteriorly through the skin. If the deeper glands are involved, the hordeolum is considered internal and is less well circumscribed in appearance. In these cases, you will see more diffuse swelling of the tarsus. The lesion may enlarge and discharge either through the tarsal conjunctiva or through the skin. The literature shows cases of multiple recurrent hordeola associ-
ated with selective IgM deficiency. Studies also show abnormal triglyceride fatty acid composition with chronic blepharitis associated with hordeola.

Progression
The most commonly encountered organisms in hordeola are *Staphylococcus aureus* and *Staphylococcus epidermidis*. Acute and chronic inflammation associated with hordeola may result in a hard retention cyst known as a chalazion, especially if it is improperly treated. Spread of infection to neighboring glands or other lid tissue anterior to the tarsal plate may lead to the formation of a preseptal cellulitis. While uncommon, hordeola can produce ocular surface disruption as a thickened lid rubs against the cornea and conjunctiva during blinking.

Treatment
So, what are the options for treating an acute, painful hordeolum? First, remember that the infection is generally self-limiting and will often resolve within a week or two with spontaneous drainage of the abscess. Traditionally, the most common treatment involves the use of hot compresses with digital massage along with topical antibiotic solutions and ointments. Some advocate for oral antibiotics instead of topical treatment due to better absorption with subsequently higher therapeutic concentrations. Incision and curettage may also be helpful.

In a survey study involving 501 ophthalmologists, warm compress usage was employed by 92% of respondents. Additionally, combined topical and oral antibiotics usage was employed by 92% of respondents. Only 2.4% of respondents used oral antibiotics alone, and 4.2% used no oral antibiotics. Incision and curettage was performed only in cases with a flocculated mass, larger lesions or if requested by patients. First choice antibiotics were a combination of topical neomycin, polymyxin and gramicdin eye drop, chloramphenicol eye ointment and oral dicloxacillin.

Clearly, doctors have numerous approaches to handling this common infection, likely with varying success. When there is debate about best treatment and practices, it is always best to look at evidence-based medicine. Randomized, controlled clinical comparative trials give the best evidence on how to manage patients when there are numerous options and speculations. Unfortunately, hordeolum, though common, is considered benign and self-limiting and, as such, has garnered little in the way of clinical research. Reviews of the best available literature did not find any evidence for or against the effectiveness of nonsurgical interventions for the treatment of an internal hordeolum. The few references specific to treating acute internal hordeolum were reports of interventional case series, case studies or other types of observational study designs and were published more than 20 years ago.

It is our feeling that topical antibiotic therapy could include dicloxacillin 250mg PO Q6h; erythromycin 250mg PO QID; or amoxicillin 500mg PO TID for 10 days. Cefalexin 500mg BID for seven to 10 days is also an excellent, economical choice. Cold compresses will help to suppress inflammation and pain, while warm compresses will enhance pointing and drainage.

Clearly, optometrists have several acceptable methods to manage patients with hordeola. Each probably has varying degrees of success and therapies can be patient specific.

In our resident’s case, she walked away still confused with yet another opinion. But we assured her that our approach “works every time.”


Treating a hordeolum, such as the one seen in this patient’s lower lid, includes using hot compresses, digital massage, topical antibiotics and ointments.
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In June 2012, a 57-year-old Caucasian female presented to the office with complaints related to headaches and periorcular discomfort that progressed over several months. She was a low hyperope wearing a hyperopic presbyopic spectacle prescription that was about five years old at the time.

She was taking no medications and reported no allergies to medications. Refractively, she had an expected change in her spectacle prescription of increased hyperopia and presbyopia, best corrected to 20/20 OD, OS, OU.

A slit lamp exam of her anterior segments was unremarkable. Applanation tensions at that visit were 15mm Hg OD and OS at 10:39am. Slit lamp estimation of her anterior chamber angles demonstrated wide-open angles in both eyes.

Through dilated pupils, her crystalline lenses were clear bilaterally. Her cup-to-disc ratios were 0.65 x 0.75 OD and 0.65 x 0.65 OS, with discs that were average size. There was slight thinning of the temporal rim in both eyes, with a common appearance of sloping temporal margins of the neuroretinal rim into the optic cup. Her macular, vascular and retinal evaluations were normal in both eyes.

I asked her if anyone had mentioned that she had some characteristics to her optic nerves that put her at risk for glaucoma, and she said no. She also denied a family history of glaucoma. I explained my findings of suspicious discs and, since she was dilated, I took the opportunity to obtain stereo optic nerve images. She was scheduled for a full glaucoma work-up in the next few weeks.

**Evaluation**

As scheduled, she presented for the glaucoma work-up, which included pachymetry, morning IOP readings, gonioscopy, threshold visual fields and Heidelberg retina tomograph (HRT 3) and optical coherence tomography (OCT) imaging of the optic nerves. At this visit, pachymetry readings were 478µm OD and 479µm OS. Applanation tensions at 9:15am were 16mm Hg OD and 17mm Hg OS. Gonioscopy demonstrated wide-open angles with minimal trabecular pigmentation, a flat iris-to-angle approach and no angle abnormalities.

Threshold standard automated perimetry (SAP) field studies were clear and reliable in both eyes. HRT 3 imaging shows a distinct change in the IT neuroretinal rim of our patient’s right eye, first noted in 2015.
nerve imaging confirmed the clinical appearance of the optic nerves with a large cup, a thinned temporal neuroretinal rim and Moorfields Regression Classification as statistically borderline optic nerves in both eyes. The RNFL circle scan of both eyes was statistically normal. Given the findings at that time, I diagnosed her as a glaucoma suspect only, as there was no frank evidence of glaucoma either structurally or functionally. I asked her to follow up in a year.

Over the subsequent years, she presented annually as directed, and essentially all findings remained stable, though we did substitute threshold flicker-defined form visual fields for SAP fields, looking specifically for early field loss. These too remained stable. However, in the summer of 2015, there appeared to be a change in the inferotemporal neuroretinal rim of the OD from baseline, while all other indices remained stable. Given that her imaging to this point was stable and consisted of high quality images, this change was worthy of further investigation. Accordingly, I asked her to return in four months for repeat nerve imaging. At that visit in December 2015, the structural defect had worsened in the right eye, indicating a conversion to early glaucoma. Her imaging of the left eye remained stable insofar as the HRT 3 images were concerned. And her OCT RNFL circle scans also remained stable. Accordingly, she was initially managed with 0.5% timolol QAM to the right eye, and observation of the left eye. Post treatment IOP readings in the right eye have averaged 11mm Hg to 12mm Hg, and those of the unmedicated the left eye have remained in the midteen range.

**Discussion**

Once the patient converts from a glaucoma suspect to a patient needing intervention, I usually see them a bit more frequently, and as such, I followed the same protocol with this patient. At this point in our management, each visit is tasked with determining whether the patient remains stable or not, which, for glaucoma, requires an evaluation of both the structural and functional aspects of the eye.

One of the advantages of having adequate instrumentation to evaluate these patients is that one can see, rather readily, when it occurs, change over time. One of the disadvantages to having a variety of ways...
to evaluate stability over time is that sometimes the quantity of information becomes overwhelming. With too much data in our hands, it’s easy to see situations where busy clinicians might not evaluate all of the pieces to the diagnostic puzzle and miss significant changes.

So how much technology is too much? And how much is too little? Those answers are going to vary from practice to practice, and there are no concrete answers I can give to you. But this case highlights why having multiple technologies can help you to offer personalized care, if you take the time to sift through the data. The radial OCT image on page 89 shows one of the more common presentations of an optic nerve with temporal sloping margins, especially in the IT sector where the HRT 3 image was showing changes.

When we look at OCTs of change, we can look at several locations. Most optometrists probably first look at the peripapillary RNFL circle scan. Now that we are scanning several different RNFL circle scans, we can detect changes that are occurring at various distances from the optic nerve, and not just the standard 3.5mm circle scan.

Finally, peeling back even more detail, we can take a look specifically at the IT sector of the OCT Bruch’s membrane opening (BMO) map for change. Given the ability to now look at BMO in various sectors of the optic nerve, and the statistical capability of looking for change over time in these individual sectors, we can now look at not only the macular and peripapillary RNFL areas, but also at the neuroretinal rim. Remember, in this case, she showed signs of conversion in the IT sector of the neuroretinal rim as seen on the HRT 3. This same sector, as seen on the BMO OCT scan, over two visits, shows complete stability.

Changes

The key diagnostic piece to this puzzle is that the initial change occurred in the IT sector of the OD, as measured by one instrument. Subsequent measurements of the same sector of the neuroretinal rim with a different instrument (OCT) show no change. And it is in this area where she would most likely show continued progression if it occurred.

Instruments change. Technology changes. Optic nerves change, if they are worsening. Knowing where to look is half the battle. Looking through all the data, though challenging, is what makes for excellent clinical care.
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Minimally invasive glaucoma surgeries (MIGS) are quickly earning a place in the glaucoma treatment regimen. Their demonstrated efficacy, along with improved safety and faster recovery compared with traditional surgeries such as trabeculectomy, have made MIGS a good option for patients showing glaucomatous damage, progression or even those who want to reduce their dependence on topical therapy. Many MIGS target the traditional outflow pathway, either by bypassing or removing the trabecular meshwork or by reconstructing Schlemm’s canal. But two recently approved devices have different approaches.

Follow a New Path

The CyPass Micro-Stent (Alcon), a fenestrated stent that connects the anterior chamber to the supraciliary space, is used with cataract surgery to reduce intraocular pressure (IOP) in patients with mild to moderate primary open-angle glaucoma. Using the corneal incisions already created for cataract extraction, the surgeon places the stent posterior to the scleral spur. The curved design allows natural passage into the supraciliary space, while retention rings are designed to keep the stent in place. In the COMPASS trial, which compared cataract extraction plus CyPass insertion with cataract extraction alone, the group receiving CyPass showed a 7.4mm Hg reduction in mean IOP, compared with 5.4mm Hg with only cataract extraction at two years.

Unlike the CyPass, the Xen 45 gel stent (Allergan) mimics the outflow pattern of traditional glaucoma procedures by draining aqueous into the subconjunctival space, but with the less invasive ab interno approach. This stent is indicated as a standalone procedure or with cataract surgery for patients with open-angle, pseudoexfoliative or pigmentary glaucoma who failed maximum topical therapy or other filtering procedures. The Xen, preloaded in a single-use injector, is inserted through the trabecular meshwork, creating a scleral channel through which the stent connects the anterior chamber to the subconjunctival space. A clinical trial of 65 patients demonstrated a drop in IOP from a mean of 25.1mm Hg preoperatively to 15.1mm Hg postoperatively, with a reduction in medications from 3.5 before the procedure to 1.7 after.

Postoperative Considerations

As with standard cataract surgery, patients will be prescribed a combination of antibiotics, steroids and nonsteroidal anti-inflammatory drugs after the procedure. Glaucoma drops may be adjusted or discontinued depending on the resulting postoperative IOP.

Gonioscopy is critical in the management of any MIGS procedure and is especially useful during pressure spikes to monitor for proper stent placement, potential iris obstruction or hemorrhage through the stent. Patients should also be watched closely in the postoperative period for hyptonia, hyphema, iritis and corneal edema. Patients with the Xen stent should also be observed for any flattening of the subconjunctival bleb, as needling of the bleb was required in 32.3% of patients.

MIGS are an exciting addition to the glaucoma management regimen. These two new procedures in particular provide many patients yet another avenue for preventing glaucomatous damage.

Dr. Kruthoff practices at Virginia Eye Consultants in Norfolk, Va., with a focus on perioperative glaucoma care.

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ASSISTANT PROFESSOR POSITIONS: CONTACT LENSES, PRIMARY CARE, LOW VISION, AND PEDIATRICS
(Full-time non-tenure track faculty positions for the Chicago College of Optometry)

RESPONSIBILITIES: Candidates are expected to be highly knowledgeable in the field of Cornea and Contact Lenses, Primary Care, Low Vision, or Pediatrics and can develop and teach courses and/or laboratories in the subject area. The candidate must also be able to provide direct patient care and clinical instruction to professional students as well as residents, and be involved in interdisciplinary practice with other educational professionals.

Candidates must be willing to actively participate in curricular assessment, professional development, student counseling and service activities within the college, university and the scientific community. Successful candidates are also expected to be involved in research and scholarly activities, and have a sincere commitment to optometric education, community service and patient care. Primary duties include, but are not limited to:

- Developing and delivering lectures and/or laboratories for related areas, as assigned;
- Embracing and enhancing the didactic philosophies in the O.D. program;
- Maintaining and expanding the high quality clinical practice environment for optometry students on rotation;
- Precepting students on clinical rotation at the Midwestern University Eye Institute;
- Helping to maintain and grow the state of the art optometry program with a strong interdisciplinary focus that meets the needs of patients in the surrounding community, is efficient, patient friendly, and cost-effective;
- Working closely together with all optometry and ophthalmology faculty to provide a complete range of eye and vision care services;
- Participating in leadership roles in state, regional, and national optometry organizations;
- Participating on College and University committees, as assigned;
- Participating in College and University service activities.
- Engaging in research and scholarly activity, including presentations at scientific meetings, research, and publication in peer reviewed journals sufficient to qualify for academic advancement in a non-tenure track position.

QUALIFICATIONS: Candidates must possess a Doctor of Optometry degree from an ACOE-accredited institution, must have completed an ACOE-accredited residency, and must be eligible for an optometric state license in the state in which the college is located. Primary eye care clinical expertise is also required.

CONTACT INFORMATION: Contact information: Interested applicants should apply online at www.midwestern.edu and include curriculum vitae and letter of interest specifying the position and college that he/she wishes to be considered for. Inquiries may be directed to Dr. Melissa Suckow, Dean, Midwestern University: msuckow@midwestern.edu.

Midwestern University is an Equal Opportunity/Affirmative Action employer that does not discriminate against an employee or applicant based upon race, color, religion, gender, national origin, disability, or veterans status, in accord with 41 C.F.R. 60-1.4(a), 250.5(a), 300.5(a) and 741.5(a).

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May 2018

- **2-10.** CE in Italy/Europe. Hotel Torbräu, Munich, Germany. Hosted by: James Fanelli. Key faculty: Joseph Pizzimenti, Lorraine Lombardi, Leonard Messner, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinitaly.com, call (910) 452-7225 or go to www.ceinitaly.com.
- **5-7.** CE in Italy/Europe. Kongresshaus, Heidelberg, Germany. Hosted by: James Fanelli. Key faculty: Joseph Pizzimenti, Lorraine Lombardi, Leonard Messner, James Fanelli. CE hours: 12. For more information, email James Fanelli at jamesfanelli@ceinitaly.com, call (910) 452-7225 or go to www.ceinitaly.com.
- **6.** 2018 Coding Update. University of Missouri St. Louis JC Penney Conference Center, St. Louis, MO. Hosted by: University of Missouri St. Louis College of Optometry. Key faculty: John McGreal. CE hours: 4. For more information, email Erin Schaeffer at mcgreal@umsl.edu, call (314) 516-5615 or go to optometry.umsl.edu/CE Courses/index.html.
- **17-20.** New Technologies and Treatments in Eye Care Orlando 2018. Disney’s Yacht Club, Orlando, FL. Hosted by: Review of Optometry. Key faculty: Paul Karpecki. CE hours: 18. For more information, email reviewmeetings@jobson.com, call (866) 658-1772 or go to www.reviewofoptometry.com/orlando2018.
- **17-20.** Pennsylvania Optometric Association Spring Congress. Seven Springs Mountain Resort, Seven Springs, PA. Hosted by: Pennsylvania Optometric Association. CE hours: 22 total, 20 per OD. For more information, email Ilene Sauertieg at ilene@sauertieg.org, call (717) 233-6455 or go to www.poaeyes.org.
- **30.** The Eleventh Central Jersey Optometric Seminar. CentraState Medical Center, Freehold, NJ. Hosted by: Optometry on West 44th. Key Faculty: Walter Whitley. CE hours: 4. For more information, go to www.optometryonwest44th.com.
- **31-June 3.** Oregon’s Meeting. Mt. Bachelor Village Resort, Bend, OR. Hosted by: Oregon Optometric Physicians Association. Key faculty: Tracy Doll, April Jasper, Derek Cunningham, Fraser Horn. CE hours: 15. For more information, email Lynn Olson at lynne@oregonoptometry.org, call (800) 922-2045 or go to www.oregonoptometry.org.

June 2018

- **4-7.** Indian Health Service Biennial Eye Care Meeting. Marshall B. Ketchum University (MBKU), Fullerton Campus, Fullerton, CA. Hosted by: MBKU Southern California College of Optometry and Indian Health Service. CE hours: 25. For more information, email Antoinette Smith at asmith@ketchum.edu, call (714) 872-5684 or go to www.ketchum.edu/ce.
- **6-10.** Art & Science of Optometric Care. A Behavioral Perspective. Nova Southeastern University, Fort Lauderdale, FL. Hosted by: The Optometric Extension Program Foundation. Key faculty: John Abbondanza. CE hours: 35. For more information, email Karen Rudor at karen.ruder@boep.org, call (410) 561-3791 or go to www.boep.org.
- **7-10.** Utah Optometric Association (UOA) Annual Congress. The Zermatt Resort, Midway, UT. Hosted by: UOA. Key faculty: Bruce Onofrey. CE hours: 20. For more information, email Alyssa White at alyyssa@utaheyedoc.org, call (801) 364-9103 or go to www.utaheyedoc.org.
- **8-10.** Everything Therapeutic: Houston. University of Houston College of Optometry (UHCO), Houston. Hosted by: UHCO. Key faculty: Bruce Onofrey. CE hours: 24. For more information, email optce@central.uh.edu, call (713) 743-1900 or go to ce.uh.edu.
- **8-10.** Northern Educational Escape. Delta Hotels, Quebec City, Canada. Hosted by: Optometric Education Consultants. Key faculty: Greg Caldwell, Joseph Pizzimenti, Barry Fraenus, Rím Mákhliouf. CE hours: 15. For more information, email Vanessa McDonald at optoce@gmail.com, call (954) 262-4224 or go to www.optometrizedu.com.
- **20-24.** AOA/Review of Optometry’s Meeting. Colorado Convention Center, Denver, CO. Hosted by: American Optometric Association and American Optometric Student Association. CE hours: 215 total, 43 per OD. For more information, email csampmani@aoa.org, call (314) 983-4124 or go to optometristsmeeting.org.

July 2018

- **1-8.** Tropical CE Ocean Reef 2018. Ocean Reef Club, Florida Keys. Hosted by: Tropical CE. Key faculty: Diana Shechtman. CE hours: 20. For more information, email Stuart Autry at sautry@tropicalce.com, call (281) 808-5763 or go to www.tropicalce.com.
- **8-18.** Therapeutic Pharmaceutical Agents Certification/Board Review Course. Nova Southeastern University College of Optometry (NSUCO), Fort Lauderdale, FL. Hosted by: NSUCO. Key faculty: Joseph Sowka, Julie Tyler, Chandra Mickleis, Diana Shechtman, Sherrol Reynolds. CE hours: 100. For more information, email Vanessa McDonald at nceoa@nova.edu, call (954) 262-4224 or go to optometry.nova.edu/ce/index.html.
- **12-15.** July Advanced Procedures. Oklahoma College of Optometry (OCO) Academic Wing, Tahlequah, OK. Hosted by: OCO. Key faculty: Nate Lighthizer, Richard Castillo, Joseph Shelter, Doug Penisten. CE hours: 32. For more information, email Callie McAtee at mcaatere@nsuk.edu, call (918) 316-3602 or go to optometry.nsuok.edu/continuingeducation.

To list your meeting, please send the details to:

- **Michael Iannucci**
  - Associate Editor
  - Email: miannucci@jobson.com
  - Phone: (610) 492-1043

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There Will Be Blood

By Andrew S. Gurwood, OD

History
An 81-year-old Caucasian female reported to the office with a chief complaint of a red and painful left eye of two weeks duration. The patient had been to multiple eye doctors previously for herpes simplex virus (HSV) keratopathy and was treated for a non-healing epithelial defect with active HSV keratitis and subsequent elevated pressure in the left eye. Her ocular history was also remarkable for keratoconjunctivitis sicca in both eyes, for which she had been using artificial tears and erythromycin ointment in both eyes, and half-strength dose of fluorometholone drops in the right eye. The patient developed HSV keratitis about 30 years ago with multiple episodes of exacerbation and remission. Her systemic history was remarkable for herpes simplex virus treated with oral valacyclovir periodically during outbreaks. She denied any allergies.

Diagnostic Data
Her best-corrected visual acuities were 20/50 OD and 20/200 OS. Her external examination was grossly intact. The pertinent anterior segment findings in the left eye are demonstrated in the photographs. Biomicroscopy of the right eye showed evidence of anterior basement membrane dystrophy with old inactive corneal opacities. The anterior chamber of the right eye appeared to be deep and quiet with a well-placed posterior chamber intraocular lens. Goldmann intraocular pressures were measured to be 18mm Hg OD and 20mm Hg OS. Dilated funduscopy was within normal limits, both eyes, revealing slightly high asymmetric cup-to-disc ratios measuring 0.5/0.5, 0.55/0.6, OD, OS respectively with normal peripheries.

Your Diagnosis
Does the case presented require any additional tests, history or information? What steps would you take to manage this patient? Based on the information provided, what would be your diagnosis? What is the patient’s most likely prognosis? To find out, please visit us online at www.reviewofoptometry.com.
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REFERENCES:
1. Results from a 7-investigator, multi-site 2-week study of Bausch + Lomb ULTRA® for Astigmatism contact lenses on 157 current soft contact lens wearers.

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References:
6. In vitro study over 16 hours to measure wetting substantivity, Alcon data on file, 2015.

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