Slit Lamp Essentials: How to Perform Lacrimal Irrigation, Page 72

Diagnostic Skills & Techniques

» SELF-TEST: DIFFERENTIAL DIAGNOSIS OF RETINAL DISEASE, PAGE 34

» DON’T OVERLOOK THE EYELIDS: WHAT A THOROUGH EXAM CAN REVEAL, PAGE 44

» POINT-OF-CARE TESTING: A LAB IN THE PALM OF YOUR HAND?, PAGE 52

» Earn 2 CE Credits: THE URGENT AND CHRONIC CAUSES OF DIPLOPIA, PAGE 63

» PLUS — CASE REPORT: IDIOPATHIC JUXTAFOVEAL RETINAL TELANGIECTASIS, PAGE 82
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AOA to Oppose 1-800’s Pricing Bills in 14 States

The AOA is sending out a rallying cry to all state optometric associations and optometrists to combat a set of 1-800-Contacts backed legislative bills that would ban unilateral pricing policies (UPP) for contact lenses.

“Although there are 14 states that are being immediately targeted by 1-800-Contacts, it’s really an attack on our profession and the essential care we provide to our patients,” says AOA president David A. Cockrell, OD. “The AOA and state associations have mobilized quickly to fight back, but we need every optometrist and optometry student to get involved and help get our message out to legislators and to the public.”

Bills are currently pending or in the works in Arizona, California, Florida, Idaho, Illinois, Louisiana, Minnesota, Mississippi, New York, Oregon, Rhode Island, Tennessee, Utah and Washington, according to the AOA.

At press time, an anti-UPP bill in Mississippi has already died in the legislature, a bill in Idaho has been approved in committee, and a bill in Utah—the home state of 1-800-Contacts—has been approved by the state Senate and introduced into the state House of Representatives.

Officials from 1-800-Contacts told Congress last July that the decision made by contact lens manufacturers to set price minimums on specific brands of their contacts would raise prices for consumers and prohibit consumers’ ability to “shop around” for their contact lenses.

Sen. Deidre M. Henderson called for a vote in the Utah Senate in favor of a bill that bans unilateral pricing policies in the state. She acknowledged that the American Optometric Association was opposed to the bill. The vote passed 21 to 8 in the Senate and, at press time, was introduced to the Utah House.

A recent clinical trial reported in The New England Journal of Medicine compared three drugs for diabetic macular edema—Eylea (aflibercept, Regeneron), Avastin (bevacizumab, Genentech) and Lucentis (ranibizumab, Genentech)—and found all three resulted in similar average improvement when initial vision was 20/40 to 20/32. However, the study found Eylea provided greater visual improvement on average when vision was 20/50 or worse at the start of the trial. Investigators found no major differences in the safety of the three drugs.

A novel intravitreal injection of an immunomodulatory protein provided a long-lasting anti-inflammatory effect in mice with a form of age-related macular degeneration, according to molecular genetics researchers at the University of Florida College of Medicine. The researchers also found the drug works for uveitis and could ultimately be used for various other inflammatory eye diseases in humans.

In the study, published in the January issue of Human Gene Therapy, the researchers used an adeno-associated viral vector to inject the inflammation-blocking protein M013 into the eye. Additional studies are needed before the therapy will be tested in humans, the researchers say.

People with Parkinson’s disease have significantly worse vision for low-contrast images at both near and far distances, according to a recent study in the Journal of Parkinson’s Disease. Even for high-contrast images, vision of patients with Parkinson’s disease was deficient at far distances.
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1-800 Backs Bills Against UPP Pricing

(Continued from page 4) doctors,” the AOA state government relations committee wrote in a memo to its state affiliates. The AOA says optometrists should defend against 1-800’s allegations by bringing the following key points to the attention of their state legislators as well as concerned doctors and patients:

• Contact lenses are medical devices and optometrists need to be involved in patient use. Any review of the scientific literature or even the lay press confirms the fact that a patient’s vision and overall health are at risk with improper contact lens wear, care or fit.

• Doctors recommend specific lenses for medical and safety reasons. Substitution of a prescribed medical device like a contact lens is wrong and poses unintended health risks to the patient.

• More needs to be done to target unscrupulous contact lens sellers and profiteers. The legislature should empower the state optometry board with the tools it needs to target contact lens sellers who threaten public health and hold them accountable for the harm they cause.

Sen. Deidre M. Henderson, who introduced the anti-UPP legislation in Utah, acknowledged to the state Senate that, “The American Optometric Association does not like this bill … But their arguments that this bill will weaken the quality of eye health care standards and undermine the doctor-patient relationship doesn’t make any sense to me.” She added, “[This bill] does not in any way change or hinder [optometrists’] ability to care for their patients at all.”

Switch to Generic Glaucoma Drug Boosts Adherence by 25%, Study Says

Eye care professionals looking to improve their patients’ adherence to glaucoma medications might have a new—and surprisingly simple—trick up their sleeves. In a recent longitudinal cohort analysis, the results of which were published online in Ophthalmology, researchers found that switching to a generic drug improved adherence by as much as 25%.1 Medication adherence is a major problem, considering more than half of glaucoma patients do not take their medications as prescribed.2 The cost of glaucoma medication could be part of the problem, say researchers from the University of Michigan Medical School and University of Michigan College of Pharmacy. “Individuals’ out-of-pocket costs for glaucoma medications can exceed $100 per month, and the high drug cost may deter patients on a
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Adherence Improves with Generic

(Continued from page 6)
tight budget from consistently buy- ing and taking their medications as prescribed,” says Joshua D. Stein, MD, associate professor of oph- thalmology and visual sciences at the University of Michigan Kellogg Eye Center and lead author of the study. “If clinicians suspect that a patient is struggling with medication adherence, it may be a good idea to switch them from a brand name to a generic drug.”

The 2011 release of generic latanoprost gave patients a cheaper option—and researchers the op-portunity to find out if cost was in fact a motivating factor.

Dr. Stein and colleagues ex- amined claims data from 8,427 glaucoma patients for the 18 months before and after generic latanoprost became available. They found that medication adherence improved on average among all pa- tients who switched to the generic, but the rate of adherence differed among certain subgroups. Those who were on bimatoprost prior to switching to generic latanoprost in- creased adherence from an average 47% to 61%; those who switched from travoprost increased adher- ence from 43% to 54%.

The study also showed the sub- set of black patients who switched to the generic drug had a substan- tial improvement in adherence compared to blacks who remained on brand-name products.

“It’s reassuring to find that switching patients to more afford- able, generic drugs could be an effective solution for a subgroup of patients who have difficulty with adherence,” Dr. Stein says.

One finding that the researchers could not explain: A considerable number of patients discontinued glaucoma drug use altogether when generic latanoprost became available.


Drug-Delivering Nanowafer Shows Better Efficacy Than Topical Drops

Researchers at Baylor College of Medicine have developed a new nanowafer that could one day revolutionize how you care for patients in need of topical drug therapy.

Investigators set out to see if a slow-release delivery system could improve treatment for corneal neovascularization (CNV) due to ocular burns in mouse eyes. They used a nanowafer (a tiny circular disc 2mm in diameter and 100µm thick) made of polyvinyl alcohol (PVA), a water-soluble polymer used in artificial tears. The research- ers found PVA to be nonstimula- tory and nonimmunogenic—an important factor when trying to deliver drugs without disrupting the cornea’s natural healing process. The polymer also dissolves after the predetermined period of drug release.

Results revealed a nanowafer loaded with 5µg of the antiangio- genic drug axitinib, delivered once a day, was more effective in inhibiting CNV compared with axitinib eye drops delivered twice a day (10µg total). After 10 days of treatment, the nanowafer-treated cornea closely resembled the healthy uninjured cornea.

“Development of a nanowa- fer drug delivery system that can be readily instilled on the ocular surface by the patient’s fingertip without any clinical procedure will be not only very convenient but also most desirable for treating eye injuries, infections, chronic dry eye, glaucoma and other ocular inflammatory conditions,” the researchers concluded.

Also, they’re hopeful this device can move through human clinical trials in a timely manner, consider- ing the materials and drugs they used in this study are already in clinical use.

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A thorough eyelid examination can be critical to keeping your patients healthy. Here’s what you should look for.

By Christine Sindt, OD

Point-of-care testing helps take the guesswork out of diagnosis by bringing data into the clinic—quickly.

By Blair Lonsberry, OD

Double vision can originate from many conditions. Learn to recognize when this presentation is an emergency and when it’s indicative of a long-term issue.

By Jim Williamson, OD

A middle-aged patient was seeing spots, prompting an uncommon diagnosis of idiopathic juxtafoveal retinal telangiectasis.

By Erin S. Cooper, OD, Paul J. Gruosso, OD, and Joseph Miller, OD

Contact lenses, wellness and ocular diseases are hot topics this year—as is new technology to keep Expo attendees engaged. See what else is in store for VEE 2015.

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Seeing Double: The Urgent and Chronic Causes of Diplopia

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Essential Procedures at the Slit Lamp:

How to Make Your Patients Stop Crying

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Vision Expo East 2015: A Focus on Education

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Departments
Review of Optometry March 2015

4 News Review
16 Letters to the Editor
18 Outlook
All in a Day’s Work
JACK PERSICO
20 Chairsid
Must the ‘No Shows’ Go on?
MONTGOMERY VICKERS, OD
22 Neuro Clinic
Pale Disc Points to Trouble
MICHAEL TROTTINI, OD, AND
MICHAEL DELGIODICE, OD
26 Ocular Surface Review
Risky Business
PAUL KARPECKI, OD
30 Clinical Quandaries
Detached Staff, Detached Retina
PAUL C. AJAMIAN, OD
94 Coding Connection
Coding Point-of-Care Testing
JOHN RUMPAKIS, OD
96 Cornea + Contact Lens Q+A
Checks and Balances
JOSEPH P. SHOVLIN, OD
99 Retina Quiz
Gone Girl
MARK T. DUNBAR, OD
103 Review of Systems
AIDS in America: 30 Years Later
CARLO J. PELINO, OD, AND
JOSEPH J. PIZZIMENTI, OD
107 Therapeutic Review
The Throwback Thursday Option
JOSEPH W. SOWKA, OD, AND
ALAN G. KABAT, OD
111 Surgical Minute
Measure Twice, Cut Once
DEREK N. CUNNINGHAM, OD, AND
WALTER D. WHITLEY, OD, MBA
113 Product Review
114 Meetings + Conferences
115 Advertisers Index
117 Classifieds
122 Diagnostic Quiz
Salt and Pepper Fundus?
ANDREW S. GURWOOD, OD

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Letters to the Editor

Off-Label Use is Off Target?
I thoroughly enjoyed “Off Label, But On Target” (January 2015). Off-label uses are necessary for successful practice, yet they present us with dilemmas to navigate on a daily basis. We tread forward when perfect, or even imperfect, clinical science is lacking and the article shed great light on this issue.

One such off-label dilemma is the notion of topical Restasis (cyclosporine A 0.05%, Allergan) for uveitis. Dr. Lou Catania is one of our greatest thought leaders, and I respect his opinion and poster citation with great humility. However, the broad evidence for uveitis treatment with Restasis just does not add up. Yes, the drug is a potent anti-inflammatory agent via its white blood cell suppression. However, it has demonstrated poor ocular penetration even at a formulated 2% concentration, which is 40 times the concentration found in commercial Restasis. The 1990 study by BenEzra and colleagues showed no cyclosporine detectable in the anterior chamber at a 2% dosage concentration.2

Top uveitis specialist Stephen Foster, MD, addressed this issue at an American Academy of Ophthalmology lecture, asserting that low concentration and poor penetration via topical route would doom this modality to failure in treating uveitis. My own clinical experience indicates that Restasis patients suffer uveitis in the same manner as everyone else.

Given the logistical difficulties in getting Restasis for many patients, even on label, I fear that this treatment approach may delay definitive care and should be avoided until controlled studies contradict what is known to be true.

—William B. Potter, OD, Freehold, NJ

Dr. Catania responds:
Your letter regarding my comments about Restasis’ use in anterior uveitis and its penetration provides some important information about a topic that has confused me for years.

My history (and comments) regarding topical cyclosporine A (CSA) dates back to the 1990s and through the FDA approval process for Restasis in 2003. Given the positive history of topical CSA with canines (and other animal studies) in ocular inflammation as well as studies proving its value in human use (a list of citations available on request) and, finally, the FDA Restasis Application Research Report (NDA21-023) with pharmacological science and a supportive comment regarding its use in uveitis (“The effects of CSA has been demonstrated in several inflammatory conditions including autoimmune uveitis...”), I found (albeit anecdotally) that use of Restasis at higher than label-recommended dosages and with topical steroids seemed to shorten the course of a number of anterior uveitic inflammations treated.

Nonetheless, you raise a valid point that I have questioned myself about CSA’s ocular penetration. Some literature (e.g., BenEzra study) identifies it as a limitation and some (e.g., Nussenblatt et al.) dispute its relevance. I agree with you that further clinical evaluations on pharmacodynamic properties, concentrations and dosages vis-a-vis therapeutic ocular tissue levels with Restasis would be valuable.

1. Michelotti M, Shtein RM, Prabhu SS, Cooney T. Topical cyclosporine A 0.05% for recurrent anterior uveitis. Poster presented at American Society of Cataract and Refractive Surgery Annual Symposium. April 25, 2014; Boston, MA.

Sight Gags
By Scott Lee, OD
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All in a Day’s Work

few months ago, I got a piece of glass in my eye while repairing a window, causing a conjunctival laceration. It was a Saturday evening, and my eye was starting to bleed.

Instead of going to the ER, I called my optometrist. The office was closed, but she provided her cell number for emergencies. She took a detailed history over the phone, called in a prescription for an antibiotic and advised me of signs of infection to look for. If it worsened, she said, I should come in for an urgent visit on Monday morning. Then she went back to baking her Christmas cookies. No big deal; all in a day’s work.

It didn’t occur to me until later just how radical all that would have seemed not that long ago—after-hours call service by an OD, ocular injury risk assessment and triage, even the prescribing of a therapeutic pharmaceutical agent. But it all felt perfectly natural.

When I started in this field (24 years ago!), none of that would have happened. Most optometrists subsisted on vision care—refraction and dispensing were their bread and butter. Clinical care didn’t go much beyond routine screenings.

It’s exciting to see how much the profession has evolved and flourished, and truly gratifying to have played a small part in encouraging it through my efforts as a medical editor serving the field. Review of Optometry has always championed the advancement of optometrists’ clinical skills, and we continue that mission to this day.

In addition to launching a new column in January called Urgent Care that covers just such a scenario as I experienced myself, this month we kick off another forward-thinking department: Neuro Clinic. New columnists Michael Trotteni, OD, and Michael DelGiodice, OD delve into topics in neuro-ophthalmic care for the practicing optometrist in 2015. Their debut column discusses a case of suspicious optic disc pallor that led them to order an MRI, identify an aneurysm and refer to neurosurgery. From the OD to the OR! Impressive, most impressive.

Also this month, we emphasize diagnostic skills and techniques so that you can make the all-important call with confidence. “Outside of laboratory settings, hard data is hard to come by; we often must rely on clinical instincts and expertise,” Blair Lonsberry, OD, says in his article about point-of-care testing on page 52. Building up those instincts is this month’s issue focus.

Optometry has progressed by leaps and bounds in the last two decades, and Review is pleased to be in the vanguard with you. But we’re glad to stay grounded too, having also launched a column on tried-and-true refraction last month. “Thank you for the recent Focus on Refraction article,” a reader wrote. “We in the trenches see so few things on these subjects we deal with day in and day out, as we do everything we can to help our patients to see clearly and comfortably.” Vision is just as vital as eye health, and we honor that great strength while still helping the profession to grow.
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Must the ‘No Shows’ Go On?

Those two little words will drive me to an early grave. Until then, I will spend my time concocting elaborate revenge plots against these patients. By Montgomery Vickers, OD

As optometrists, one thing we all have in common, other than our ability to break dance, is that we all have no shows, what we list in my office as “NS.”

And these NS patients seem to travel in packs, because one day half your schedule is obliterated by the initials NS. Throughout my career, I have found NS patients fascinating, just like kidney stones are fascinating. They are often patients who don’t even pay a dime, as their examinations are my tax dollars at work. A statistically significant number of them called me for the appointment. Why set up an appointment yourself and then just not come?

We’ve tried everything: (a) sent them a bill, (b) sent them a bill with a personal note saying “this one time the accountant will NOT charge a fee because I know how busy life can be,” (c) told them we cannot reserve an appointment for them but would be happy to work them in the same day they call, (d) sent a handwritten apology that I was not in when they showed up for their appointment, and even (e) reappointed them for Friday (we are closed on Fridays). So far, nothing has helped, but I’ve only been working on solutions for 35 years.

Many, many “experts” have ideas about this very issue, such as:

1. Pre-appoint. If I had a nickel for every practice management guru who says “pre-appointing reduces no shows,” I’d be chillin’ in the Caymans right now. I do pre-appoint, and all things considered, it does help keep you booked up sometimes. On the other hand, patients routinely forget the Wednesday appointment they made on Monday. Can they remember the one they made 13 months ago? Apparently not!

2. Mail a reminder. We do. The postal service has an amazing record of reliable delivery, except, of course, for my appointment reminders. No shows “never got one.” I’m thinking of writing the reminders on a $10 check so when they cash them I can prove they actually did receive them!

3. Call and remind them. We call them the week before and the day before. Well, of course, that depends upon your definition of “we call them.” Back in the day, each family had one phone, and when you called a person answered, and you could actually reach them. Now, every person has a phone attached to his ear 24/7. We are the most connected generation in the history of the world, yet we can’t actually reach anyone.

Those two words, no show, have taken on a life of their own. When I see them, or even NS, I become almost ill. More like homicidal. I want revenge...

Don’t complain, No Show! You should have come to your appointment instead. And, if perchance you do happen to wander in for your appointment, you will ALWAYS be dilated—I think 50% atropine should do the trick. Don’t worry. It will wear off a couple weeks before you are due next year.

Do I sound bitter? One man’s bitterness is another man’s honesty. We all have to live and die by our decisions and YOU missed the appointment, NS. Not me … unless it was one of those appointments we rescheduled to a Friday when we are closed, in which case, NYAH-NA-NA-NA-NYAH, I wasn’t there!

Thank you all for allowing me to vent about no-shows. Now I gotta go. I have a patient waiting. Wonder what he’d say if I no showed?
SYMPTOMATIC VITREOMACULAR ADHESION (VMA)

SYMPTOMATIC VMA MAY LEAD TO VISUAL IMPAIRMENT FOR YOUR PATIENTS1-3

IDENTIFY
Recognize metamorphopsia as a key sign of symptomatic VMA and utilize OCT scans to confirm vitreomacular traction.

REFER
Because symptomatic VMA is a progressive condition that may lead to a loss of vision, your partnering retina specialist can determine if treatment is necessary.1-3

THE STEPS YOU TAKE TODAY MAY MAKE A DIFFERENCE FOR YOUR PATIENTS TOMORROW

A 43-year-old white female presented for a routine examination. Her only complaint was near vision difficulty related to presbyopia. She had a history of breast cancer with mastectomy, for which she was taking tamoxifen. Her other medications included Lupron (leuprolide acetate, AbbVie) and vitamin D. Her past ocular, family and social history was unremarkable.

Her best-corrected visual acuity was 20/20 OD and OS. Ocular motility was full with no limitation, with no afferent pupillary defect, and she was orthophoric in primary and lateral gazes in both eyes. She noted 10/10 color plates in each eye.

Intraocular pressure measured 15mm Hg OD and 16mm Hg OS. The anterior segment exam was unremarkable. Fundus exam showed cup-to-disc asymmetry OD>OS, and evidence of disc pallor in the right eye.

Although she was asymptomatic, we ordered spectral-domain optical coherence tomography (OCT) and visual field testing due to the presence of both cup asymmetry and optic nerve pallor. The SD-OCT showed significant nerve fiber layer thinning of the right disc, with moderate asymmetry compared to the left. Her left optic nerve was normal. Visual field showed subtle temporal field defects in the right eye that respected the vertical meridian. The left field was normal.

The findings of cupping and pallor, generalized depression of the nerve fiber layer and centralized temporal field defects respecting the vertical midline pointed to a non-glaucomatous etiology. Given her history and demographics, the differential diagnosis included demyelinating disease, a primary or metastatic intracranial space occupying lesion and immune-related optic neuropathy. Consequently, she was scheduled for magnetic resonance imaging (MRI) of the brain and orbits with and without contrast and fat suppression.

Imaging studies revealed a lobulated aneurysm on the right side in the region at or above the cavernous sinus, which compressed the right optic nerve.

The patient was referred to neurosurgery, where she had cerebral angiography that localized a bi-lobed right internal carotid artery para-ophthalmic aneurysm. The two lobes measured 8mm and 6mm. She underwent successful pipeline embolization of the aneurysm. As of her last follow-up, she was doing wonderfully. There was no further damage to her optic nerve.

Discussion

Optic atrophy is caused by damage to ganglion cells and axons of the optic nerve. This can be a result of ischemia, inflammation, compression, infection or trauma. Ischemic optic neuropathy, optic neuritis and papilledema are the more common conditions that can lead to optic atrophy, although the complete list of differentials is extensive.
INDICATIONS AND USAGE
TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration
The recommended dosage is 1 drop in the affected eye(s) once daily in the evening. TRAVATAN Z® Solution should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the IOP-lowering effect. TRAVATAN Z® Solution may be used concomitantly with other topical ophthalmic drug products to lower IOP. If more than 1 topical ophthalmic drug is being used, the drugs should be administered at least 5 minutes apart.

IMPORTANT SAFETY INFORMATION
Warnings and Precautions
Pigmentation—Travoprost ophthalmic solution has been reported to increase the pigmentation of the iris, periocular tissue (eyelid), and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periocular tissue and eyelash changes have been reported to be reversible in some patients. The long-term effects of increased pigmentation are not known. While treatment with TRAVATAN Z® Solution can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes—TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Use With Contact Lenses—Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Adverse Reactions
The most common adverse reaction observed in controlled clinical studies with TRAVATAN Z® Solution was ocular hyperemia, which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain, and pruritus. In postmarketing use with prostaglandin analogs, periocular and lid changes including deepening of the eyelid sulcus have been observed.

Use in Specific Populations
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

For additional information about TRAVATAN Z® Solution, please see the brief summary of Prescribing Information on the adjacent page.

Help patients start strong and stay on track with the Patient Support Program

*Study Design: Double-masked, randomized, parallel-group, multicenter non-inferiority comparison of the efficacy and safety of travoprost 0.004% preserved with benzalkonium chloride (BAK) to TRAVATAN Z® Solution after 3 months of treatment in patients with open-angle glaucoma or ocular hypertension. Baseline IOPs were 27.0 mm Hg (n=322), 25.5 mm Hg (n=322), and 24.8 mm Hg (n=322) at 8 AM, 10 AM, and 4 PM for TRAVATAN Z® Solution. At the end of Month 3, the TRAVATAN Z® Solution group had mean IOPs (95% CI) of 18.7 mm Hg (-0.4, 0.5), 17.7 mm Hg (-0.4, 0.6), and 17.4 mm Hg (-0.2, 0.8) at 8 AM, 10 AM, and 4 PM, respectively. Statistical equivalent reductions in IOP (95% confidence interval about the treatment differences were entirely within ±1.5 mm Hg) were demonstrated between the treatments at all study visits during the 3 months of treatment.

TRAVATAN Z® (travoprost ophthalmic solution) 0.004%

INDICATIONS AND USAGE

TRAVATAN Z® (travoprost ophthalmic solution) is indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION

The recommended dosage is one drop in the affected eye once daily.

TRAVATAN Z® (travoprost ophthalmic solution) should be administered more than once daily. It should not be administered in eyes with known sensitivity to travoprost.

TRAVATAN Z® Solution may be used concurrently with other topical ophthalmic drug products to lower intraocular pressure. However, more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

CONTRAINDICATIONS

None

WARNINGS AND PRECAUTIONS

Pigmentation

Travoprost ophthalmic solution has been reported to cause changes in pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, peribulbar tissue (eyelid and eyelashes). Pigmentation is expected to increase as long as travoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the peribulbar tissue and eyelash change have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation.

The long-term effects of increased pigmentation are not known. Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither new nor thickened of the iris appear to be affected by treatment. While treatment with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes

TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intracocular Inflammation

TRAVATAN Z® Solution should be used with caution in patients with active intracocular inflammation (e.g., uveitis) because the inflammation may be exacerbated.

Macular Edema

Macular edema, including cystoid macular edema, has been reported during treatment with travoprost ophthalmic solution. TRAVATAN Z® Solution should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

Angio-closure, Inflammatory or Neovascular Glaucoma

TRAVATAN Z® Solution has not been evaluated for the treatment of angle-closure, inflammatory or neovascular glaucoma.

Bacterial Keratitis

There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

Use with Contact Lenses

Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS

Clinical Studies Experience

Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates observed in the clinical studies of another drug and may not reflect the rates observed in practice. The most common adverse reactions observed in controlled clinical studies with TRAVATAN (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% were ocular hyperemia which was reported in 30% to 50% of patients. Up to 3% of patients discontinued treatment due to conjunctival hyperemia. Conjunctival adverse reactions reported at an incidence of 5% to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus. Ocular adverse reactions reported at an incidence of 1% to 4% in clinical studies with TRAVATAN or TRAVATAN Z® Solutions included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal drying, dry eye, iris disk color, keratitis, lid margin crusting, photophobia, subconjunctival hemorrhage and tearing.

Nonocular adverse reactions reported at an incidence of 1% to 5% in these clinical studies were allergy, angina pectoris, anxiety, back pain, bradycardia, bronchitis, chest pain, cold/flu syndrome, depression, dizziness, dyspepsia, gastrointestinal disorder: headache, hypercholesterolemia, hyperpyrexia, hypotension, infection, pain, postural disorder, sinusitis, urinary incontinence and urinary tract infections.

In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid margin have been observed.

USE IN SPECIFIC POPULATIONS

Pregnancy

Pregnancy Category C

Teratogenic effects: Travoprost was teratogenic in rats, at an intravenous (IV) dose up to 10 mcg/kg/day (350 times the maximum recommended human ocular dose (MRHOD), evidenced by an increase in the incidence of skeletal malformations as well as external and visceral malformations, such as fused sternebrae, domed head and hydrocephaly. Travoprost was not teratogenic in rats at IV doses up to 3 mcg/kg/day (75 times the MRHOD), or in mice at subcutaneous doses up to 1 mcg/kg/day (25 times the MRHOD). Travoprost produced an increase in post-implantation losses and a decrease in fetal viability in rats at IV doses > 3 mcg/kg/day (7.5 times the MRHOD).

In the offspring of female rats that received travoprost subcutaneously from Day 7 of pregnancy to lactation Day 21 at doses of >0.12 mcg/kg/day (3 times the MRHOD), the incidence of postnatal mortality was increased, and neonatal body weight gain was decreased. Neonatal development was also affected, evidenced by delayed-eye opening, pinna detachment and preputial separation, and by decreased motor activity.

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® Solution should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

A study in lactating rats demonstrated that radio labeled travoprost and/or its metabolites were excreted in milk. It is not known whether this drug or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when TRAVATAN Z® Solution is administered to a nursing woman.

Pediatric Use

Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

Geriatric Use

No overall clinical differences in safety or effectiveness have been observed between elderly and other adult patients.

Hepatic and Renal Impairment

Travoprost ophthalmic solution 0.004% has been studied in patients with hepatic impairment and also in patients with renal impairment. No clinically relevant changes in hematology, blood chemistry, or urinalysis laboratory data were observed in these patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility

Two-year carcinogenicity studies in mice and rats at subcutaneous doses of 10, 30, or 100 mcg/kg/day did not show any evidence of carcinogenic potential. However, at 100 mcg/kg/day, male rats were only treated for 82 weeks, and the maximum tolerated dose (MTD) was not reached in the mouse study. The high dose (100 mcg/kg) corresponds to exposure levels over 400 times the human exposure at the maximum recommended human ocular dose (MRHOD) of 0.04 mcg/kg based on plasma active drug levels. Travoprost was not mutagenic in the Ames test, mouse micronucleus test or rat chromosome aberration assay. A slight increase in the mutant frequency was observed in one of two mouse lymphoma assays in the presence of rat S-9 activation enzymes. Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 mcg/kg/day (310 times the maximum recommended human ocular dose of 0.04 mcg/kg/day on a mcg/kg basis (MRHOD)). At 10 mcg/kg/day, the mean number of corpora lutea was reduced, and the post-implantation losses were increased. These effects were not observed at 3 mcg/kg/day (75 times the MRHOD).

PATIENT COUNSELING INFORMATION

Potential for Pigmentation

Patients should be advised about the potential for increased brown pigmentation of the iris, which may be permanent. Patients should also be informed about the possibility of eyelash skin darkening, which may be reversible after discontinuation of TRAVATAN Z® (travoprost ophthalmic solution) 0.004%.

Potential for Eyelash Changes

Patients should also be informed of the possibility of eyelash and eyelid hair changes in the treated eye during treatment with TRAVATAN Z® Solution. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or eyelid hairs, and/or direction of eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container

Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice

Patients should also be advised that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctiva and eyelid reactions, they should immediately seek their physician’s advice concerning the continued use of TRAVATAN Z® Solution.

Use with Contact Lenses

Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Use with Other Ophthalmic Drugs

If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

Rx Only

U.S. Patent Nos. 5,631,287; 5,889,052; 6,011,062; 6,235,781; 6,503,497; and 6,489,253

Alcon LABORATORIES, INC.
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9/14 TRAVATAN Z® 0.004%
Determining the etiology of optic atrophy can be challenging, as there are multiple causes. A careful history and examination often pinpoints the cause, but unexplained optic atrophy needs to be explored. In one study, compressive lesions were the etiology in 20% of patients with isolated and unexplained optic atrophy. As a result, neuroimaging is warranted in all patients with unexplained optic atrophy. The preferred modality is MRI of the brain and orbits with and without contrast and with fat suppression. If there is a contraindication to MRI, computed tomography (CT) may be used instead.

Lab testing is of low yield unless historical or exam elements suggest an underlying etiology. Common lab tests for evaluation of optic atrophy include Lyme titer, fluorescent treponemal antibody absorption (FTA-ABS), rapid plasma reagin (RPR), angiotensin converting enzyme (ACE), Bartonella titer, antinuclear antibody (ANA), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). The recommendation when considering lab studies is to tailor based on suggestive history, exam findings or endemic locations that may suggest a greater likelihood of diagnosing conditions such as syphilis, Lyme disease or sarcoidosis. If the patient has a prior diagnosis of optic atrophy, be sure to obtain past medical records, imaging reports or laboratory studies, and document those in the patient’s chart.

This case also highlights the importance of distinguishing glaucomatous optic neuropathy (GON) from non-glaucomatous optic neuropathy (NGON), the latter of which was seen in our patient. Optic nerve cupping is most frequently caused by glaucoma, yet compressive lesions can cause secondary optic disc excavation and mimic glaucoma. While cupping asymmetry has a high predictive value of GON, the key clinical finding that helped distinguish GON from NGON in this patient was pallor of the neuroretinal rim, which is 94% specific for a non-glaucomatous etiology.

Ancillary studies that helped to confirm NGON were the use of formal visual field testing and SD-OCT. Visual fields are an invaluable asset in distinguishing glaucoma from neuro-ophthalmic-related optic nerve disorders. In the setting of presumed optic atrophy, a vertically-oriented centralized temporal field defect indicates an etiology other than glaucoma and prompts further investigation. Additionally, SD-OCT has been shown to distinguish glaucomatous from compressive optic neuropathies by analyzing patterns of nerve fiber layer loss. Specifically, cupping from NGON is associated with thinner nasal and temporal sectors when compared to that of glaucomatous disease.

In our patient, the SD-OCT revealed significant nerve fiber layer loss in all quadrants when compared to the Fellow eye. Lastly, her younger age, normotensive and symmetric IOPs and healthy disc in the fellow eye were all red flags for NGON.

Assuming there have been no acute vision changes or neurologic deficits, working up optic atrophy is generally non-urgent because the atrophic process takes a minimum of six weeks to develop. So, visual field, OCT and MRI/laboratory studies can be obtained within a few weeks.

This case highlights the importance of evaluating unexplained optic atrophy and differentiating GON from NGON. In-office assessment of the disc with digital photography, visual field testing and SD-OCT are invaluable in differentiating glaucoma from alternative neuro-ophthalmic pathology.

In this patient’s case, while there were subtle findings of pallor, there was no afferent pupillary defect, the visual acuity and color vision were normal and the patient was asymptomatic. So, in the presence of optic nerve asymmetry, consider alternative diagnoses and perform a detailed inspection of the neuroretinal rim for color changes that are not consistent with a glaucomatous process.

Risky Business

If you understand the influence of predisposing risk factors on dry eye, you’ll improve your odds of successful diagnosis. By Paul M. Karpecki, OD

Clinicians are well aware of the hallmark symptoms of dry eye, but if you based your decision to treat solely on symptoms, you might be missing some diagnoses; research shows that fewer than 60% of patients with dry eye disease (DED) are symptomatic. Often overlooked, but equally critical, is an awareness of predisposing factors that raise a patient’s risk level. Connecting these two is one of the most important steps in developing a successful ocular surface disease protocol.

This article will consider how predisposing factors combined with key findings become paramount to making a proper diagnosis and eventual treatment.

When Signs and Symptoms Don’t Correlate

In most conditions we encounter as optometrists, symptoms are paramount. For example, we judge our management of allergic conjunctivitis to be a success when a patient responds to treatment with an absence of itching, redness and swelling. Adopting this strategy—basing success on the relief of symptoms—works well for most ocular disease states and results in happy patients and a successful clinical outcome. But in DED, many patients with symptoms of dryness, grittiness, burning or stinging may not actually have “dry eyes,” and thus a treatment that targets dry eye would likely not alleviate symptoms. The result would be a patient returning with the same symptoms, and often more frustrated than before. They may even seek out another doctor.

For example, a patient experiencing dryness, fluctuating vision and gritty, burning eyes (especially late in the day or while using a computer) may actually have convergence insufficiency, vertical disparity computer vision syndrome or other eye alignment issues. This patient would receive a much better response from visual training or prism in their spectacles, or both, than any dry eye therapy. Other non-DED causes of similar symptoms include anterior blepharitis etiologies (e.g., Demodex), contact lens solution issues (e.g., corneal infiltrates), allergic conjunctivitis, epithelial membrane dystrophy/mild recurrent corneal erosion, pingueculitis, giant papillary conjunctivitis, Salzmann’s nodular degeneration, conjunctivochalasis and others.

In some circumstances, a patient with one of these conditions may experience mild resolution of symptoms when treated with classic dry eye therapies, but in many cases it will never fully resolve with an approach that targets DED.

At the other extreme, some of the worst cases of DED actually present with little to no symptoms. A patient suffering from severe dry eye who is largely asymptomatic (other
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than perhaps blurred vision from the advanced ocular surface disease findings) likely has a neurotrophic cornea.9 These include patients with active Sjögren’s syndrome, diabetes and those in the immediate aftermath of a herpes simplex keratitis, herpes zoster outbreak, a course of chemotherapy or radiation treatment. New research into neurotrophic keratitis shows a decreased corneal sensitivity secondary to decreased sub-basal corneal nerve length.10 This decreased sensitivity results in loss of sensation and a decreased blink rate, which only further exacerbates the condition.11

These patients are most in need of care; if symptoms were your sole determinant, they would be missed. Unfortunately, many such patients are overlooked, frustrated and seeking doctors who understand ocular surface disease management. A recent Harris Interactive study showed that only 29% of patients with true DED felt their optometrist provided adequate care and knowledge of their disease.

Suspicious Minds

It is a foundational principle of medical care that a proper diagnosis is the most important step to treatment. But if symptoms are not reliable, how might we make the proper diagnosis?

One way to raise your probability of success is to employ advanced testing such as osmolarity, meibography, true dry eye staining, meibomian gland expression, blink analysis and other precise measures now clinically available.12 But as many doctors just starting out may not have access to these technologies, another important determinant is to look at the number of predisposing factors a patient may display.

This approach alone may not be 100% accurate in establishing a diagnosis, but combined with testing, it can help confirm the disease.

A patient with a significant number of these predisposing factors may heighten the clinician’s suspicion of DED. When the level of suspicion is commensurate with risk factors, implementing and understanding dry eye diagnostics can point to the cause and true diagnosis.

Advanced Testing Options

As an example, I recall using osmolarity testing when it was first approved and had a series of patients with consistently normal osmolarity measurements. I’d look at the patient and ask them about their symptoms; they described their eyes as dry, gritty and burning with fluctuating vision that worsened late in the day and especially while reading or spending significant time on a computer. An expert in the field of binocular vision and eye alignment enlightened me and visited my clinic to help. He discovered that nine out of 10 patients that day had eye alignment issues, and he was able to resolve all of their symptoms with appropriate binocular vision management. I had a whole new respect for the accuracy of these advanced testing options and a newfound humility about what I thought was my advanced clinical knowledge in the field.

Since then, I’ve gleaned many other insights that have changed how we run our ocular surface disease clinic to achieve highly successful outcomes in some of the most advanced OSD referrals.

Dry eye is a complex condition to manage. But the critical first step is identification—with as much specificity as possible. Understanding the predisposing factors, realizing that signs and symptoms may not align and learning about new diagnostic technologies will aid clinicians in making the right diagnosis.

From there, an appropriate treatment becomes much more likely, resulting in successfully managed OSD patients.

Key Predisposing Factors

- Advanced age
- Female gender
- Hormone replacement therapy
- Systemic antihistamine use
- Lack of healthy essential fatty acid intake (e.g. omega-3 fatty acids)
- Connective tissue disorders
- Refractive surgery
- Androgen deficiency
- Contact lens use
- Rosacea
- Certain medications (e.g., some anti-depressants, diuretics, beta-blockers, isotretinoin)
- Diabetes
- Chemotherapy
- Low humidity environments
- Cigarette smoking

Source: The Dry Eye Workshop

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Detached Staff, Detached Retina

A poorly handled phone call likely worsened this young woman’s outcome. Don’t let this happen to your patients. Edited by Paul C. Ajamian, OD

Q A 21-year-old white female, a regular contact lens patient, called my office with a complaint of recent-onset blurry vision. My office was booked up that day, and my front desk staff recommended the patient see her primary care doctor. We later learned that the primary care doctor told the patient she probably had a migraine.

Afterward, I happened to check this patient’s chart and saw that she had a history of lattice degeneration. Now I’m concerned. What can I do to make sure such a snafu doesn’t happen again?

A “No matter what the patient’s complaint is—even if it’s itchy eyes—the staff should pull up the record at the time of the phone call,” says Steven Holbrook, OD, of The Eye Center of Southern Indiana.

Dr. Holbrook recounts the situation of a similar patient who wound up at his secondary-care center upon referral from the ER. In this young woman’s case, she too had called her eye doctor’s office with a complaint of sudden loss of vision and was referred to her PCP on suspicion of migraine.

The patient’s symptoms worsened over the weekend and she went to the ER. A CT scan performed at the ER was negative and she was referred to Dr. Holbrook’s practice.

“When she came in to my office on Monday, I found she had a macula-off retinal detachment with hand motion vision. In her other eye, she had extensive lattice degeneration,” he says.

In the patient’s other eye, she had extensive lattice degeneration.

Dr. Holbrook sent the patient to a retina specialist, who performed a scleral buckle the next day. At the patient’s most recent follow-up visit, her visual acuity had improved to 20/150.

Had her original eye doctor seen the patient that day, the situation might have turned out much better.

As it is, “I feel so bad for this girl. In all likelihood, she’s going to have permanent vision loss,” Dr. Holbrook says. In these cases, every clinician should be able to look back at an old record and see the results of a dilated peripheral retinal exam.

Also, be sure to train your staff—with regular reminders—to check the record of every patient before making a recommendation, Dr. Holbrook says.

And if there is something in the chart that raises a red flag, err on the side of bringing the patient in! But in the hopefully rare instance where you are unable to see the patient, send him or her to your nearest comanagement center. (Optometrists have worked for many years to gain diagnostic and therapeutic privileges, so we shouldn’t abdicate them to someone else at the patient’s expense.) Both the patient and your practice will be the victors with this strategy.
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COBURN TECHNOLOGIES
How to Educate Patients About the Dangers of Harmful Light

By Kirk Smick, OD

From the moment we wake up until we fall back to sleep, most of us are using some sort of digital device, including tablets, laptops and smartphones. In fact, nearly one-third of our adult patients spend more than half of their waking hours using a digital device.¹

While the benefits of digital technology can be life-changing, so can the hazards. Unfortunately, most adults (72.5%) are unaware of the potential dangers of blue light.¹ In the following pages, I’ll review the latest research on the risks posed by blue light exposure, examine its increasing presence in our everyday lives, and provide practical advice on what optometrists can do to better educate patients about protection.

THE ROLE OF LIGHT IN OUR LIVES

Misunderstandings abound with respect to the role that light plays in both helping and harming us. In fact, light is a biological requirement essential to health, and it plays an essential role in visual and systemic functions.

From the perspective of sight and visual functioning, light is essential for us to see and recognize colors, and it also plays a key role in contrast sensitivity. In particular, Blue-Turquoise light that ranges from 465 nm to 495 nm is essential to our vision, as it adjusts the size of the pupil to allow enough light through. Any way you look at it, light helps in providing visual acuity; without enough of it, vision is affected and is not clear.

Light is also necessary for various nonvisual functions of the body. It’s well known that some sun exposure is vital as it delivers vitamin D to the body. Beyond basic sunlight requirements, however, Blue-Turquoise light aids in the regulation of our sleep/wake cycle and also helps us distinguish day from night. This in turn, enables us to maintain and regulate memory, mood and hormonal balance.

NOT ALL LIGHT IS CREATED EQUAL

Although some light is good, too much can lead to cumulative damage to the eye tissues. The damaging effects of ultraviolet light on the cornea and the lens are well-established. Many of our patients are familiar with the potential dangers of ultraviolet light, however, very few patients are aware that certain wavelengths of visible light could also damage their eyes.

Even within the eyecare profession, we’ve questioned and have sought to understand the wavelengths at which blue light can cause damage. This is especially important because, while only 1 percent of ultraviolet radiation reaches the retina in adults, visible blue light easily penetrates to the retina² and can cause oxidative stress in both the photoreceptor outer segment and the retinal pigment epithelium. As a result, increasing attention is being paid to blue light—how its cumulative effects can cause damage to the retina and its implications in the development of age-related macular degeneration (AMD).³⁻⁵

In 2008, the Paris Vision Institute began ground-breaking research to better understand blue light.⁶ To determine whether specific bands within the Blue-Violet spectrum are responsible for blue light’s phototoxic effects on the retinal pigment epithelium (RPE), researchers developed a unique illumination system that allowed cultured porcine retinal cells to be exposed to narrow (10-nm) bands of light at moderate irradiances normalized to typical retinal sunlight exposure. Using this test system, it was discovered that RPE phototoxicity was concentrated in a relatively narrow band. They found that maximum cell damage occurs from 415 nm to 455 nm, with a peak at 435 nm ± 20 nm. This damaging band of light was termed Blue-Violet light and therefore, is a risk factor for age-related macular degeneration.
Nearly one-third of adults (30%) spend more than half their waking hours (9+) using a digital device.1

The Pervasiveness of Patient Exposure to Blue Light

Unfortunately, exposure to harmful blue light is almost unavoidable. Many Americans have made the switch to new energy-saving bulbs in their homes and offices. This, together with the increasing use of smartphones, tablets, and laptops, is causing a rise in blue light exposure.

According to the literature, the amount of blue light from light-emitting diode (LED) and compact fluorescent light (CFL) sources is substantial and can pose a risk of retinal damage to the eye.2 Approximately 26 percent of the light from the energy-efficient and increasingly popular CFLs is in the blue portion of the spectrum, and 35 percent of the optical radiation from cool white LEDs is blue.3

Similarly, there is no question that digital devices emit blue light, but just how much of it are patients being exposed to? The Vision Council recently released its third annual survey to examine the increasing usage of digital devices and consumer knowledge about its impact on vision.4 Nationwide, 9,749 adults participated in the survey. The findings show a stunning increase in the everyday use of digital devices across all age groups.5 As the 2015 report reveals, on average, more than nine in 10 adults (93.3%) spend more than two hours each day using a digital device, and this is taking a significant toll on patients’ eyes.

Specifically, nearly seven in 10 millennials (born 1981 to 1996) report symptoms of digital eye strain, with more than 37 percent spending at least nine hours each day on digital devices.6 In the generation X group (born 1965 to 1980), nearly one-third spends at least nine hours each day on digital devices and six in 10 report symptoms of digital eye strain.7 Kids and baby boomers spend at least nine hours each day on digital devices.8 And, among children, one in four kids spend more than three hours a day using digital devices,9 despite reports that too much screen time may cause accelerated myopia.8

Patients are being exposed to blue light in their homes and in their everyday lives at higher levels than ever before. Since these levels will only continue to rise in the coming years,10 it’s time we do something about it. The connection between blue light and ocular diseases, such as AMD, makes it imperative for us to educate patients and help them protect their eyes from these potentially damaging elements.

INTERVENTION

In addition to teaching patients about ergonomics and offering strategies for reducing digital eye strain, we now can also confidently offer lens treatments that limit the amount of blue light that penetrates to the eyes. The early blue blockers typically featured amber lenses that filtered out 100 percent of blue light. More recently introduced melanin-tinted blue blockers have advanced and are designed to protect the eyes from blue light, improve contrast, reduce eye fatigue and maintain color balance. However, these melanin lenses deflect 45 percent of the blue light spectrum, including both harmful Blue-Violet and beneficial Blue-Turquoise light.

A newer choice was inspired by the 2008 research out of Paris that identified the narrow Blue-Violet band at which most RPE phototoxicity occurs. This knowledge paved the way for selective photofiltration: the creation of lenses that reduce the level of exposure to the harmful portion of the Blue-Violet spectrum while permitting the rest of the visible spectrum to enter the eye at a normal level. Thus, the eye’s necessary visual and nonvisual functions can be maintained while exposure to hazardous wavelengths is reduced. The lens that was born of this research was Crizal® Prevencia™.

Crizal® Prevencia® No-Glare lenses are able to deflect up to 20 percent of the harmful Blue-Violet light that our patients are exposed to on a daily basis. The Paris Vision Institute’s in vitro study showed that this amount of deflection reduced retinal cell death in porcine cells by an impressive 25 percent.11

Dr. Smick is Chief of Optometry Services at Clayton Eye Center and an owner of the facility. He also serves as a technical advisor to many companies in the ophthalmic industry and has helped pioneer several visual advances.

11. The new normal. Make no mistake, digital devices are part of our lives and are here to stay. By 2020, it is estimated that 90 percent of all light sources will be based on LED products. Furthermore, LED light is expected to increase in residential settings by 50 percent for 2016 and 70 percent for 2020.11 As more and more patients spend longer periods of time engaged in digitally based activities, we have an increasing responsibility to find solutions aimed at protecting their vision. Crizal® Prevencia® No-Glare lenses are an excellent place to start.

Dr. Smick is Chief of Optometry Services at Clayton Eye Center and an owner of the facility. He also serves as a technical advisor to many companies in the ophthalmic industry and has helped pioneer several visual advances.
Pathology in Perspective: Differential Diagnosis of Retinal Disease

Many retinal conditions can be easily confused. Can you spot the masqueraders?

By Mohammad Rafieetary, OD, Stephen Huddleston, MD, and Eric Sigler, MD

Masquerading features of various retinal and chorioretinal disorders can result in a diagnostic conundrum. By familiarizing oneself with the differentiating nuances of these findings, we can overcome many of these clinical challenges. This article demonstrates a few examples of such cases and presents a series of self-test questions to sharpen your diagnostic skills.

CASE #1:
The above fundus photographs depict two separate patients, each complaining of blurred vision for a short but indeterminate length of time.

Both are new to your practice and have never had an eye exam before.

Questions
1. Which systemic processes may be involved in each patient presentation?
   a. The patient on the left has poorly controlled diabetes, hypertension or a mix of both. The patient on the right may have a sub-clinical systemic infection.
b. The patient on the left has poorly controlled diabetes, whereas the patient on the right has high blood pressure.
c. The patient on the left has a branch vein occlusion, while the patient on the right has increased intracranial pressure.
d. Both patients have no systemic disease related to their ocular findings.

2. What does not need to be included in the differential diagnosis for the patient on the right, who has no prior medical history and normal vital signs?
   a. Bartonella henselae.
   b. Acute viral illness.
   c. Sarcoidosis.
   d. Punctate inner choroidopathy.

3. Assuming the patient on the right is a 10-year-old with no significant past medical history, what is the best course of action?
   a. Intravitreal dexamethasone.
   b. Intravitreal anti-VEGF.
   c. Topical third-generation quinolone antibiotic.
   d. Short course of oral azithromycin.

4. You determine that the patient on the left has not been compliant with his antihypertensive medications. What do you do next?
   a. Observe closely after counseling.
   b. Contact patient’s primary care provider.
   c. Treat with topical steroids and NSAIDs.
   d. Prescribe oral steroids.

Answers
1) a; 2) d; 3) d; 4) b

Diagnoses
The patient on the right has hypertensive retinopathy whereas patient on the left has neuroretinitis caused by seropositive Bartonella henselae (cat scratch disease).

Discussion
Exudates appear in the intra- or subretinal space when the blood/retina barrier is interrupted within the retinal and/or choroidal circulation. The etiology of the vascular breakdown is wide-ranging and potential mechanisms include vascular disorders, such as hypertension or diabetes, as well as inflammatory and infectious diseases (see “Possible Causes of Retinal Exudates,” below).

In each of these conditions, the pattern of exudate—or lipid deposits resulting from leakage—may be different. For example, the two common conditions resulting in a so-called “macular star” exudate are hypertensive retinopathy and neuroretinitis. In other conditions, the exudate may be noted in the localized region of disease involvement, such as around aneurysms or along the involved vessels in a vein occlusion.

In neuroretinitis (right image), the exudates tend to have a perivascular distribution, located surrounding blood vessel terminations. Therefore, by combining the patient’s history (including existing medical conditions) and clinical presentation, you can develop a focused differential diagnosis and investigative process by which the cause, and the course of treatment, is determined.

Some investigators have speculated that a macular star is formed by transudation from capillaries deep in the optic nerve through the intermediate tissue of Kuhnt—a barrier that separates the optic nerve from the retina. Therefore, this type of exudative pattern is more common when there is both optic nerve and retinal (“neuroretinal”) involvement.

Possible Causes of Retinal Exudates

<table>
<thead>
<tr>
<th>Vascular Disorders</th>
<th>Diabetic retinopathy</th>
<th>Hypertensive retinopathy</th>
<th>Retinal vein occlusion</th>
<th>Coats’ disease</th>
<th>Retinal macroaneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious Disorders</td>
<td>Toxoplasmosis</td>
<td>Viral disease (e.g., herpes, HIV, CMV)</td>
<td>Syphilis</td>
<td>Bartonella</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Inflammatory Autoimmune Disease</td>
<td>Sarcoidosis</td>
<td>Systemic lupus erythematosus</td>
<td>Posterior uveitic disorders (multifocal choroiditis)</td>
<td>Vogt-Koyanagi-Harada disease</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Choroidal neovascularization (any etiology)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CASE #2:
Two new patients come into your clinic. Both are accompanied by their entire immediate families. Both are 22 years old, and both are excellent historians. This is the first eye exam for each patient and neither one has any complaints. Their fundi are shown on page 36.

Questions
1. Both patients are surprised by your exam findings, but only one family is shocked. This patient’s family has suffered through...
a string of relatives who had cancer diagnoses at a young age. Which patient has the strong family cancer history and what kind of cancer has her family suffered from?
   a. Top patient, colorectal.
   b. Bottom patient, lung.
   c. Top patient, lung.
   d. Bottom patient, colorectal.

2. Which patient has an underlying infectious etiology? What is the causative agent?
   a. Top patient, histoplasmosis.
   b. Bottom patient, toxoplasmosis.
   c. Top patient, toxoplasmosis.
   d. Bottom patient, histoplasmosis.

3. During your extensive counseling with the patient on the top, you recommend:
   a. A six-week course of erythromycin by mouth.
   b. Observe and return in six months to ensure no change.
   c. Immediate referral to the appropriate gastroenterologist.
   d. Referral for retina evaluation.

4. When explaining the diagnosis to the patient on the bottom, you decide to:
   a. Start a six-week course of sulfamethoxazole/trimethoprim by mouth.
   b. Refer for retina evaluation.
   c. Refer to a pulmonologist.
   d. Order serology studies to confirm the diagnosis.

Answers
1) a; 2) b; 3) c; 4) d

Diagnoses
The patient in the top set of images has Gardner’s syndrome. The patient in the bottom set of images has toxoplasmosis.

Discussion
Pigmentary lesions of a nonchoroidal origin represent reactive changes occurring in the retinal pigment epithelium (RPE). Both hyperplastic and hypertrophic changes may result in any combination of increased pigmentation, increased number or increased size of the RPE cells. Toxoplasmosis causes these changes on either a congenital or acquired basis. Most occur from a single systemic infection, resulting in static lesions of varying size and significance to vision. However, the typical ocular
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findings in Gardner’s syndrome, a subtype of familial adenomatous polyposis, result in the progressive accumulation of lesions mimicking congenital hypertrophy of the RPE. The presence of an unusually high number of hypertrophic retinal pigment epithelial lesions (similar to congenital hypertrophy of retinal pigment epithelium [CHRPE]) and a typical teardrop appearance should clue the clinician in to a possible diagnosis of Gardner’s syndrome and the need for referral to a gastroenterologist.

While typical old, inactive non-macular toxoplasmosis lesions can be observed, patients with lesions involving the macula or of a significant size should be sent for a retina evaluation. So, the patient in this case (bottom images) has significant macular lesions and should be referred to a retinal specialist.

CASE #3:
Two new patients come into your clinic with similar complaints of gradual loss of central vision. Both measure 20/200 centrally, and both report a decade of worsening. Both patients have smoked cigarettes for years.

Their fundus photos are shown above.

Questions
1. What is your best assessment based on the presented information?
   a. Both have dry AMD.
   b. Both have wet AMD.
   c. The patient on the right has AMD and the patient on the left has macular dystrophy.
   d. The patient on the left has AMD and the patient on the right has macular dystrophy.

2. Assuming one of these patients suffers from a macular dystrophy, which of the following is the most likely form?
   a. Best disease.
   b. Adult vitelliform.
   c. Multifocal pattern dystrophy.
   d. Sorsbys.

3. Which test is the least invasive, yet particularly beneficial to aid with the differential diagnosis in either patient?
   a. Electroretinography.
   b. Fundus autofluorescence imaging.
   c. Genetic testing.
   d. Fluorescein angiography.

4. For the patient on the left, which is the most significant counseling you need to provide?
   a. Smoking cessation.
   b. Genetic testing.
   c. Recommend anti-VEGF therapy.
   d. Annual eye health examination.

Answers
1) d; 2) c; 3) b; 4) a

Diagnoses
The patient on the right has atrophic AMD. The patient on the left has multifocal pattern dystrophy.

Discussion
Fundus autofluorescence has become a valuable diagnostic tool for multiple retinal conditions, particularly

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in the management of patients within the spectrum of macular degenerative and dystrophic conditions. For example, in age-related macular degeneration, drusen as an early indicator for the disease shows subtle and varying degrees of hypo- or hyperautofluorescence, whereas the diseased retinal pigment epithelium (RPE) accumulating lipofuscin typically demonstrates hyperautofluorescence corresponding to the so-called “vitelliform” lesions.

Eventually, the death of RPE (clinically known as geographic atrophy) represents the total absence of autofluorescence (A).

The RPE involvement in various macular dystrophies can have a distinctive appearance. For example, in multifocal pattern dystrophy (B), the RPE disease can be seen in a reticular pattern, whereas in fundus flavimaculatus (C), these lesions are in the so-called pisciform (fish bone) pattern (C).

CASE #4:
Two asymptomatic male patients were referred for evaluation of the lesions seen in the photos on page 42.

Questions
1. Based on the OCT scans shown, what is the primary site of the lesions?
   a. Inner retina.
   b. Outer retina.
   c. Retinal pigment epithelium.
   d. Choroid.

2. Which of the following would you use to evaluate and diagnose these lesions?
   a. Lesion’s height.
   b. Presence of subretinal fluid.
   c. Presence of orange pigment.
   d. All of the above.

3. Which is the most appropriate referral for further management of these patients?
   a. To patient’s primary care provider.
   b. To a general ophthalmologist.
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c. To an ophthalmic oncologist.

d. Follow-up only; no referral needed.

4. If the patient on the right elects to have treatment, which is the most likely plan?

a. Retinal photocoagulation.
b. Brachytherapy.
c. Enucleation.
d. Intravitreal anti-VEGF.

**Answers**

1) d; 2) d; 3) c; 4) b

**Diagnoses**

The patient on the left has a choroidal hemangioma.
The patient on the right has a choroidal melanoma.

**Discussion**

Both of these patients have vision-threatening lesions that require urgent or emergent referral for evaluation by an ocular oncologist, but only one of the lesions is life threatening. Correctly identifying an ocular mass as a choroidal melanoma instead of a choroidal hemangioma involves adequate counseling and coordination with your collaborating ocular oncologist.

Recognizing high-risk features—such as the presence of orange pigment, subretinal fluid and change in tumor size—are crucial for differentiating a melanoma from a nevus.

Choroidal hemangiomas (left) are non-malignant vascular tumors that either exhibit an indolent course without growth or leakage or an aggressive course with growth and leakage. Diagnosis can be made with OCT, FA and B-scan. FA will show a distinctive, course, vascular pattern and B-scan will exhibit high internal reflectivity.

Choroidal melanomas (right) are the most common primary malignant intraocular tumor and they are missed or misdiagnosed daily. Indolent peripheral choroidal hemangiomas may be safely observed, while vision-threatening lesions are treated with anti-VEGF agents or PDT. With appropriate management, successful outcomes are common.

Once a diagnosis is made, correctly classifying the tumor (based on its ultrasonographic size into the small, medium and large categories described by the Collaborative Ocular Melanoma Study publications) can give patients a better idea of their chance of successful treatment and mortality rates.

Remember, essentially all primary choroidal melanoma metastases are to the liver, and routine surveillance with abdominal ultrasound or CT is required for detection of early metastatic disease after diagnosis and treatment.

Performing thorough exams and keeping your clinical skills honed are the keys to providing the level of care your patients deserve.

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Dr. Sigler is a retina specialist at Ophthalmic Consultants of Long Island.
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The eyelids provide several important functions: they protect the globe from external threats, supply lipid secretions necessary to create and maintain a healthy tear film, and they distribute and drain the tears. Unfortunately, when evaluating a patient at the slit lamp, it is altogether too easy to overlook the lids and zoom in on the ocular surface. A thorough examination of the lids can be critical for systemic health in addition to ocular integrity. Let’s review some of the critical factors to consider and signs to look for.

**Position**

Lid examination begins during the patient intake. While reviewing the patient history, note the position of the lids. When the eye is open, the upper lid should cover 1/6th of the cornea, with the lower lid just touching the inferior limbus. The lid should approximate the globe along the entire lid margin, without turning in or out.

Unequal apertures could indicate a serious condition such as thyroid disease or a space-occupying lesion. It must be determined whether there is proptosis or an enophthalmos of the contralateral eye. A Hertel ophthalmometer is useful for determining globe position disparity; a difference of 2mm is considered abnormal.

**Entropion and Ectropion**

A turning in of the lid margin represents *entropion*, which is one cause of lashes dragging across the ocular surface (*trichiasis*). Symptoms include redness and pain around the eye, sensitivity to light and wind, sagging skin around the eye, epiphora and decreased vision, especially if the cornea is damaged. Causes include congenital abnormalities, aging (which creates loose skin), stretched and loose ligaments and muscles, scarring, trauma, trachoma and involution spasms.

Entropion may not be seen when the patient is at rest in the chair but generally can be elicited when the patient squeezes the lids.

**Ectropion**, by contrast, is a condition in which the eyelid margin turns outward. A myogenic ectropion in particular is caused by a weakness...
in the lid muscles. Ectropion may be a natural result of age or a consequence of facial paralysis caused by Bell’s palsy or tumor. Other potential etiologies include scarring from burns or trauma, benign or cancerous lid lesions, blepharoplasty, radiation for neoplasms or cosmetic laser skin resurfacing, cicatrizng conditions like Stevens-Johnson syndrome or ocular cicatricial pemphigoid; or it may be congenital, as in Down’s syndrome.

Blepharoptosis

Ptosis can be either congenital or acquired. Congenital ptosis is present at birth and is prevalent equally in both males and females. It is caused by levator development abnormality, resulting in fibrosis and fatty infiltration of the muscle. A congenital ptosis may be either unilateral or bilateral. The lid crease will be poorly formed in the affected eye(s) and individuals with congenital ptosis may suffer from nocturnal lagophthalmos. Bilateral cases develop a chin-up posture to see under the drooping lid, while unilateral cases are at risk for amblyopia. Sixteen percent of congenital ptosis cases will have abnormal superior rectus function.1

Acquired ptosis may arise from floppy lid syndrome, levator dehiscence from contact lens wear or aging, mechanical/traumatic conditions or neurogenic causes.

Floppy lid syndrome is defined by easily everted eyelids with a thickened, elastic tarsal plate and lash ptosis. It has been associated with giant papillary conjunctivitis and keratoconus, chronic eye rubbing and obesity.2 Up to 85% of patients with floppy eyelids may have obstructive sleep apnea, a potentially life-threatening condition, and therefore should be evaluated in a sleep clinic.3

The majority of levator dehiscence is associated with rigid contact lens wear. It is generally accepted that removal of lenses by tugging on the upper lid and harsh blinking is the cause.4 Slippage of the aponeurosis is the most common cause of mild to moderate ptosis in the elderly. It should be differentiated from neurogenic causes of ptosis. Other conditions that mimic lid ptosis are dermatomalgia and brow ptosis. Patients with ptosis may complain about vision loss (especially when reading), headaches, eyebrow strain and cosmesis. The palpebral aperture should be documented, and it may be helpful to compare the lid position to old photographs. Medicare requires at least 20 degrees of documented visual field restriction to pay for repair.

Other, more rare, causes of acquired ptosis include neoplasm, neurofibromas and cicatricial disease.

Innervation

The eyelids are innervated by the oculomotor nerve (CN III), the facial nerve (CN VII), the trigeminal nerve (CN V) and sympathetic fibers. The orbicularis oculi is responsible for shutting the eye and is innervated by the facial nerve. The oculomotor nerve innervates the levator and tarsal plate and opens the eye. The ophthalmic division of the trigeminal
nerve is purely sensory. Sympathetic nerve fibers of the lids innervate Müller’s muscles.

Marcus-Gunn jaw winking is an aberrant connection of the oculomotor nerve fibers that innervate the levator and the trigeminal nerve fibers of the muscles of mastication (motor nerve). Jaw winking is present in approximately 5% of congenital ptosis cases, with nearly 4% being bilateral.5

A seventh, or facial, nerve palsy causes an inability to close the eyelid. Bell’s palsy is the most common subset of conditions causing facial nerve paralysis. Bell’s palsy affects 11 to 40 persons per 100,000 each year, with peak incidence between the ages of 15 and 50.6,8

Reactivation of herpes simplex and herpes zoster is postulated to account for the majority of cases of Bell’s palsy.9 The condition typically presents with a sudden and rapid onset of unilateral facial weakness, often within a few hours. Less than 1% of cases are bilateral. While the majority of cases are self-limiting with complete recovery, recent evidence shows the early treatment with corticosteroids may improve recovery results.9 Antiviral therapy does not improve chance of recovery.10 Differential diagnosis includes brain tumor, stroke, myasthenia gravis and Lyme disease.

In contrast to facial nerve palsy, several conditions result in the inability to open the eyes. Horner’s syndrome is impaired innervation of sympathetic to Müller’s muscle. It is often associated with the classic triad of ptosis, miosis and anhydrosis, although patients rarely present with all three signs. It can be either congenital or acquired, and the risk to patient health varies from benign to life threatening based on the underlying cause.11 Patients should undergo a thorough neurological work-up.

Third nerve palsy also results in an inability to open the lid. Examination may show a dilated, poorly reactive pupil; reduced ocular movements; and ocular misalignment, where the eye is located down and out. Pupil-sparing third nerve palsy involves ischemic cranial neuropathy (such as in diabetes or hypertension), while pupil-affecting third nerve palsy involves compressive lesions or aneurysm.

Acquired myasthenia gravis involves inability to open the eye, which may be variable throughout the day and worsen when tired. This autoimmune condition affects 20 in
Lid papillomas may not always be as obvious as that seen in the left image. They may in fact be more subtle and difficult to spot, as in the lid margin papilloma at right.

100,000 people and is caused by a reduction in acetylcholine receptor sites. Common symptoms of myasthenia gravis include a drooping eyelid, blurred or double vision, slurred speech, difficulty chewing and swallowing, weakness in the arms and legs, chronic muscle fatigue and difficulty breathing.12

Lesions
Skin lesions found around the eye can be confusing—benign ones may have a dramatic appearance while malignant lesions look unassuming. Papillomas most commonly present in middle-aged adults and the elderly. Squamous papillomas represent a benign hyperplasia of squamous epithelium. Viral papillomas are associated with local infection involving the human papilloma virus (HPV). Papillomas on the skin will appear skin-colored, tan or brown. They may be round or pedunculated. They are easily removed with excision. On the conjunctiva, a papilloma may appear as a mass of vessels or a cluster of hyperemic cells and must be differentiated from a squamous cell carcinoma.13

Actinic keratosis is rare under 30 years of age. It presents as a flat, light tan lesion, most common on the face, trunk and upper extremities, which becomes pigmented, elevated and warty over time. There is a 20% risk of progression to squamous cell carcinoma. The etiology is presumed to be sun exposure and the treatment is biopsy, excision and cautery.14
**Eyelids**

Lid notching, ectropion and missing lashes accompany this case of eyelid basal cell carcinoma.

*Epidermal inclusion cysts* can occur at any age and are equally prevalent in males and females. They arise from hair follicles and appear as smooth, round elevated cysts filled with keratin. Ablation of entire cyst walls is necessary for eradication.

*Sebaceous cysts* clinically look like epidermal inclusion cysts; however, they arise from blocked glands of Zeis, meibomian or sebaceous glands. They are filled with epithelial cells, keratin, fat and cholesterol crystals. They are treated with expression or surgical excision.

*Acquired nevus* begins in childhood when the basal epithelium migrates to the dermis surface. They may appear deeply pigmented or amelanotic. The lesion may be flat or pedunculated. A key characteristic is maintenance of lashes through the lesion. While lid nevi are benign, about 5% will transform to malignant melanoma and therefore should be photo-documented and closely monitored.15

*Sebaceous cell carcinoma* arises from the glands of Zeis at the lid margin or the meibomian glands in the deep tarsus. Sebaceous cell carcinomas account for 2% to 7% of eyelid tumors.15 Clinically, they appear as a solitary lesion with diffuse lid thickening. There is a loss of lashes. They are frequently mis-diagnosed as recurrent chalazion, chronic meibomitis or blepharoconjunctivitis.16 These can be aggressive tumors, with orbital extension in 17% of cases and systemic metastasis in 8% of cases. Biopsy/excision is recommended for suspected sebaceous cell carcinomas.16

*Basal cell carcinoma* is the most common tumor of the skin with >400,000 people treated annually.17 It has been associated with sunlight exposure and *Demodex* infestation. On the eyelids, 65% will involve the lower lid, 15% the medial canthus, 15% the upper lid and 5% the lateral canthus.17 They appear as pearly, waxy translucent lesions with rolled borders. There is often telangiectasia near borders and loss of lashes. Tumor extensions are possible, but no distant metastasis. The mortality rate is <1%.17

*Primary malignant melanoma* accounts for only 1% of malignant eyelid tumors.18 They may be either a primary lesion or a metastasis from another location. They generally appear in sun-exposed areas as a variably pigmented mass, which can bleed or ulcerate. Malignant melanoma must be histopathologically proven, and early diagnosis is critical, since prognosis depends on if the lesion metastasizes.18

**Disorders of the Lashes**

Growth of lashes from within the meibomian glands is *distichiasis*. Congenital distichiasis is caused by the epithelial germ cell’s failure to differentiate completely to meibomian glands. It is dominantly inherited with complete penetrance and may be isolated or associated with ptosis, strabismus or congenital heart defects. Acquired distichiasis is seen on the lower lid, where the lashes may be either pigmented or non-pigmented. It is associated with chronic inflammation.

*Madarosis* is a decrease or loss of lashes. It is commonly seen in patients with long-standing anterior blepharitis, but is also seen with tumor, thermal burns and trichotillomania. It can be associated with alopecia, atopic dermatitis, systemic lupus and ichthyosis.

*Hypertrichosis* is a benign condition with excess or abnormally long lashes. It can either be congenital or drug induced. Some drugs that have been known to cause hypertrichosis include: prostaglandins (e.g., latanoprost, travoprost, bimatoprost), pentoxytin, acetazolamide, streptomycin, cyclosporine, psoralen, diazoxide and minoxidil.

*Poliosis* is premature whitening of the hair, lashes and eyebrows. It is associated with vitiligo, Waardenburg syndrome (iris heterochromia, white forelock) or chronic lid margin inflammation.

**Infection**

The eyelids are prone to infection from multiple sources. *Molluscum contagiosum* is an itchy viral infection (poxvirus) of the skin that presents with white/pink dome-shaped, smooth, waxy or pearly umbilicated papules 2mm to 5mm in diameter. It is spread either by direct contact or via fomites. Immunocompromised individuals may have more widespread infection and can be difficult to treat. Lesions generally regress spontaneously after one to two years, and treatment is only indicated for...
Avenova™ with Neutrox™ (pure hypochlorous acid) removes microorganisms and debris from the lids and lashes. Avenova is an ideal addition to any daily lid and lash hygiene regimen, including for use by patients with Blepharitis and Dry Eye. Avenova may also be used after make-up removal as well as pre and post contact lens wear.
cosmesis and prevention of spread. The condition is most common in children as a benign childhood entity; when seen in adults, it may represent a sexually transmitted disorder and should be treated as such until discovered otherwise.

Blepharitis is a common condition (found in 70% of the population) affecting the eyelid margins. Because of its ubiquity, it is often overlooked in the early stages. Associated morbidities include dry eye, loss of visual function and loss of well being and ability to carry out activities of daily living. Over time, damage to the lids occurs, including trichiasis, notching, entropion and ectorrhaphy. The cornnea may be damaged due to inflammation and limbal stem cell deficiency, leading to scarring, loss of surface smoothness and optical clarity. Other signs of chronic blepharitis include crustling and collaretttes, marginal telangiectasia, marginal keratinization, decreased and brittle lashes, poliosis and distended or blunting of meibomian glands.

Blepharitis has been separated into anterior vs. posterior blepharitis. Anterior blepharitis involves the anterior portion of the lid margin, including the lash follicle, while posterior blepharitis involves the meibomian glands. Normal (common) bacteria of the eyelids include Staphylococcus epidermidis (95.8%), Propionibacterium acnes (92.8%), Corynebacterium sp. (76.8%), Azinemobacter sp. (11.4%), and Staphylococcus aureus (10.5%).\(^\text{19}\) The lids may also be colonized with Demodex.

Demodex is an eight-legged mite that lives in hair follicles and oil glands. There are 65+ species of Demodex, only two of which live on humans (D. folliculorum and D. brevis), with a life span of two to three weeks. They are spread either through direct contact or in dust and towels containing eggs. They eat skin cells, hormones and oils in the follicles and glands. In addition to lid margin dysfunction, Demodex has been implicated in rosacea, seborrheic dermatitis and other skin conditions.\(^\text{20}\)

Demodex signs include cylindrical dandruff around a “volcano-like” lash base. Dry eye symptoms increase with Demodex infestation with more than 85% of patients showing evaporative dry eye.\(^\text{21}\)

Demodex is acquired shortly after birth, and bio load increases with age. Demodex has been correlated with other inflammatory ocular states such as Salzmann’s nodular degeneration, ocular rosacea and peripheral ulcers. Studies show nearly 90% to 100% of people with blepharitis have Demodex.\(^\text{22}\)

Blink and You’ll Miss It

The eyelids are easy to pass over during an ocular exam. The periorbital region feels oddly foreign to many eye care professionals. Yet, the lids are vitally important to not only the health of the eye, but can indicate systemic health integrity as well. A systematic approach to lid examination, which begins chairside, ensures the eyelids will not be overlooked.
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D iagnosis is rarely an exact science. Patients suffering from a multitude of ailments might display strikingly similar signs and symptoms, and subtle distinctions between different classifications of disease types can easily go unrecognized. Optometrists today benefit from an ever-increasing sophistication in our understanding of the causes and presentations of ocular disease. Still, outside of laboratory settings, hard data is hard to come by; we often must rely on clinical instincts and expertise when making the call.

That clinical conundrum has given rise to various in-office testing services. These seek to bridge the gap between the lab and the clinic by delivering hard, measureable evidence that may yield a diagnosis in relatively short order. With the advent of point-of-care (POC) testing, the clinician is able to obtain a result in a timely fashion—essentially in real time, for many—avoiding the hassle of sending the patient to a different facility, risking incomplete or inaccurate testing.

While many of our most confounding differential diagnoses remain purely clinical, several conditions now allow us to turn to POC testing to provide a more comprehensive exam than ever before. Let’s look at its use in practice, with a general overview of what the literature documents on the available tests and a series of illustrative cases.

Adenoviral Conjunctivitis
Red eye is one of the top emergent reasons a patient will report to an eye clinic. A child with a red eye is often kept home from daycare or school until infectious “pink eye” is ruled out. Health care workers and individuals who work with the public also take the day for fear of spreading an infection. However, a patient presenting with acute-onset ocular redness can have any number of potential underlying etiologies. Clinicians prefer to treat these patients empirically.

• Pathogen screening. AdenoPlus (RPS) is the only currently available CLIA-waived point-of-care test that detects adenoviral infections. Incorporating the AdenoPlus test into a red eye work-up can help increase the accurate diagnosis of viral conjunctivitis, narrow down appropriate treatment options and help reduce the potential spread of the virus.

In a recent study, researchers looked at the specificity and sensitivity of AdenoPlus at detecting the presence of adenovirus in tear fluid as compared with both viral cell culture with confirmatory immunofluorescence assay (CC-IFA) and polymerase chain reaction (PCR).1 Viral culture uses a series of primary cell lines that allow the replication of a wide variety of clinically relevant viruses. Specimens are inoculated onto cell culture monolayers and monitored for changes that occur in response to viral infection.2 Immunofluorescence assay (IFA) uses fluorescent-labeled antibodies to detect
specific target antigens and is used extensively scientifically and clinically to identify specific antigens.\(^2\) PCR allows for the synthesis of a new strand of DNA complementary to the offered template strand (e.g., adenovirus). DNA polymerase can synthesize DNA complementary to a presented target sequence, resulting in the production of billions of copies of the unknown virus.\(^2\)

The study results demonstrated that when compared with CC-IFA, AdenoPlus showed a sensitivity of 90% and specificity of 96%. When AdenoPlus was compared only with PCR, it showed a sensitivity of 85% and specificity of 98%. When compared with both CC-IFA and PCR, AdenoPlus showed a sensitivity of 93% (27/29) and specificity of 98%.\(^1\)

Dry Eye

Numerous factors can predispose patients to dry eye, including increasing age, contact lens wear, meibomian gland dysfunction and autoimmune disease.\(^3\) However, diagnosis is challenging, as patients may report with significant symptoms but no obvious signs of ocular surface compromise, or vice versa.\(^1\)

Dry eye patients often experience inflammation associated with an increase in matrix metalloproteinases (MMPs). These proteolytic enzymes are produced by compromised epithelial cells on the ocular surface. Investigators have consistently shown MMP-9 elevated in the tears of patients with dry eyes.\(^4\)

- **Measuring MMP.** InflammaDry (RPS) samples tears for the presence of elevated MMP-9. Sambursky et al. evaluated 143 patients with clinical signs and symptoms of dry eye, and 63 healthy individuals serving as controls. Participants were assessed as healthy controls or clinically diagnosed with dry eye using a combination of the Ocular Surface Disease Index, Schirmer tear test, tear break-up time, and corneal and conjunctival staining. InflammaDry demonstrated a sensitivity of 85% and specificity of 94% in diagnosing the dry eye cohort as compared with the healthy cohort.\(^5\)

The ability to rapidly and reliably assess MMP-9 levels may lead to the clinician making an earlier diagnosis of dry eye and initiating management. Patients with MMP-9 levels less than 40ng/mL are thought to be in the “normal range” and not suffering from ocular surface disease,

### Adenoviral Conjunctivitis Case

Courtesy of Blair Lonsberry, OD

A 42-year-old white male presented on an emergent basis, with a red eye. His employer (at a fast food restaurant) expressed concern that he may be carrying “pink eye” and shouldn’t be at work until it was ruled out. The patient, however, reported that working around the heat of the cooking fryers occasionally triggers his allergies and causes eye redness.

The patient reported that his right eye became red a “couple of days ago” and he has noticed the eye feeling “tender,” but not itchy. He reported taking Claritin for allergies. He stated that he hasn’t been sick recently, is not a contact lens wearer and notices some excess watering, especially outside. He reports his wife and daughter recently developed red eyes and were being treated.

- **Testing.** VA was measured at 20/25 uncorrected in both eyes with EOMs, pupils and visual fields unremarkable. A slit lamp examination (see image) revealed 2+ injection of the right eye and trace injection in the left eye. His cornea was clear, with no staining by sodium fluorescein or lissamine green, and trace papillae were noted on the upper lids. We performed the AdenoPlus test, collecting a sample from his right eye.

The results of the test are indicated through two lines, which appear in the result window: the control line and the result line. The control line is blue and indicates the correct application and performance of the test, and must appear for the test to be valid.\(^1\) The presence of both a blue and red or pink line in the result zone indicates a positive result. Even if the red or pink line is faint in color, incomplete over the width of the test strip or uneven in color, it must be interpreted as positive. A positive result indicates the presence of Adenovirus antigens.\(^1\) The AdenoPlus test we performed presented with a dark red or pink line and the blue line, confirming a positive result for adenovirus.

- **Diagnosis and Treatment.** We diagnosed the patient with epidemic keratoconjunctivitis (EKC) secondary to the severity of the patient’s symptoms, his family members having a similar red eye and the intensity of the positive response on the AdenoPlus test. We treated him with betadine, prescribed a topical steroid to be used QID and informed him that he would not be able to return to work due to the potential infectious etiology of his condition. Educating him included advising him to limit contact with others, not share towels or washcloths and to wash his hands frequently. He was asked to return to the clinic the next day for follow up.

- **Analysis.** This case demonstrates the importance of being able to provide a patient with a concrete diagnosis, the appropriate management and patient education, in a timely manner. Until the AdenoPlus test confirmed a viral infection, the patient was convinced his symptoms were a result of allergies and dryness, and sought clearance to return to work. Performing the AdenoPlus test gave us an objective result indicating that his red eye was secondary to a viral infection and not allergies. The test prevented his coworkers and customers from exposure.
MMP-9 and Sjögren’s Case

Courtesy of Douglas Devries, OD
A 59-year-old pseudophakic Hispanic female returned to the clinic reporting constant foreign body sensation and dryness in both eyes. She had undergone cataract surgery 13 months earlier. She reported no relief with preservative-free artificial tears. She also reported a dry mouth and her speed score was recorded as 10.

- **Testing.** Slit lamp evaluation revealed 2+ MGD OU, 3+ conjunctival chalasis OU, and 1 to 2+ inferior SPK OU. RPS InflammaDry testing was positive OU. The results of the Sjö test was positive for the salivary protein (SP-1) EAg antibodies.
- **Diagnosis and Treatment.** She was diagnosed with conjunctivochalasis, early Sjögren’s syndrome with keratoconjunctivitis sicca. She was placed on Restasis, warm compresses, and scheduled for LipiView. She was also scheduled for conjunctival resection with fornix rebuild.
- **Analysis.** This patient has 3+ conjunctivochalasis, which likely contributed to her foreign body sensation as well as the inferior SPK due to poor wetting. The positive InflammaDry testing suggests a moderate MMP-9 level, which appears as a result of stressed epithelial cells, a common finding in both dry eye disease and conjunctivochalasis. This patient will require comprehensive treatment for all of her ocular surface disorders.

and levels above 40ng/mL is considered elevated and abnormal and indicative of some form of ocular surface disease. The determination of a patient having elevated MMP-9 levels, and therefore elevated inflammatory makers, may provide the clinician with an avenue of treatment that may have better efficacy, such as the use of anti-inflammatory agents (topical steroids, cyclosporine and other immunomodulators). An accurate measure of MMP-9 as an indication of inflammation on the ocular surface could be particularly important for the management of refractive or cataract surgery patients, where a stable tear film is important in post-operative outcomes.

- **Testing tear osmolarity.** The Dry Eye Workshop consensus opinion published in 2007 included tear osmolarity changes in its dry eye definition. This is now seen as a hallmark sign, and a possible central mechanism, of the pathogenesis of dry eye. A study by Bron et al. showed that, in healthy subjects, tear osmolarity is maintained in a relatively narrow range, with an average value of approximately 302mOsmol/L. Also of note, the study showed no significant difference in osmolarity between eyes (average around 6.9mOsmol/L).

The study shows that increased osmolarity has a predictive relationship with dry eye inflammation. TearLab provides a POC testing unit able to measure a patient’s tear osmolarity in-office. The unit obtains a 50nL sample of tears from a patient’s tear film and analyzes the sample, providing a measure of the patient’s tear osmolarity. The process of acquiring the tears is brief and straightforward, typically obtained from the lower temporal canthal area, resulting in minimal stimulation of reflex tearing.

In determining what “normal” osmolarity levels are for this protocol, 300 subjects were analyzed and...
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In-Office Testing

Diabetes Case

Courtesy of Paul Chous, OD

A 27-year-old white male presented for an eye exam. He has had Type 1 diabetes for 15 years. When asked what his last A1c value was, he said 120. He said his vision was fine, but his sister insisted that he get his eyes checked. The patient was taking Lantus and Humalog, his vision is correctable to 20/20 with an additional -1.25D (obvious myopic shift).

- Testing. His dilated fundus exam showed mild nonproliferative diabetic retinopathy in both eyes with no observable diabetic macular edema; however, an OCT image revealed significant macular thickening and the presence of diabetic macular edema. Using the A1cNow test, his level was determined to be 12%.

- Diagnosis and Treatment. The patient returned with his spouse to discuss what was observed on the OCT and his current diabetes control. The patient was educated that his control is not at an acceptable level and he was referred to an endocrinologist. He was placed on an insulin pump with continuous blood sugar monitoring. His A1c dropped to 7% over the next year with no changes in his nonproliferative diabetic retinopathy or OCT over 23 months. He has since referred some 20 patients to the office for vision exams—patients with and without diabetes.

- Analysis. Optometric physicians can order other tests and even obtain the necessary sample from the patient to be analyzed, but these tests do not provide a result immediately and often require a day or two to obtain the results.

Get CLIA Certified for POC Testing

By John Rumpakis, OD, MBA, Clinical Coding Editor

Many tests performed at the point of care require that your office have a Clinical Lab Improvement Amendments (CLIA) waiver.

- What is CLIA? In 1988, Congress passed CLIA to establish quality standards for all laboratory testing to ensure the accuracy, reliability, and timeliness of patient test results, regardless of where the test is performed.1

CLIA-waived tests were also defined as simple laboratory examinations and procedures that are cleared by the Food and Drug Administration (FDA) for home use, employ methodologies that are so simple and accurate as to render the likelihood of erroneous results negligible, or pose no reasonable risk of harm to the patient if the test is performed incorrectly. These regulations were further revised in 1997 to make it clear that tests approved by the FDA for home use automatically qualify for CLIA waiver.

- How do you become CLIA certified? Performing CLIA-waived tests, and obtaining reimbursements for them, require your office be designated as a CLIA-approved laboratory and one of your doctors be designated and approved as a clinical lab director.

Applications for CLIA certification (www.cms.gov/Regulations-and-Guidance/Legislation/CLIA/How_to_Apply_for_a_CLIA_Certificate/International_Laboratories.html). Having this certification for both the office and the physician is absolutely critical to perform and bill for these point-of-care tests.


a composite disease severity index was created from the following standard dry eye tests: TBUT, Schirmer I, corneal and conjunctival staining, meibomian grading, osmolarity, and the Ocular Surface Disease Index (OSDI) for symptomology.7 Each patient was assigned a severity grade between 0 and 1, with 0 representing the absence of disease and 1 representing the most severe form of dry eye.7 TearLab correctly identified 88% of the normal subjects, 75% of the mild and moderate disease patients, and 95% of the severe dry eye patients. Investigators determined a value of 308mOsm/L was the diagnostic cut off between “normal” and “mild dry eye.”7

According to colleagues who have incorporated TearLab into their practices, regional variability does exist for what is considered “normal” tear osmolarity. Geographical areas featuring lower humidity levels tend to show a higher baseline tear osmolarity. It may be important to evaluate your patient population and determine a normal range of osmolarity for your area.

Measurement of tear osmolarity is not only a diagnostic tool; it has potential for use in patient education and compliance. Patients who undergo treatment for dry eye and have repeated osmolarity testing will often ask for their “number” in hope that whatever treatment has been initiated has lowered their osmolarity score. You can use that concrete number, and patients’ interest in it, to spur adherence on their part.

- Testing for Sjögren’s. The autoimmune disease Sjögren’s syndrome affects the exocrine-producing glands throughout the body. Clinically, patients who present with Sjögren’s suffer from dry eyes and dry mouth. Due to its multisystem involvement, this condition
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is often difficult to diagnose, as a patient may end up seeking care from multiple specialists, who may overlook the overall condition, as each specialist only addresses their particular area of expertise without putting the entire picture together. Two forms of Sjögren’s syndrome exist: primary Sjögren’s syndrome, which has no additional autoimmune disease associated, and secondary Sjögren’s syndrome, which can present in conjunction with an additional autoimmune disease (usually rheumatoid arthritis). Patients are often symptomatic with Sjögren’s syndrome for upwards of 10 years before being diagnosed with the condition. This can result in significant quality of life concerns and may also be life-threatening, as Sjögren’s syndrome is associated with a 16-fold increased risk of development of lymphoma.

The Sjo (Nicox) test can help determine if a patient’s dry eye can be attributed to an underlying systemic etiology. A doctor or technician obtains blood on a collection card and sends it to a lab for analysis. A report is generated with results including rheumatoid factors, antinuclear antibodies, rho and la (Sjögren’s syndrome antibodies) and three biomarkers (salivary gland protein-1, carbonic anhydrase-6 and parotid secretory protein) unique to the test that the company says can pick up Sjögren’s earlier than the others.

An early diagnosis of Sjögren’s syndrome is crucial for the long-term management of these patients.

Diabetes
Diabetes is a major public health concern. Dealing with diabetes involves a complicated dance of managing blood sugar, blood pressure and cholesterol. Patients are asked to routinely check blood sugar levels, which gives them an indication of daily fluctuation. Additionally, an A1c (glycosylated hemoglobin) is a measure of a patient’s blood sugar over a three-month time period with normal values between 4% and 6%. A1c tests give the patient and practitioner an indication of the patient’s blood sugar control over an extended period of time.

When reviewing diabetic patients’ medical histories, patients are often hard-pressed to give concrete numbers with respect to blood sugar control and, in particular, being able to recall A1c values.

In-office A1c testing units provide POC testing that can give doctors those concrete numbers. Several CLIA waived POC A1c testing units are available on the market. Some check only A1c. Others have a combined function and analyze a variety of lab tests.

Patients’ development of complications secondary to diabetes, such as diabetic retinopathy, kidney and heart disease, and peripheral neuropathy, is dependent on a combination of length of the disease state, blood sugar, blood pressure and cholesterol control.

The ability for an optometric physician to obtain a current A1c value provides valuable management information.

Spaeth et al. assessed the use of point-of-care A1c testing in a
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remote village in Australia and determined that the use of POC A1c testing improved patient A1c values as compared to laboratory A1c testing.\textsuperscript{10} On average the testing was able to lower A1c values by 1.5% by providing patients with a reliable and rapid indication of blood sugar control as compared with lab testing, where blood samples had to be sent off for analysis and results often took several days to weeks.\textsuperscript{10}

**AMD**

Most patients with age-related macular degeneration are in the “dry” or non-exudative category where vision loss is modest and presents with retinal findings such as drusen, RPE clumping and potential atrophy. Patients who develop significant vision loss are typically in the “wet” or exudative stage, where significant vision loss are typically in the “dry” or non-exudative category where vision loss is modest and presents with retinal findings such as drusen, RPE clumping and potential atrophy. Patients who develop significant vision loss are typically in the “wet” or exudative stage, where

The addition of point-of-care testing gives optometrists a greater variety of diagnostic tools to consider and reduces our reliance on the old “wait-and-see” approach. Using these tests can provide doctors with first-hand evidence of disease—yielding an advantage in time and confidence that we can use to deliver early, targeted treatment.

The tests aren’t foolproof, and hundreds of other conditions are not currently within their purview, but this newer approach is a welcome complement to a clinical work-up.\textsuperscript{11}

- **Genetic tests.** Risk factors for the development of AMD include increasing age, smoking, UV exposure, family history and genetic profile. Genetic tests may provide a sense of risk of developing AMD.\textsuperscript{12}

  Macula Risk PGx (ArcticDx) combines a patient’s current AMD status, genetic predisposition and non-genetic risk factors to determine the two-, five- and 10-year risk of developing advanced AMD, defined as either geographic atrophy or choroidal neovascularization (CNV).\textsuperscript{12}

  RetnaGene (Valeant) is a genetic test for Caucasian patients age 55 and older with early or intermediate AMD that evaluates genotype, phenotype, age and environmental risk (e.g., smoking status) and predicts a patient’s individual risk for disease progression to CNV within two-, five- and 10 years. RetnaGene predicts a patient’s individual lifetime risk at age ≥5 for developing advanced AMD.\textsuperscript{12}

  Macula Risk PGx and RetnaGene tests rely on a sample of epithelial cells obtained by a buccal (cheek) swab and sent for analysis.

  The addition of point-of-care testing gives optometrists a greater variety of diagnostic tools to consider and reduces our reliance on the old “wait-and-see” approach. Using these tests can provide doctors with first-hand evidence of disease—yielding an advantage in time and confidence that we can use to deliver early, targeted treatment.

  The tests aren’t foolproof, and hundreds of other conditions are not currently within their purview, but this newer approach is a welcome complement to a clinical work-up.\textsuperscript{11}


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**Sjögren’s Syndrome Case**

**Courtesy of Dave Kading, OD**

A 38-year-old female presented with irritation and dryness. She noted the irritation had been increasing over the last few years. Dry eye evaluation noted a slight decrease in tear volume (through tear meniscus height evaluation), but what a decrease in the length of her meibomian glands was significant. A blink evaluation showed partial blinks in both eyes.

- **Testing.** The lids were normal in appearance, but with pressure (using the TearScience Meibomian Gland Expresser) we noted a significant decrease in the flow of meibum. Her InflammaDry test was negative for inflammation. We performed Sjö testing. Staining was grade 1 corneal and positive on the lid wiper area (defined as that portion of the marginal conjunctiva of the upper lid that wipes the ocular surface during blinking).

- **Diagnosis and Treatment.** After reviewing the tests, our assessment was dry eye secondary to MGD and gland atrophy. Our treatment plan involved treatment with Lifelip, lipid-based artificial tears and blink exercises.

- **Analysis.** At the patient’s follow up, she indicated improvement in symptoms. Clinically, we observed decreased lid wiper staining, normal meibomian gland secretion and resolution of the previous corneal staining. At the follow-up, we went over the Sjö test results; it indicated positive markers for Sjögren’s. This gave us the evidence we needed to begin the patient on a long-term Restasis ( cyclosporine, Allergan) regimen, continue blink exercises and refer her to rheumatology for further evaluation and management.
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Seeing Double:
The Urgent and Chronic Causes of Diplopia

Double vision can originate from many conditions. Learn to recognize when this presentation is an emergency and when it’s indicative of a long-term issue.

By Jim Williamson, OD

Optometrists are no strangers to double vision, but when a patient presents with it, ensuring that we correctly identify the cause can be a daunting task. While diplopia can present for numerous reasons, the first thing we need to do is either recognize or rule out urgent conditions, and then (in the latter case) explore the possibility that it may stem from a chronic condition. This article will focus on recognizing the chronic and urgent medical causes of diplopia. These will be loosely grouped by systemic disease, neurological etiologies and eye or orbital conditions.

First, we’ll review oculomotor control, as the diagnosis may involve systemic or neurological factors with potentially serious consequences.

Oculomotor Control
Two main pathways are involved in oculomotor function: the supranuclear and infranuclear pathways. The supranuclear route controls pursuits, saccades, vergences and horizontal and vertical gaze. As these are all symmetrical ocular movements, lesions here generally do not prompt diplopic complaints, though exceptions exist which will be discussed later.

CT scan of the same patient before and after the diagnosis of Graves’ disease. Note the markedly enlarged medial recti.

Release Date: March 2015
Expiration Date: March 1, 2018
Goal Statement: Double vision can occur from many serious conditions involving the neuromuscular anatomy and ocular media. This article will review the chronic conditions (e.g., myasthenia gravis, Graves’ disease, multiple sclerosis, diabetes) as well as urgent presentations (e.g., stroke, aneurysm, tumor, giant cell arteritis) that may cause it.

Faculty/Editorial Board: Jim Williamson, OD
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Disclosure Statement: Dr. Williamson has no relevant financial relationships to disclose.
The infranuclear pathway begins in each cranial nerve nucleus and proceeds to innervate the respective extraocular muscles (EOMs), as follows:

The oculomotor nerve, CN III, emerges from the anterior aspect of the midbrain. From there, it enters the subarachnoid space in the interpeduncular cistern between the posterior cerebral and superior cerebellar arteries and then runs lateral to the posterior communicating artery (PCA). After penetrating the arachnoid and internal layer of the dura, it moves into the cavernous sinus, where it lies superior to the trochlear nerve and lateral to the internal carotid artery. The nerve then splits into a smaller superior and larger inferior division, which enter the orbit through the superior orbital fissure. The superior division supplies the superior rectus and the levator palpebrae. The inferior division further divides into three branches to innervate the inferior oblique and the medial and inferior recti. Additionally, the longest inferior oblique branch has parasympathetic fibers that synapse in the ciliary ganglion. The postganglionic fibers pass through the short ciliary nerves to the sphincter pupillae and ciliary muscles.

The trochlear nerve, CN IV, is the only cranial nerve to emerge from the posterior aspect of the midbrain. As it supplies just one muscle—the superior oblique—the trochlear is the smallest of all cranial nerves and yet has the longest intracranial course. It moves laterally and forward in the subarachnoid space around the cerebral peduncle and enters the lateral wall of the cavernous sinus inferior to the oculomotor nerve. Continuing through the superior orbital fissure above the fibrous ring, it arrives at the superior oblique muscle as a series of small branches.

The abducens nerve, CN VI, leaves the ventral aspect of the brainstem between the border of the pons and medulla oblongata. It ascends through the subarachnoid space along the clivus as it courses anterolaterally through the pontine cistern. Following a sharp bend over the petrous part of the temporal bone (which makes it vulnerable to injury and increased intracranial pressure), it passes through the cavernous sinus where it first lies lateral and then inferolateral to the internal carotid artery. Like CN III and IV, the superior orbital fissure provides orbital access to the abducens nerve, where it enters the medial side of the lateral rectus muscle.

**Systemic Diseases**

Eye care practitioners must evaluate whether the onset of diplopia is a sign of a larger, systemic disease. Patients presenting with diplopia may or may not be aware of systemic conditions behind their visual symptom. These can include life-altering diagnoses such as Alzheimer’s, Parkinson’s or giant cell arteritis. Here are ways optometrists can learn to recognize the signs associated with these diplopia-causing systemic diseases:

- Myasthenia gravis. Optometrists are often the first to encounter myasthenia gravis (MG), as many of these patients have ocular sequelae such as ptosis and diplopia. In MG, acetylcholine receptor (AChR) antibodies block effective transmission of the chemical to muscle synapses, resulting in weakness. In addition to ophthalmic involvement, MG may also lead to arm and leg fatigue, difficulty chewing and swallowing, speech problems and, in severe cases, breathing impairment.

The key to distinguishing this from other neurological disorders lies with the fluctuation in symptoms, which are typically worse as the day progresses. Remember that ptotic patients may not complain of diplopia, due to occlusion. Extraocular muscles should be examined with the lid retracted. Statin use has been associated with MG and, though rare, should be considered, given the widespread use of this drug class.

Three quick and simple in-office tests can help confirm the suspicion of MG. First, ask the patient to sustain upgaze while you look for superior rectus or levator fatigue. Second, perform the highly specific ice pack test by placing ice on the eyelid for two minutes and evaluating for ptosis improvement.
Third, evaluate for orbicularis oculi weakness by attempting to open the patient’s eyelids after forceful closure. For bloodwork, order the AChR-antibody tests (binding, blocking and modulating), but keep in mind that this can be negative in 45% to 65% of patients with ocular symptoms only (i.e., ocular myasthenia gravis).

- Parkinson’s disease. Among the neurological conditions that can cause diplopia, only Alzheimer’s has a higher prevalence than Parkinson’s disease, so optometrists can expect to see many of these patients. Degenerating cells within the substantia nigra result in a biochemical loss of the neurotransmitter dopamine, which in turn may affect oculomotor control. Saccades, pursuits and vergences can all be involved. Dopaminergic treatment, though effective in providing some improvement in gross motor skills, has little effect on oculomotor deficits, so even well-controlled Parkinson’s disease patients can report visual changes. Remember when assessing EOMs in elderly patients that some restriction may occur even without a pathological cause. This typically involves upgaze and could be due to changes in orbital tissue. Though optometrists don’t treat Parkinson’s disease, some authors suggest obtaining an OCT scan to measure macular ganglion cell/inner plexiform layer thickness as a way to monitor its progression.

- Microvascular disease. In 2013 researchers reviewed 109 patients with isolated CN III, IV and VI palsies and identified microvascular ischemia as the cause in nearly 85% of cases. At risk are those with diabetes, hypertension or hyperlipidemia, as well as patients over 60. Because of this, consensus opinion varies regarding the need for costly neuroimaging of these infranuclear incidents. The decision to delay should coincide with the clinician’s ability to obtain a thorough history, uncover systemic symptoms and locate relevant ophthalmic findings. Experts agree, however, on the need for an immediate radiological work-up in the following scenarios:
  - patients younger than 50
  - history of cancer
  - evidence of combined cranial neuropathies
  - incomplete or pupil-involving CN III palsies

Regarding the latter, up to 20% of ischemic CN III palsies have anisocoria, but often less than 2mm. Features of aberrant regeneration of CN III indicate a non-microvascular etiology necessitating immediate work-up.

If the CN palsy is microvascular in etiology, it should show significant resolution in three months (the 90-day rule). If neuroimaging is not done, the patient needs to be monitored closely over the first several months. If the magnitude of the deviation worsens, or does not improve as expected with microvascular disease, obtain prompt neuroimaging.

- Orbital inflammatory disease. Thyroid eye disease can be caused by hyperthyroidism, Hashimoto’s thyroiditis and primary hypothyroidism. Of these, Graves’ disease is the most common. In this autoimmune disorder, antibodies bind to the thyroid surface and stimulate the overproduction of thyroid hormones, which leads to hyperthyroid-
isism. Approximately half of patients will develop thyroid-associated orbitopathy (TAO)—a type of orbital inflammatory disease.\textsuperscript{11} Extraocular muscle inflammation or fibrosis limits eye movement and produces diplopia. The inferior rectus is often affected first, followed by the medial, superior and lateral recti.\textsuperscript{16}

Smoking is a well-known, though not completely understood, risk factor for TAO. Some say it activates the pathways associated with adipogenesis and inflammation.\textsuperscript{17} Regardless, Graves’ disease patients should be advised to quit.

Several recent studies suggest a relationship between TAO and elevated immunoglobulin G4 (IgG4).\textsuperscript{18,19} They advocate screening for these levels to determine the likelihood of developing TAO.\textsuperscript{19} Patients with increased IgG4 tended to be older, responded well to antithyroid medications, and were more prone to hypothryoid after drug treatment.\textsuperscript{18}

Other systemic inflammatory conditions associated with orbital inflammatory disease include sarcoidosis and granulomatosis with polyangiitis (GPA), formerly known as Wegener’s. Non-caseating granulomas characterize sarcoidosis, along with hilar lymphadenopathy seen on chest X-ray and elevated serum levels of angiotension converting enzyme (ACE). GPA is a disease of small- and medium-sized blood vessels, with patients having abnormal levels of cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA). Additionally, infection and tumor must be considered in orbital inflammatory disease cases.

- **Giant cell arteritis.** The most common systemic vasculitis, giant cell arteritis is also the most feared from an ophthalmic standpoint due to the potential for permanent vision loss.\textsuperscript{20} While the incidence of diplopia with giant cell arteritis is small (between 2-15%),\textsuperscript{21,22} it deserves mentioning, especially if it occurs with other symptoms such as headache, neck pain, fever, malaise, weight loss, jaw claudication and temporal artery tenderness. Also, diplopia usually precedes visual loss and may be an early indication of giant cell arteritis.\textsuperscript{21} If suspected, immediately order an erythrocyte sedimentation rate and C-reactive protein. A positive temporal artery biopsy confirms the diagnosis,\textsuperscript{22} but a negative result does not exclude it.\textsuperscript{21} Prompt diagnosis and treatment with high-dose systemic corticosteroids are keys to a more favorable outcome.

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**Neurological Etiologies**

When double vision begins in the brain, the cause can range from a mild injury to serious conditions such as a tumor or aneurysm. To narrow down the possibilities, get a proper history and understand which patients are at-risk with the following guide:

- **Traumatic brain injury.** Types and severities of TBI range broadly from a mild concussion to those involving loss of consciousness, post-traumatic amnesia, disorientation or other neurological disorders.\textsuperscript{23} Dysfunctions of oculomotor control may be seen with all classifications. Symptoms include blurred vision, difficulty following targets, reading problems and diplopia.\textsuperscript{24} It is important to note that some patients experience a delayed onset of symptoms—therefore, a remote history of head trauma could prove significant. One of the most common issues in mild TBI is convergence insufficiency.

- **Supranuclear lesions.** As noted previously, the supranuclear pathway controls symmetrical eye movements; thus, lesions here should not cause diplopia. The two main exceptions are internuclear ophthalmoplegia (INO) and skew deviation. INO stems from a lesion in the highly myelinated medial longitudinal fasciculus, which connects the abducens nuclei in the pons to the oculomotor nuclei in the midbrain.\textsuperscript{25} Patients with INO are unable to adduct and may have a nystagmus when abducting the fellow eye. A bilateral INO presentation or occurrence in a young individual frequently indicates multiple sclerosis (MS), while vascular ischemia accounts for the majority of unilateral or elderly INO cases.\textsuperscript{26} Convergence may be affected in some patients, depending on the location of the lesion. The center for convergence is in the midbrain. So, midbrain lesions affect convergence while pontine lesions spare it.

Skew deviation is a vertical misalignment that does not localize to any one particular cranial nerve or cyclovertical muscle (i.e., superior and inferior recti or obliques). Skew deviation most commonly results from faulty prenuclear vestibular input to the ocular motor nuclei or damage to the MLF in the brainstem.\textsuperscript{27,28} It is most commonly associated with cerebellum, brainstem or vestibular lesions. When skew deviation is accompanied by ocular torsion and head tilt, it is called the ocular tilt reaction.\textsuperscript{28}

### Table 1. Questions to Ask Patients with Diplopia

- Does it go away when you cover one eye?
- Are the images side-by-side or on top of each other?
- Is it better or worse at distance or near?
- Is it constant or variable?
- Do you experience any associated pain?
- How long has this been occurring?
- Have there been any previous episodes?
- Have you noticed a droopy eyelid?
**Aneurysm.** A common location for intracranial aneurysms is the junction of the internal carotid artery and the posterior communicating artery.²⁹ It should be no surprise, then, that an aneurysm in this area puts the oculomotor nerve at risk, since it runs lateral to the PCA. Also, since pupillomotor fibers run on the surface of CN III, a compressive aneurysm would likely produce pupillary involvement, which may be the first feature. An aneurysm should be ruled out in any CN III palsy with associated pain or full/partial pupillary involvement, especially in patients less than age 50 or without vasculopathic risk factors.³⁰ Standard of care in these patients dictates emergent referral with immediate imaging. While the gold standard for aneurysm detection is cerebral catheter angiography, computerized tomographic angiography (CTA) is preferred.³¹ Should a contraindication exist for contrast dye (e.g., pregnancy, allergy, impaired renal function), a magnetic resonance angiography (MRA) would be performed. CTA takes only a few minutes, with the bulk of time spent on image post-processing. It outperforms MRA in detecting aneurysms <5mm by an impressive 94% to 54% margin, making it an ideal test, given the typical size of a third-nerve palsy aneurysm.³¹

- **Cavernous sinus.** Since the cavernous sinus plays host to all the oculomotor nerves, any primary or extending secondary pathology affecting this structure may produce diploic complaints. Two processes that originate here are carotid-cavernous fistula (CCF) and Tolosa-Hunt syndrome (THS).

A CCF is an abnormal communication that allows blood to flow from the carotid artery into the cavernous sinus. The broad classifications of CCF are direct vs. indirect. A direct CCF arises from a traumatic or aneurysmal rupture etiology, progresses rapidly and requires urgent treatment.³² In contrast, an indirect CCF tends to be more insidious in onset, leading to a delay in diagnosis due to its chronic nature.³² Clinicians should add indirect CCF to their differentials when treating conjunctival injection, as this is a common ocular sequelae.

THS is an idiopathic, nonspecific inflammation of the cavernous sinus, causing painful ophthalmoplegia. These changes should be evident on neuroimaging, but not always. The term “benign THS” applies when this occurs.³³
• **Tumor.** A pituitary adenoma is a familiar entity in the eye care field due to its potential ophthalmic manifestations. Two types exist, differentiated by size: microadenoma (<10mm) and macroadenoma (>10mm). Only the latter produces ocular findings, due to compression of the optic chiasm and resulting visual field defects. Further expansion of the tumor into the cavernous sinus may elicit a CN III, IV or VI palsy. Of those, CN III and the levator palpebrae superioris are the most affected nerve and muscle, respectively.34

While a macroadenoma itself is not typically an urgent situation, acute hemorrhage or ischemia within the pituitary tumor produces the emergent and possible life-threatening condition pituitary apoplexy (PA). Headache, nausea and impaired mental status join decreased visual acuity and ophthalmoplegia as common presenting symptoms, which develop from hours to days, or longer.35

Eye and Orbital Conditions

When neither a systemic disease nor a neurological condition can explain diplopia, the problem may be limited to the eye itself. Here are several possible eye and orbital conditions that can lead to double vision:

• **Herpes zoster ophthalmicus.** Ocular complications arise when the latent varicella zoster virus (VZV) invades the ophthalmic division of CN V during a shingles outbreak, resulting in herpes zoster ophthalmicus (HZO). Cranial nerve palsies occur in up to 30% of HZO patients.36 CN III is affected the most, followed by CN VI and CN IV.37 Perform a careful EOM evaluation, as over 25% remain asymptomatic with diplopia only being noticed in extreme gaze.38

A gradual decline in immunity to varicella zoster virus has led to increased rates of herpes zoster (HZ) and postherpetic neuralgia (PHN).39 Some suggest the cause is decreased exposure to VZV stemming from the 1996 recommendation for immunization in children. Repeated exposure, they say, boosts a person’s cell-mediated immunity against the virus. However, one of several studies does not support that conclusion and notes accelerated rates even before the vaccine’s introduction.40

The Advisory Committee on Immunization Practices (ACIP) and Centers for Disease Control (CDC) recommend the 2006 FDA approved zoster vaccine Zostavax for patients age 60 or older. The Shingles Prevention Study showed markedly reduced morbidity from HZ and PHN and an over 50% reduction in HZ incidence.41 Screen younger patients with HZ for an underlying immunocompromising etiology, such as HIV.

• **Idiopathic orbital inflammatory disease.** After ruling out all potential causes of OID, the clinician is left with the diagnosis of exclusion—idiopathic orbital inflammatory disease (IOID). Oral steroids remain the definitive treatment for IOID, with the expected course being rapid improvement
on these agents. It is of note that lymphoma can mimic IOID, and can initially respond to steroid treatment, delaying its diagnosis and proper treatment.

Immunosuppressive drugs can also be used. Some cases are associated with abnormal IgG4 levels, where patients initially respond to glucocorticoids but relapse during taper.

Several have reported suc-

- **Orbital blow-out fractures.** Ocular trauma renders the small and thin orbital bones susceptible to damage, with orbital floor fractures being the most common. Since the infraorbital nerve runs through this area, patients often cite hypoesthesia along its distribution (i.e., cheek and upper lip). The incidence of diplopia, usually involving upgaze, varies; it has been reported to be from 20% to 45%. The diplopia occurs due to extraocular muscle edema, nerve palsy or an entrapped muscle within the fracture. A longstanding entrapment often leads to fibrosis.

Though typically not emergencies, urgent surgery should occur if the ocularadical reflex can be stimulated, a muscle is entrapped or the oculocardiac reflex can be stimulated. A longstanding entrapment often leads to fibrosis.

**Superior orbital fissure syndrome.** The superior orbital fissure provides orbital access to not only CN III, IV and VI as previously discussed but also the frontal, lacrimal and nasosial branches of the ophthalmic nerve. An infiltrative, inflammatory, ischemic or traumatic event affecting this 3mm x 22mm area is called superior orbital fissure syndrome (SOFS). With all the nerves traversing through this location, SOFS results in ophthalmoplegia, forehead and upper eyelid hypoesthesia, ptosis and pupillary mydriasis.

Loss of extraocular muscle tone—which usually exerts a retracting force on the globe—leads to proptosis. Vision loss indicates optic nerve involvement, and when this occurs the diagnosis changes from SOFS to orbital apex syndrome (OAS). The latter is a more urgent condition that may require early intervention such as high-dose corticosteroids or surgical decompression of the optic canal, depending on the etiology.

**Conclusion**

Though neurologic imaging is important when dealing with diplopia, it is only helpful when one knows the anatomic location upon which to focus attention. Nothing can replace a thorough history and clinical exam. Using these, ophthalmologists can try to localize the diplopia and determine if it is likely a process of systemic or neurologic disease, or an orbital condition. This will help establish the urgency of the situation and the need for additional imaging, treatment or further referral.
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1. The oculomotor nerve, CN III, runs lateral to this artery:
   a. Posterior cerebral artery.
   b. Anterior communicating artery.
   c. Posterior communicating artery.
   d. Anterior cerebral artery.

2. Which of the following cranial nerves makes a sharp bend over the petrous part of the temporal bone, making it vulnerable to injury and increased intracranial pressure?
   a. VI.
   b. IV.
   c. III.
   d. II.

3. The ice pack test is performed by placing ice on the eyelid for:
   a. Two hours.
   b. Two minutes.
   c. Two seconds.
   d. 10 minutes.

4. Patients at risk for a microvascular cranial nerve palsy are those with:
   a. Diabetes.
   b. Hypertension.
   c. Hyperlipidemia.
   d. All of the above.

5. All of the following require immediate neurological imaging, except:
   a. Patients with combined cranial nerve palsies.
   b. A 35-year-old with diplopia and a history of cancer.
   c. Incomplete or pupil-involving CN III palsy.
   d. Isolated CN VI palsy in a 65-year-old uncontrolled diabetic.

6. Which rectus muscle is often affected first in Graves’ disease?
   a. Superior.
   b. Medial.
   c. Inferior.
   d. Lateral.

7. Several studies suggest a link between thyroid-associated orbitopathy (TAO) and elevated:
   b. IgG3.
   c. IgG2.
   d. IgG1.

8. Patients with INO from a midbrain lesion are unable to__________ and also unable to__________.
   a. Abduct, diverge.
   b. Adduct, converge.
   c. Abduct, converge.
   d. Adduct, diverge.

9. Which of the following is false regarding a skew deviation?
   a. Commonly associated with cerebellum, brain stem or vestibular lesions.
   b. Results from faulty prenuclear vestibular input to the ocular motor nuclei.
   c. Will localize to one specific cyclovertical muscle (i.e., superior and inferior recti or obliques).
   d. May be accompanied by ocular torsion and head tilt.

10. The preferred imaging technique for aneurysm detection is:
   a. MRA.
   b. CTA.
   c. X-ray.
   d. MRI.

11. The cavernous sinus plays host to which of the following cranial nerves?
   a. CN II.
   b. CN IV.
   c. CN VI.
   d. All of the above.

12. An idiopathic, nonspecific inflammation of the cavernous sinus causing painful ophthalmoplegia is called:
   a. Ophthalmoplegic migraine.
   b. Traumatic brain injury.
   c. Tolosa-Hunt syndrome.
   d. Cavernous sinus fistula.

13. A microadenoma:
   a. Measures <10mm.
   b. Always compresses the optic chiasm.
   c. Frequently causes visual field defects.
   d. Measures >10mm.

14. Approximately what percentage of patients with herpes zoster ophtalmicus (HZO) experience cranial nerve palsy?
   a. 90%.
   b. 60%.
   c. 30%.
   d. 10%.

15. All of the following are true, except:
   a. There has been a gradual decline in immunity against the varicella zoster virus.
   b. CN III is affected the most, followed by CN
VI and CN IV.
c. Ocular complications arise when the latent varicella zoster virus invades the ophthalmic division of CN V.
d. All herpes zoster patients with cranial nerve palsies are symptomatic.

16. Regarding the zoster vaccine Zostavax:
a. It is recommended for patients age 40 or older.
b. It resulted in an over 50% reduction in herpes zoster incidence.
c. It resulted in increased morbidity from herpes zoster.
d. It is only recommended in immunocompromised patients.

17. Idiopathic orbital inflammatory disease is treated with:
a. Topical steroids.
b. Oral steroids.
c. Oral antibiotics.
d. Tylenol.

18. Diplopia from an orbital blow-out fracture usually involves:
a. Upgaze.
b. Downgaze.
c. Lateral gaze.
d. None of the above.

19. The superior orbital fissure provides orbital access to all of the following cranial nerves, except:
a. CN III.
b. CN IV.
c. CN VI.
d. CN VII.

20. Which of the following is/are signs of superior orbital fissure syndrome (SOFS):
a. Ptosis and pupillary mydriasis.
b. Forehead and upper eyelid hypoesthesia.
c. Enophthalmos.
d. Only a and b.

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It’s the end of a busy morning and you’re just about to see your last patient before a well-deserved lunch. You walk in and the patient says, “Doctor, I have tears running down my left cheek all the time.”

Put lunch on hold.

For most doctors, epiphora may not be as daunting as the dreaded double vision complaint; however, determining the cause of epiphora can be stressful and time-consuming.

For whatever reason, many eye care providers dread or fear performing lacrimal dilation and irrigation, which often is essential to determine the cause of the patient’s epiphora.

Perhaps this is because it involves putting an extremely sharp, pointed metal object into the patient’s punctum. Or maybe it’s because you remember learning it in optometry school, but you haven’t performed it in quite a long time.

Fear not. It’s a relatively simple procedure that’s often indicated to treat or diagnose several common ocular conditions. The aim of lacrimal irrigation is straightforward: to open an obstructed tear duct and allow the tears to flow properly. This article—the third in a six-part, print-and-video, how-to series—explains how easily lacrimal dilation and irrigation can be done.

Know the Anatomy
Before performing lacrimal dilation and irrigation (D&I), be sure you understand the anatomy of the nasolacrimal drainage system. The puncta are the openings to the drainage system, with aperture sizes that vary from patient to patient. Both the superior and inferior punctum open into a superior and inferior canaliculus, respectively.

The canaliculus has a vertical...
portion that extends approximately 2mm in length before bending horizontally and running another 8mm in length nasally. The two canaliculi join at the common canaliculus just before entering into the nasolacrimal sac, which is located within a fossa in the medial orbital wall.

The nasolacrimal sac extends roughly 10mm before draining into the nasolacrimal duct, which is located within the maxillary bone. This extends to the inferior meatus of the nose and ends at the valve of Hasner. This valve prevents the backflow of fluid from the nasal cavity up into the duct.1

Causes of Epiphora
Potential causes of epiphora include:
• Lid appositional abnormalities—entropion, ectropion and floppy eyelid syndrome
• Ocular surface disorders—including trichiasis, abrasion, foreign body, dry eye syndrome and recurrent corneal erosion
• Obstructed tear flow tract—canicular, punctal or nasolacrimal duct obstruction
• Infectious causes—canaliculitis and dacryocystitis

Nasolacrimal duct obstruction can be acquired, which is often due to aging, or the obstruction can be congenital. Infants may suffer from congenital obstruction of the nasolacrimal duct when the valve of Hasner fails to spontaneously open at birth. This resolves on its own in about 90% of cases before the child is one year old. So, it’s not usually recommended to perform dilation and irrigation on a child younger than one year.2

You must determine the underlying cause of the epiphora prior to considering lacrimal dilation and irrigation, as it’s not indicated in all of these cases. A diagnosis of dacryostenosis is typically considered after you rule out lid malposition and ocular surface disorders. Dry eye syndrome is usually the fallback diagnosis, but consider it only after exhausting other possibilities.

Be sure to perform a thorough slit lamp examination to determine if dilation and irrigation is indicated. If a patient complains of sudden-onset tearing and a painful eye upon waking, for instance, then the diagnosis and cause of the tearing is most likely a recurrent corneal erosion, and D&I is likely not needed.

Indications
Consider performing lacrimal dilation and irrigation when a patient has a complaint of epiphora and you suspect an obstruction in the nasolacrimal drainage system. Lacrimal irrigation is advised in canicular obstruction, which often occurs from stenosis, mucous plugs or small stones in the tear ducts. In this case, the patient will exhibit epiphora, typically unilaterally, in a white and quiet eye.

Performing irrigation will either clear the obstruction or allow you to determine the location of the blockage. Depending on the cause and severity of the obstruction, a dacryocystorhinostomy (DCR) may be indicated.3

Thorough evaluation is also necessary to rule out infectious causes of epiphora, such as canaliculitis and dacryocystitis. In the case of canaliculitis, the puncta will be “pouting” (red and turning outward), and the surrounding area will be erythematous. With slight palpation, you’ll often be able to excrete mucopurulent discharge or concretions from the punctum. Treatment varies among clinicians; however, lacrimal irrigation with an antibiotic solution is a viable option to consider, as well as topical and/or oral antibiotics.

In cases of dacryocystitis, more swelling, tenderness and pain over the lacrimal sac will be observed. Don’t perform irrigation in the case of dacryocystitis; instead, prescribe oral antibiotic treatment, such as amoxicillin/clavulanate 500/125mg TID.2,3

Lacrimal dilation and irrigation can be performed behind the slit lamp or outside of it. Once the patient is in position, apply a drop of topical anesthetic to the eye.
Equipment
- Lacrimal dilator
- 27- to 19-gauge cannula (depending on the patient’s punctum size)
- 3cc syringe
- 1cc to 3cc sterile saline

Get in Position
Lacrimal dilation and irrigation can be performed either behind or outside the slit lamp. A benefit of performing D&I outside the slit lamp is the easier accessibility to the patient and more freedom of movement; however, one of the downsides is not having the magnification the slit lamp offers. A benefit of performing D&I behind the slit lamp is that it allows you to more accurately assess the punctum with the increased magnification. The increased magnification enables you to better determine exactly where to place the dilator and the cannula.

We prefer performing dilation and irrigation outside the slit lamp with our patient reclined to a height that is comfortable for us to easily manipulate the punctum from above. Keep in mind, with her or his head reclined, the patient may experience a slight gag reflex when they taste the saline.

We also adjust the stand lamp so the light shines directly on the eye undergoing the procedure.

How to D&I
Before dilation and irrigation (D&I), numb the punctum for patient comfort.
- Apply proparacaine or another topical anesthetic to a cotton-tip applicator and gently rest it on the patient’s punctum for at least 30 to 60 seconds.
- Roll the dilator between your thumb and index finger to achieve proper dilation.

Pull the patient’s lid temporally and insert the dilator vertically approximately 1mm to 2mm into the punctum.

A drop of proparacaine into each eye also helps eliminate a blink reflex.
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Once the patient is sufficiently numbed, begin the procedure.

- It’s a good idea to tell your patients to look “up and away” or “down and away” from the punctum being dilated, so they don’t become apprehensive at seeing the lacrimal dilator.

- Pull the patient’s lid temporally and insert the dilator approximately 1mm to 2mm vertically into the punctum. Start with the smaller end of the dilator and switch to the larger end once the punctum is sufficiently dilated.

- To achieve proper dilation, roll the dilator with your thumb and index finger or move it in a circular manner.

- After sufficient vertical dilation, re-orient your dilator almost 90 degrees horizontally, all the while keeping your dilator in the patient’s punctum, and continue in the same rolling or circular manner.

- Once you’ve dilated the punctum enough for your cannula to comfortably fit, you know you’ve achieved sufficient dilation.

- After you’ve dilated the punctum, remove the dilator and insert the cannula attached to the saline-filled syringe, in the same manner as the dilator; first vertically 1mm to 2mm with the lid pulled temporally, then re-orient it horizontally approximately 8mm. Take caution when moving the cannula horizontally and nasally; if you go too far, you’ll hit nasal bone and cause some discomfort for the patient.

- Gently press the plunger on the syringe to release the sterile saline into the patient’s lacrimal system.

**Results**

If the lacrimal system is open, the patient will feel or taste the salty
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saline in the back of his or her throat. With this result, the cause of the epiphora is likely not nasolacrimal duct obstruction. If the lacrimal system is blocked, you'll feel resistance of the plunger and see regurgitation of the saline. If regurgitation is coming out of the punctum being irrigated, then the blockage is proximal to the common canaliculus. If regurgitation happens in the opposite punctum, then the blockage is distal to the common canaliculus. Either way, further lacrimal probing is indicated.

Or, you can attempt to remove the blockage, or move it farther down the lacrimal drainage system, by irrigating multiple times—you may need to irrigate more forcefully for this to occur.

If you are still unsuccessful after several attempts to move the blockage, refer the patient for lacrimal probing.

If, however, you are able to successfully move the blockage and the patient can taste or feel the saline, no further treatment is necessary—these patients generally do not need to be seen for follow-up.

Billing and Coding
A simple procedure code of 68810 (“Probing of nasolacrimal duct, with or without irrigation”) is used with a –LT or –RT modifier stating which punctum was dilated and irrigated. The ICD-9 diagnosis codes to accompany the procedure code may include epiphora (375.20), nasolacrimal duct obstruction (375.55) and nasolacrimal duct stenosis (375.56).

As with any patient, always document thoroughly. Include the patient’s symptoms, why you chose to dilate, how you performed the procedure and the results you achieved. Also document how the patient tolerated the procedure and when you expect them to return for follow-up.

As you can see, lacrimal D&C is actually a simple procedure to perform and needn’t be dreaded or avoided when clinically indicated. Knowing the anatomy of the nasolacrimal drainage system well will remind you of the proper placement and movement of the dilator and cannula.

When performed correctly, this quick procedure helps your patients stop crying, gets them smiling again and gets you on your way to an enjoyable lunch.

Dr. Provence-Perry is currently completing an ocular disease and family practice residency, with an emphasis in disease management and anterior segment laser procedures, at Northeastern State University Oklahoma College of Optometry.

Dr. Smith is currently completing a residency in vision rehabilitation: low vision and neuro-optometry at NSU Oklahoma College of Optometry.

Dr. Lighthizer is the assistant dean for clinical care services, director of continuing education, and chief of both the specialty care clinic and the electrodiagnostics clinic at NSU Oklahoma College of Optometry.

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SYSTANE® Brand products are formulated for the temporary relief of burning and irritation due to dryness of the eye.

Continuing education classes at Vision Expo East, held March 19-22, allow eye care professionals from around the world to explore new ideas and exchange updates on the latest in health, technology and business-building strategies.

Cultivating CL Knowledge

Returning to Expo after what Mark Dunbar, OD, co-chairman of the VEE Conference Advisory Board, deemed a “hugely successful debut last year at Vision Expos East and West,” the Global Contact Lens Forum (GCLF) once again hosts an ample supply of classes pertaining to the business and the craft of fitting contact lenses. This forum marks the 7:00 AM start to classes at Expo on Thursday. Topics include: the art and science of managing astigmatism with contact lenses; successful scleral lens fitting; troubleshooting cases; and how to ensure that the dollars and “sense” of a contact lens practice allow room for growth.

Experts will envision advancements and predict upcoming trends by sharing updates on disease and technology, as well as a “farmer’s almanac” view of the future of contact lenses. Although pre-registration is required, there is no fee for signing up, and those who register may accrue six CE credit hours.

If the early start to classes on Thursday poses an issue, don’t fret. There are over 15 credit hours of other contact lens-related courses available outside of the GCLF. These courses are scattered throughout the class schedule, with the first one, “Hashtag Contact Lens: What’s New and What’s Coming,” held on Thursday at 1:30 PM.

Sowing Seeds of Wellness

Healthy eyes begin with healthy bodies, so it is essential for eye care practitioners to have the tools to educate patients on the importance of regular checkups and keeping their eyes and bodies well nourished.

“For the very first time at Vision Expo, we are introducing our Ocular Wellness Program, which will debut on Thursday afternoon,” says Dr. Dunbar. “Optometry needs to take a leading role in helping to inform and educate our patients on the need for ocular wellness.”

This three-hour program uses evidence-based medicine to provide clinical guidelines that emphasize the advantages of preventive medicine and focus on improving each patient’s quality of life. “Through our Ocular Wellness Program, we are hoping to create awareness to our patients and colleagues of the importance of preventing ocular problems before they develop, with...
the overall goal for our patients of maintaining good vision and healthy eyes for a lifetime,” Dr. Dunbar says.

Other classes that highlight health and wellness can be found throughout the lineup at Expo, including: “Feed Your Retina: Nutrition and Retinal Health”; “Diabetes: What we ALL Need to Know”; and “Omega 3 Fish Oil—Benefits for Your Patients and Practice.”

Combating Disease
Another way we can help our patients’ health thrive is to arm ourselves with the knowledge necessary to recognize problems early and to treat them successfully with the most up-to-date, proven techniques. “We will have the full armament of ocular disease clinical courses [at Expo],” says Dr. Dunbar, “focusing on glaucoma, external disease, nutrition and retinal disease among others.”

Within the Friday schedule is the “Greatest Ever” series—a set of courses on managing ocular disease. The first class of the series, “The Greatest Anterior Segment Disease and Medical Management of Contact Lens Complications Course—Ever!” takes place in the morning, and its complement, “The Greatest Posterior Segment Disease Course—Ever!” takes place in the afternoon. These classes are designed to enhance the practitioner’s clinical diagnostic and treatment skills and provide strategies on handling a patient suffering from problems related to medical vision correction options as well as retinal disease.

On Saturday, the entertaining and knowledgeable duo of Drs. Ron Melton and Rondall Thomas is back, offering six hours of disease-oriented continuing education classes, including: “What the Primary Care Optometrist Should Never Miss,” “Good, the Bad and the Ugly—Is it Glaucoma or Not?” and “Mystery Solved—Conquering Anterior Segment Pathology.”

Exploring New Technologies
One of the enhancements to some lectures this year is the use of technology to increase speaker-attendee interaction. “This past year we have broadened our approach to deliver education in more interesting and creative ways, including our Crowd-Sourced Learning courses that use smartphone and polling technologies,” says Dr. Dunbar. Specific classes will allow audience members to participate through their mobile device or audience response system (ARS) technology. These technologies will help attendees become more engaged by being able to answer poll questions, suggest topics and request specific information. “We launched this at Vision Expo West, and it was a huge success. We think it will be even better for Vision Expo East this year,” says Dr. Dunbar.

There are also plenty of classes to help businesses reach new heights. This year they “have expanded the Business Solutions curriculum to include over 100 hours of education on a broad range of topics, including the popular Visionomics Program, Frame Buyers Program, billing and coding, as well as a Retail track and the Wearable Technology track. We have even brought in The Ritz-Carlton for our Spotlight series discussing Memorable Customer Service Experiences,” Dr. Dunbar says.

He adds that there will also be “unique courses delivered through our Optical Technology tracks by some of the top lecturers in the industry, including Edward De Gennaro, Laurie Pierce, Michael Vitale and Barry Santini, among others. Courses will include the latest in lens technologies as well as the popular Optical Bootcamp.”

Eye Care for Athletes
Sundays aren’t always for resting; they’re also for sporting events—and Sunday has a lineup of eight credit hours that cannot be defeated. Kicking off at 7:15 AM, the Sports Vision CE classes are designed to give eye care professionals proficiency and an appropriate game plan on how to draft athletes to their practice, help them enhance their performance, protect them from injury—and grow profits in the process. By pairing these classes with the wide variety of courses available at Vision Expo East this year, attendees are sure to walk away with a record-breaking score of eye care and eyewear know-how.

Visit www.visionexpoeast.com to see the full program.
Exam Reveals Subtle Macular Disease

A middle-aged patient was seeing spots, prompting an uncommon diagnosis of idiopathic juxtafoveal retinal telangiectasis. By Erin S. Cooper, OD, Paul J. Gruosso, OD, and Joseph Miller, OD

Diabetic juxtafoveal retinal telangiectasis (IJRT) is a rare disorder. The condition is discernible by clinical presentation along with the use of fluorescein angiography and ocular coherence tomography.

IJRT is subdivided into different types, and further subdivided into subtypes and stages. The treatment of the disease is controversial and mostly depends on the patient's level of symptoms and clinical signs.

History
A 55-year-old white male presented with an emergent complaint of increased black spots in the vision of his right eye. He had noticed that the black spots had been increasing in size and number, along with increased blurry vision over the past six to eight months. His past ocular history was unremarkable. He had not had an eye exam in over three years.

His systemic history was positive for poorly controlled Type 1 diabetes; his last A1c reading was 9.8. He also reported hyperlipidemia and hypertension. His blood pressure was 132/78 the morning of the exam. Systemic medications included temazepam, atenolol, glipizide, insulin, metformin, omeprazole, multivitamins and aspirin (325mg).

Diagnostic Data
Visual acuity at presentation was 20/60 OD with pinhole to 20/40 and 20/30 OS with pinhole to 20/25. Following refraction, the patient's best-corrected visual acuity was 20/40 OD and 20/25 OS. The refraction yielded -1.00 DS in the right eye and -0.50 DS in the left eye. The patient had no deficiencies on extraocular motility or confrontation visual field testing. He had equal, round, reactive pupils with no afferent defect. Amsler grid revealed metamorphopsia nasally in both eyes.
Biomicroscopy uncovered trace seasonal allergic conjunctivitis with mild papillae and punctate keratitis. He had mild nuclear sclerosis in the lenses of both eyes. Goldmann applanation tonometry pressures measured 18mm Hg OU.

Dilated fundus exam revealed two small dot hemorrhages inferior to the central macula with a few scattered hemorrhages temporally, along with a pseudohole appearance in the macula of the right eye. The macula of left eye had a slightly elevated appearance with pigment mottling and a few small hemorrhages surrounding it. The left eye also had a pseudohole appearance. Both eyes had a negative Watske-Allen sign.

Optical coherence tomography (OCT) showed a subfoveal macular cyst-like appearance secondary to atrophy that affected the foveal contour in both eyes.

**Diagnosis**

Based on the OCT results, we tentatively diagnosed the patient with idiopathic juxtafoveal retinal telangiectasis. We referred the patient to a retina specialist, who confirmed the diagnosis of type 2 IJRT using fluorescein angiography.

Fluorescein angiography is the diagnostic tool of choice for IJRT. The irregular dilation of capillaries is easily seen on the arteriovenous phase, along with leakage of dye from the affected capillaries into the intracellular spaces of the retina. In our patient, both eyes showed punctate hyperfluorescent spots within the central macula in the early phases, along with leakage seen in the late phases.

OCT is another important diagnostic tool used in this disease. Features of IJRT seen on OCT are most commonly loss and disruption of the photoreceptor layer, cyst-like structures in the fovea and within intra-retinal layers, possible neovascularization, and intra-retinal OCT scans, both OD (left) and OS (right), show subfoveal macular cyst-like appearance due to atrophy affecting the foveal contour.

---

**Fluorescein angiography (FA) in the patient’s right eye: early arteriovenous phase (left) and late arteriovenous phase (right).**

**FA in the left eye: early arteriovenous phase (left) and late arteriovenous phase (right). Both eyes show punctate hyperfluorescent spots within the central macula in the early phases, and leakage in the late phases.**
deposits and plaques. The discontinuity of the photoreceptors is correlated with loss of visual acuity. If the photoreceptor layer is intact, the visual acuity is usually preserved. Gass concluded the loss of central vision is due to photoreceptor atrophy in the absence of macular edema. Another unique feature seen on OCT is the internal limiting membrane (ILM) drape, as seen in our patient. The draping of the ILM over the fovea is due to underlying loss of tissue, causing a cystoid space. This draping shows the loss of the outer plexiform layer due to a cystoid space underneath the fovea that allows the retina to have a normal foveal contour and foveal thickness. Foveolar atrophy is easily seen on OCT and may mimic a lamellar macular hole. This atrophy is the main cause of slow progressive vision loss over time, whereas rapid and severe vision loss occurs with the development of a neovascular membrane.

Treatment and Follow-up
The patient did not receive any treatment for this condition. He will be monitored by the retina specialist closely on a monthly basis initially, with the goal of every other month for the balance of the year. He was dispensed an Amsler grid and educated on the signs and symptoms of decompensation. After his initial visit, the patient missed his two follow-up appointments and returned for a three-month follow-up and then again two months later. The patient’s visual acuity had slightly decreased in subsequent follow-up visits.

Discussion
Idiopathic juxtafoveal retinal telangiectasias are a group of rare retinal vascular disorders characterized by alterations of the juxtafoveal capillary network in one or both eyes that is detectable even in asymptomatic patients. This disease is more specific to the parafoveal region of the retina. Therefore, it becomes easily distinguishable from other generalized retinal telangiectasias such as Coats’ disease or a secondary telangiectasis found in retinal vein occlusions, carotid artery disease or diabetes.

IJRT was first identified by Gass and Oyakawa in 1982. They classified the disease into four groups based on clinical features and results from fluorescein angiography. Gass and Blodi updated this classification system in 1993. They divided IJRT into three groups with smaller subgroups within the first group. (See “Classification of IJRT by Type,” below.) This most recent classification system is widely accepted and described as follows:

- **Type 1.** The hallmark characteristic of type 1 is variable sized aneurysmal dilations and easily visible telangiectasis of the retinal capillaries.

  Type 1 is subdivided into types 1A and 1B. Each are unilateral and congenital, typically affecting men in their 30s and 40s. The vision loss in type 1A is more significant than in type 1B.

  **Subgroup 1A** is the second most common of all types and can be found in males ranging in age from 15 to 54. The telangiectasis of the capillaries is typically found in the temporal half of the macula involving an area of two disc diameters or greater. In these patients, the decreased vision ranges from 20/25 to 20/40.

  **Subgroup 1B** is rare and affects mostly middle-aged men between

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<thead>
<tr>
<th>Classification of IJRT by Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Frequency</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Congenital/acquired</td>
</tr>
<tr>
<td>Laterality</td>
</tr>
<tr>
<td>Visual acuity at presentation</td>
</tr>
<tr>
<td>Classic signs</td>
</tr>
<tr>
<td>Systemic associations</td>
</tr>
</tbody>
</table>
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the ages of 40 and 54. The difference between subgroup 1B and 1A differs primarily in the size of the area of telangiectases. These telangiectases occupy small focal areas less than one disc diameter adjacent to the foveal avascular zone. The amount of vision loss in this subgroup is much less where the men are either asymptomatic or have vision better than 20/25.

In both subtypes, cystoid macular edema may develop and will show classic petaloid leakage on fluorescein angiography. Macular edema and lipid exudation are the main cause of vision loss in type 1.

- **Type 2.** Type 2 is bilateral, acquired and has no gender predilection. This is the most common type of IJRT and is significantly different from type 1. Although type 2 is considered the most common, its prevalence was found to be very low—0.1%—by the Beaver Dam Eye Study and even lower—0.004% to 0.022%—in the Melbourne Collaborative Cohort Study. Age of onset is typically in the 40s or 50s.

The most common clinical sign in type 2 is foveolar atrophy. The characteristics are usually symmetric, but vision loss may be asymmetric. Vision loss ranges from asymptomatic to mild blurring of central vision to slow progressive loss of central vision. The vision loss can occur slowly, but may become rapid with the development of intra-retinal neovascularization. Some research suggests that diabetes may be an underlying cause for this group.

IJRT type 2 is further subdivided into five stages (see “Stages of Type 2 IJRT,” right) by characteristics of the different signs and symptoms:

- **Stage 1.** Patients are typically asymptomatic in stage 1. The retina typically looks slightly grayish and

<table>
<thead>
<tr>
<th>Stages of Type 2 IJRT</th>
<th>Signs</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Retina appears grayish; telangiectasis visible by IVA only</td>
<td>Typically asymptomatic</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Graying of parafoveal region on temporal aspect of macula; thinning of foveal region seen</td>
<td>Typically asymptomatic</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Right-angle venules mostly temporal to fovea; blunted foveal reflex</td>
<td>Mild vision loss (usually when patient presents to clinic)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Clumps of RPE hyperplasia seen around right angle venules; pseudo-vitelliform lesion sometimes seen</td>
<td>Vision loss worsens</td>
</tr>
<tr>
<td>Stage 5</td>
<td>Development of a neovascular membrane; superficial crystalline deposits may develop</td>
<td>Most detrimental vision loss</td>
</tr>
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About Rick
Rick Bay served as the publisher of The Review Group since 1991.

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

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there could be a loss of retinal transparency.3 Stage 1 signs may be difficult to identify by biomicroscopy alone. Fluorescein angiography will show juxtafoveal telangiectasis in stage 1 even if there are no abnormalities seen during clinical examination.10

Stage 2. In stage 2, evidence of the disease starts to be slightly more obvious with biomicroscopy. There is graying of the parafoveal region that is usually confined to the temporal side or forming a horizontal oval around the fovea while sparing the foveal center. Although the fovea is spared, it may look slightly thinner than the surrounding retina. Minimal telangiectatic changes usually occur at this stage, giving it a subtler appearance than type 1 IJRT.3

Stage 3. Stage 3 exhibits right-angle venules, mostly found temporal to the fovea, but they can also surround the fovea with a blunting of the foveal reflex.6 Most patients present to clinic during stage 3 due to the complaint of mild vision loss. This vision loss appears to be due to progressive foveal atrophy.10

The foveal depression may mimic a macular hole in this stage when viewed by biomicroscopy, but is easily distinguishable by fluorescein angiography.3

Stage 4. Stage 4 is characterized by clumps of retinal pigmented epithelial hyperplasia seen around the right-angled vessels in the parafoveal region that are associated with chorioretinal anastomosis.3,10 A stellate pattern of retinal pigmented epithelial hyperplasia may envelop the macula during this stage, and some patients may develop a pseudovitelliform lesion.3,6

Stage 5. Stage 5 is the final stage of type 2 IJRT. This is the stage that is most detrimental to vision due to the development of an intraretinal neovascular membrane (NVM). The NVM may occur as a result of retinal capillary remodeling, proliferation and invasion of the outer retina that has progressively atrophied from the disease.3 In 40% to 45% of eyes in stage 5, superficial retinal crystalline deposits of unknown etiology have been reported. The significance of these refractile deposits is currently unknown, and they may occur at any time between stages 2 and 5.10

Refractile crystals may be present in other retinal disorders, but these crystals have distinguishing characteristics, such as showing specular reflection and being distributed in an annular pattern around the fovea, sparing it centrally.12

*Type 3.* The final group is type 3, which is bilateral perifoveal telangiectasis with capillary obliteration. This type is extremely rare and usually occurs in a patient’s 40s with no gender predilection. Aneurysmal dilation occurs, along with the capillary obliteration without leakage. This causes slow, progressive vision loss in patients. This type is associated with other sequelae such as optic nerve head pallor, hyperactive deep tendon reflexes and central nervous system (CNS) symptoms.6 Type 3 is suspected to be autosomal dominant in inheritance pattern and is known to be associated with cerebroretinal vasculopathy.4 Researchers have shown evidence of CNS pseudotumor characterized by unusual vasculopathy with fibrinoid necrosis and necrosis of white matter.5 If you suspect CNS symptoms, refer patients to a neurologist.6
Overall, the most common symptoms reported by patients are blurred vision, metamorphopsia and possible scotomas when checked with Amsler grid. This contrasts with the many asymptomatic patients with type 1 or the early stages of type 2. (See Classification of IJRT by Type,” page 84.)

**Pathogenesis**

The pathogenesis and etiology of IJRT are unknown at this time. One study by Sallo et al. suggested looking into the physical and chemical properties of the retinal crystals to find information on the metabolic pathways involved in the pathogenesis of the disease.12

Other researchers have suggested that the histopathology of IJRT may be linked to dysfunction of the Muller cells. The crystalline deposits have been thought to be remnants of degenerated Muller cells due to the location near the ILM.

**Management**

Although a number of studies have attempted to determine the best treatment of IJRT, no treatment modality has been able to show consistent efficacy.7,13,14 Some of these studies are contradictory, but the current treatments include intravitreal bevacizumab injections, focal grid laser photocoagulation or photodynamic therapy (PDT).7,13,14

If patients are asymptomatic or have minimal vision loss, usually no treatment is recommended.6

If a pronounced macular edema is seen in type I, patients may benefit from intravitreal bevacizumab injections.13 One study of type 1 patients used laser photocoagulation to prevent the accumulation of lipids in the fovea, especially if no prior accumulation of exudates existed.11

The treatment of type 2 IJRT is more controversial. A study concluded that if minimal cystic changes are seen on OCT, repeated injections will not produce functional improvement.13 Another study found intravitreal bevacizumab provided short-term improvement in vision due to decrease in retinal thickness and a reduction of angiographic leakage.14 The use of focal laser photocoagulation is performed only if neovascularization is present outside the foveal area.6

Again, this treatment is controversial because some studies have shown no significant improvement of visual outcome with focal/grid photocoagulation. Reports show an increased risk of the development of subsequent neovascularization after treatment.

Recently, PDT has been used to resolve leakage of a neovascular membrane, but there is no effect on edema associated with telangiectasia.10 Currently there are no treatments available for the ocular manifestations of type three IJRT.

Unfortunately, the rarity of this disease makes the treatment difficult to assess in a controlled but random manner. Gass and Blodi did not recommend laser photocoagulation in type 2 if it is non-proliferative due the long-term prognosis already being poor. If type 2 is proliferative, anti-VEGF treatment such as bevacizumab may be efficacious.1

Comanage the treatment of these patients with a retinal specialist, as a fluorescein angiography is necessary to confirm the diagnosis and staging of IJRT.

IJRT is not a common disease entity. It is often misdiagnosed as retinopathy secondary to other diseases such as diabetes or sequelae of an old venous occlusion. Fluorescein angiography and ocular coherence tomography are tools that aid in the definitive diagnosis of this retinal disease. The different groupings and stages assist the clinician in determining an accurate prognosis and the best course of treatment.

**Dr. Cooper is a fee-basis optometrist at the C.W. Bill Young VA Medical Center in Bay Pines, Florida.**

**Dr. Gruosso is a staff optometrist also at the C.W. Bill Young VA Medical Center, where he is the director of the low vision clinic and student externship program.**

**Dr. Miller is a staff optometrist and residency program coordinator at the C.W. Bill Young VA Medical Center.**

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Coding Point-of-Care Testing

Take care when coding for point-of-care tests, and be aware that carrier rules are ever-changing. By John Rumpakis, OD, MBA, Clinical Coding Editor

Point-of-care testing is becoming more popular in the average optometric practice as technology improves and weaves its way into our daily clinical care. Many of the testing devices are small and simple to use—but that doesn’t mean that coding for point-of-care testing can be taken lightly, nor is it something you should assume is “bundled” into an office visit. In fact, many point of care tests require that your office have a Clinical Lab Improvement Amendments (CLIA) waiver. (See “Get CLIA Certified for POC Testing,” page 56.)

Lab tests are paid from a national laboratory fee schedule and don’t follow the RBRVS reimbursement model. CPT codes are designated in the 8XXXX range. Only those tests that have the “waived” designation can be performed in your office. For coding purposes, CLIA-waived tests are designated by the -QW modifier.

Here are some common point-of-care tests that ODs perform:

**TearLab (TearLab).** CPT code 83861-QW: Microfluidic analysis using an integrated collection and analysis device, tear osmolarity. If you’re testing both eyes and coding for it, this is what the claim form would look like:
- 87809-QW-RT
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**InflammaDry (Rapid Pathogen Screening).** CPT Code 83516-QW: Immunoassay for analyte other than infectious agent antibody or infectious agent antigen, qualitative or semiquantitative, multiple step method. If you’re testing both eyes and coding for it, this is what the claim form would look like:
- 83516-QW-RT
- 83516-QW-LT

Not All Tests Are Created Equal

However, not all point-of-care tests are clinical lab tests or require CLIA certification. For example, both point-of-care tests for macular degeneration—Macula Risk NXG (ArcticDx) and RetnaGene AMD (Sequenom)—are not billable tests because the test itself is performed by an outside lab and the OD only collects the clinical sample by method of a buccal (cheek) swab. Swabbing is not a distinct and separate procedure according to the CPT and therefore is part of the office visit.

Tests for Sjögren’s disease (Sjö) and diabetes both require collection of blood, which may not be within the scope of practice in your state. Keep in mind that the patient may stick his or her own finger for the blood sample, but you may not be able to. Again, this is a sample collection only, but in this case is described with CPT code 36415 (routine venipuncture) and pays about $3. Be cautious, as one could argue that you really didn’t perform the procedure if the patient stuck himself or herself.

Point-of-care testing is certainly on the rise in optometry. Be sure that you follow the rules of medical necessity when ordering any tests. Follow the guidelines by properly obtaining your CLIA certification. Finally, be aware that carrier rules are ever-changing and may not support the use of testing as you see fit.

Send questions and comments to ROcodingconnection@gmail.com.

Tear Testing May No Longer Be Covered?

Be aware of a recent development in dry eye testing (TearLab and InflammaDry): Some CMS carriers are proposing that neither test be covered due to inconclusive medical evidence that they affect the outcome of treatment. Here is an excerpt from one CMS carrier (Novitas):

“Due to the lack of supporting data to demonstrate patient benefit from microfluidic analysis (tear osmolarity) or immunoassay analysis of tears relative to treatment choice and planning, disease outcome or evolution, Novitas considers tear testing for osmolarity and immunoassay analysis for other than infectious agent, antigen or antibody not medically reasonable and necessary. Subsequently, tear testing (83516, 83861) for the evaluation of dry eye or its associated disease entities is non-covered.”

By John Rumpakis, OD, MBA, Clinical Coding Editor

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Checks and Balances

Reestablishing a pH balance is key to controlling ocular damage from chemical burns.

Edited by Joseph P. Shovlin, OD

Q I recently saw a patient referred from the local emergency department with a severe alkali burn (fertilizer) to the conjunctiva and cornea. His eye was irrigated copiously for 40 minutes for the chemical insult prior to presenting to our office. How valuable is pH testing in cases like this one, i.e., those that have been copiously irrigated? Is it considered passé?

A Litmus paper can be helpful in certain instances; however, it does not take precedence over immediate and aggressive irrigation following an ocular chemical injury.

“Alkalis penetrate the conjunctiva and cornea quite readily, while acids precipitate on the surface, which tends to limit their penetration and injury severity. Consequently, alkali injuries are generally more damaging than acid injuries,” says Christopher J. Rapuano, MD, director of cornea service at Wills Eye Hospital in Philadelphia. Examples of alkali offenders include ammonia (fertilizers or refrigerants), lye (cleaning agents) or lime (building materials). Damage can vary depending on the type of chemical, how much entered the eye and when initial irrigation began.

The primary goal of irrigation following a chemical injury is to reestablish a pH level of approximately 7.5, the pH level of healthy human tears, Dr. Rapuano explains. “If the initial pH is much higher or lower than that, it will usually take more fluid for longer periods of time than if the initial pH is close to 7.5,” he says. Consequently, he adds, there is no exact quantity of fluid or duration of effort established as a minimum to achieve adequate irrigation.

It is in this situation that pH testing is useful: first, to check initial pH before irrigation (if possible) or upon arrival after being irrigated elsewhere, and then once or twice at 20-minute intervals to monitor irrigation efforts. Note, however, that the effectiveness of irrigation by evaluating with litmus paper has never been scientifically studied.

Ocular irrigation can be performed by dripping either water or saline from an IV bag or nasal cannula, or using a Morgan lens, Dr. Rapuano says. He suggests considering the possibility that foreign material—for example, wet concrete, which is alkaline in nature—might adhere to the eye if the pH remains abnormal after copious irrigation. Check the lid area and fornix with double eversion, then carefully remove any particulate material and excise devitalized tissue.

J. James Thimons, OD, ophthalmic medical director at Ophthalmic Consultants of Connecticut, points out that pH testing can also help in certain unique cases. “In patients exposed to chemical agents, pH testing is a useful adjunctive tool for the clinician in cases where either the agent is unknown or the actual pH is outside of the routine chemicals that are seen in primary care practice,” he says.

However, Dr. Thimons cautions, the somewhat limited shelf-life of test strips can confound pH testing. “Due to infrequent use, test strips can become ineffective and demonstrate inaccurate pH levels,” he says. “The remedy to this concern is to periodically test the strips on normal patients to ensure accuracy.” Error can also result from the presence of too little or too much tear film, which can alter pigment levels in the strip response, and from using the strips too soon after irrigation.

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* Conjunctival hyperemia, limbal hyperemia, corneal staining, papillary conjunctivitis, corneal vascularization. The average incidence of conjunctival staining was not clinically equivalent to the naked eye, but results were consistent with another etafilcon A neophyte study and the level does not require clinical management.

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ACU-A44407-B February 2015
THE OPTOMETRIC RETINA SOCIETY AND REVIEW OF OPTOMETRY PRESENT:

RETINA UPDATE 2015

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A 38-year-old Hispanic female from Honduras presented with a painless, sudden onset of blurred vision in the left eye of unknown duration. She stated that her central vision was most affected. She denied any pain, discomfort, flashes, floaters, nausea, fever or weight loss. She reported her right eye was unaffected. Her past ocular history was unremarkable and she reported good health. She neither takes any medications nor has a history of any prior ocular trauma or surgeries. She was not nursing or pregnant.

Upon examination, her best-corrected visual acuity was 20/20 in her right eye and hand motion vision in her left eye. The confrontation fields were full to careful finger counting in her right eye and constricted in her left eye. Ocular motilities were normal and her pupils were equal, round and responsive to light with no afferent pupillary defect. The anterior segment in the left eye was positive for trace cell, but otherwise normal in both eyes.

On dilated fundus exam, the right eye was unremarkable. The vitreous of the left eye was positive for 1+ vitreous cells with vitreal haze. Other changes are seen in *figure 1*. Lattice degeneration was present inferiorly in both eyes. An SD-OCT of the macula in the left eye was obtained and is available for review (*figure 2*). An autofluorescence image (*figure 3*) was also obtained.

### Take the Retina Quiz

1. What do the fundus changes in the right eye macula show?
   a. Papilledema.
   b. “Headlights in the fog.”
   c. “Snowballing.”
   d. Macular star.

2. What additional testing would be most helpful in making the correct diagnosis?
   a. Blood test for IgG and IgM.
   b. Blood test for FTA-ABS, RPR.
   c. PPD.
   d. HLA-B27, HLA-A29.

3. What is the treatment for this patient?
   a. Bactrim PO, clindamycin PO, steroids PO, folic acid.
   b. Azithromycin PO.
   c. Doxycycline PO.
   d. Steroids IV.

4. What is most likely the diagnosis?
   a. Histoplasmosis.
   b. Tuberculosis.
   c. Toxoplasmosis.
   d. Toxocariasis.

*For answers, turn to page 122.*

### Diagnosis

Our patient has a white, chorioretinal, inflammatory lesion with an overlying vitritis adjacent to a dark, chorioretinal scar. The lesion was directly overlying the macula. This presentation is more commonly known as “headlights in the fog,” which represents an active vitritis overlying a white foci of retinochoroiditis. Adjacent to the active lesions is an old chorioretinal scar from a previous active lesion. This presentation most likely represents ocular toxoplasmosis. The patient did not admit to having any cats nor eating any raw or undercooked pork, lamb or venison.

Toxoplasmosis, the most common cause of posterior uveitis, accounts for approximately 90% of focal necrotizing retinitis. *Toxoplasma gondii* is a small, oblong shaped, intracellular protozoan parasite known to cause toxoplasmosis. The rate of seropositivity for toxoplasmosis is estimated around 22.5% in the United States. More than 60 million in the United States may have been affected by the parasite but, of those, few display symptoms. This is because a healthy immune system usually keeps the parasite from causing illness.

The diagnosis is usually made based on the clinical presentation. Blood work can be performed, but a positive result does not always...
Retina Quiz

ensure a diagnosis. Interestingly, there is a high seropositivity in the general population, which means that a positive titer does not necessarily confirm the diagnosis, but a negative titer should prompt strong deliberation of alternate diagnoses. IgG antibodies are produced within two weeks of infection and peak at two months, then later present for life.\(^3\) Detection of IgM antibody titers are usually possible within the first two weeks of infection and suggest a recently acquired infection.\(^2\) Blood work-up was not performed on our patient because it was a pretty “classic” presentation.

The disease can be transmitted via several potential methods. Ingesting undercooked meats such as pork, lamb or venison could be a culprit. Another involves consuming water contaminated with the parasite or accidental exposure through contact with cat feces. The most common transmission is mother-to-child (transplacentally).

Mothers who are seropositive for toxoplasmosis show rates of transmission that range between 60% and 81%, which can be more noticeable in the third trimester.\(^4\) Manifestations of congenital toxoplasmosis include: hydrocephalus, seizures, intracranial calcifications and retinochoroiditis.

Clinical symptoms of active toxoplasmosis included blurred vision, floaters and potential redness or photophobia or both. Pain is typically absent, except with iridocyclitis. The clinical appearance of a toxoplasmosis lesion may vary depending on whether the infection is active or inactive. The active state usually appears as a unilateral, whitish, fluffy, retinal lesion with blurred margins. When the disease reoccurs, it often does so next to a “satellite lesion” also known as an old choriretinal scar.

Toxoplasmosis is generally self-limiting in immunocompetent patients, and treatment is reserved for lesions threatening or involving the macular or optic nerve. We elected to treat our patient in two ways. Since the lesion was active and directly involving the macula...
and her vision was drastically affected, the patient was given an intravitreal injection of dexamethasone 0.4mg/clindamycin 1mg in office. Studies show that intravitreal injection of clindamycin and dexamethasone is as effective as conventional oral therapy in treatment of ocular toxoplasmosis with fewer systemic side effects. A study at the Nikookari Eye Hospital in Iran tested oral therapy vs. intravitreal for ocular toxoplasmosis, and within six months the results were equally effective in terms of increased BCVA, a decrease in lesion size and a termination of the vitritis.

After the injection, the patient was placed on clindamycin 30mg PO TID, Bactrim DS PO BID and prednisolone 40mg PO QD. She will continue this oral dosage for a month. At her two-week follow-up, the vitritis resolved and the acuity improved to 20/800 OS. The visual prognosis is guarded secondary to macular involvement. Spiramycin is reserved for fetal prophylaxis or to decrease disease severity in cases of maternal infection without fetal infection. It is prescribed as 1g every eight hours for the duration of pregnancy once diagnosed.

In cases of confirmed infection of the mother and the fetus (through cranial ultrasound or amniocentesis), the mother is given oral Bactrim PO until delivery.

This case was written and provided by Nikolaos C. Zagorianos, OD, an optometric resident at Bascom Palmer Eye Institute.

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11:00 AM–11:15 AM  TOTAL 3D SOLUTIONS: THE COMING AGE OF VR  Annette Tindall, Principal
11:30 AM–11:45 AM  VISIONCARE OPTHTALMIC TECHNOLOGIES, INC.: THE IMPLANTABLE MINIATURE TELESCOPE FOR INDIVIDUALS WITH END-STAGE AMD  Karen Murphy, MS  OTR/L, SCLV
12:00 PM–12:15 PM  3-D FRAME SOLUTIONS: PRINTING YOUR PRACTICE PROFITS  Dr. Elvin Fenton
12:30 PM–12:45 PM  PIVOTHEAD: PIVOTHEAD SMART  Jordan Elder, Sales Account Executive
1:00 PM–1:15 PM  AVI WEAR: HOW SMART GLASSES WILL BE CHANGING THE WAY WE LIVE  Dan Shannon, Vice President and Co-Founder
1:30 PM–1:45 PM  VUZIX: SMART GLASSES - GEEK OR SHEIK?  Dan Cui, Vice President, Sales and Business Development
2:00 PM–2:15 PM  ROCHESTER OPTICAL: GETTING OVER FUTURE SHOCK  Timothy Moore, CMO
2:30 PM–2:45 PM  MINT: INCREASING ECP PROFITABILITY THROUGH INDUSTRY  Jack Gill, MBA
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HIV is a blood-borne retrovirus. Acquired immune-deficiency syndrome (AIDS) is caused by HIV and is characterized by profound immune suppression that leads to opportunistic infections (OIs), secondary neoplasms, neurological manifestations and possible death.

In the United States, HIV was first described in 1981 among two groups, one in San Francisco and the other in New York. Numerous young homosexual men presented with opportunistic infections that were typically associated with severe immune deficiency: Pneumocystis pneumonia (PCP) and aggressive Kaposi sarcoma.1 HIV itself was not identified for another two years.2

People at Risk
Men who have sex with men continue to bear the greatest burden of infection. Among races/ethnicities, African Americans and Hispanic/Latinos are disproportionately affected, as are injection drug users.3,4 People with sexually-transmitted diseases (STDs) often also have, or are more likely to get, HIV.4,5

Transmission and Transcription
HIV is typically transmitted via sexual intercourse, shared intravenous drug paraphernalia and mother-to-child transmission, which can occur during birth or breastfeeding. HIV disease is caused by infection with HIV-1 or HIV-2, which are retroviruses in the Retroviridae family, Lentivirus genus.

Retroviruses use RNA as their genetic material, but the host cell must synthesize a “DNA copy” of the RNA before it can be transcribed or translated. This task is aided by the action of an enzyme known as reverse transcriptase.6

Like others in the lentiviral genus, HIV creates a long interval between initial infection and serious symptoms. It targets the immune system by primarily affecting an arm of adaptive immunity called “cell-mediated immunity.”

The cluster of differentiation 4 (CD4) molecule, found on T helper (Th) and other immune system cells, is a high-affinity receptor for HIV. Binding to CD4 is not sufficient for infection, so HIV must also bind to other surface molecules for entry. Mature T cells that express CD4 (CD4+T cells), along with macrophages and follicular dendritic cells contained in lymphoid tissues, are the major sites for HIV infection and persistence.7

Clinical Signs
Viral replication occurs in the regional lymph nodes and leads to viremia with eventual seeding of lymphoid tissue. Acute retroviral syndrome, the initial response to HIV infection, occurs approximately four weeks after infection. Clinically, this phase is a self-limited acute illness with flu-like symptoms such as rash, sore throat, vomiting, myalgias, fever, weight loss and fatigue.

The following chronic stage may last for several years. The patient may be asymptomatic, but generalized lymphadenopathy, thrush, herpes zoster and thrombocytopenia may be noted during this time. Activation of CD8+ cytotoxic T cells appears to be the primary mechanism for immunologic control of HIV. T cell responses are correlated with the steady-state viral load and thus the rate of progression.5,7

The Eye and HIV
These are the most frequent ocular complications of HIV/AIDS:7,8
- Irritation of the conjunctiva
- Keratoconjunctivitis sicca
- HIV-related retinal microangiopathy
- Cytomegalovirus
- Immune recovery uveitis
- Acute retinal necrosis
- Progressive outer retinal necrosis
- Molluscum contagiosum
- Syphilis
- Toxoplasmosis
- Pneumocystis jiroveci
- Mycobacterium tuberculosis
- Neoplasm (Kaposi sarcoma)
Review of Systems

The final phase is progression to AIDS. In the United States, a CD4+ T cell count less than 200/µL is used as a measure to diagnose AIDS. Long-lasting fever, fatigue, weight loss and diarrhea may occur, as well as OIs, secondary neoplasms and neurological disease. These account for the majority of deaths in patients with AIDS.²,⁷

A high-sensitivity enzyme-linked immunoabsorbent assay (ELISA) should be used for screening; a positive result should be followed with confirmatory testing. The CD4+ T cell count reliably reflects the current risk of acquiring OIs. Viral load in peripheral blood is used as a surrogate marker of viral replication rate. Patients with viral loads greater than 30,000/µL are 18.5 times more likely to die of AIDS than those with undetectable viral loads.⁷

Antiretroviral Treatment

The use of combination antiretroviral therapies and prophylaxis for OIs dramatically improves survival.⁷,⁸ The US Food and Drug Administration has approved a once-daily, fixed-dose triple-combination pill (Triumeq, ViVi Healthcare) containing the antivirals dolutegravir, abacavir and lamivudine for the treatment of patients aged 18 years or older. Overall, with the increasing use of antiretroviral therapy and the introduction of better regimens, survival with HIV has increased.

Society has made substantial progress, but we continue to face challenges. Patients with HIV should be counseled on the risks of infecting sexual partners. Safe sex practices and treatment of STDs, in the patient and sexual partners, considerably reduces the risk of transmission. Intravenous drug users should be counseled on the risks of sharing paraphernalia.

Ocular Complications

Approximately 80% of HIV-infected patients will be treated for an HIV-associated eye disorder.⁹ The optometrist may be presented with adnexal, anterior segment, posterior segment or orbital manifestations. Adnexal manifestations may include herpes zoster or conjunctival microvasculopathy, among others. Anterior segment findings include tumors of the periocular tissues and a variety of external infections. Posterior segment changes include an HIV-associated retinopathy/vasculopathy and a number of OIs of the retina and choroid. Orbital manifestations of HIV are rare; however, the most common complications include orbital lymphoma and orbital cellulitis due to Aspergillus infection.⁹

Increasing longevity of individuals with HIV may result in greater numbers of patients with ocular complications. Fortunately, many of these are now treatable. Partial immune system recovery following initiation of effective antiretroviral therapy may modify clinical presentation of OI and can affect response to treatment. In addition, several infections may occur in a single eye at the same time, rendering diagnosis and therapeutic intervention more difficult.

Due to the potentially devastating and rapid course of OI, patients with HIV should undergo regular evaluations. With early-stage HIV, ocular syndromes associated with immunosuppression are uncommon. Nonetheless, ocular infections associated with STDs may be more frequent in patients with HIV; therefore, clinicians should screen for HIV in the presence of these infections.

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The Throwback Thursday Option

Recognize when a situation calls for doctors to revert to old-model drugs.

By Joseph W. Sowka, OD and Alan G. Kabat, OD

Today, optometrists can access an impressive drug armamentarium to treat our patients. The anti-infectives, steroids, antihistamines and glaucoma medications currently available are second to none and represent a huge step forward from the options of only a few years ago. With the potency and efficacy of new therapies, often there comes a high price. Clinically, these new medications are outstanding, but when patients cannot afford them, how should an optometrist respond? Are clinicians justified in reaching back to an older, tried-and-true standby?

This month’s column is a “Throwback Thursday” edition (with author photos to match), wherein we’ll explore older medicines that still have a place today.

Infectious Bacterial Keratitis

The first case we’ll look at involves a 45-year-old woman recently released from eight months of incarceration. A high myope, she never disclosed to prison officials that she wore soft contact lenses, and used a single pair of lenses incessantly for the duration. She presented with a painful, red, right eye. Her findings included profound conjunctival injection, a significant secondary anterior chamber reaction, dense paracentral corneal infiltration—which fortunately did not immediately threaten her visual axis—and epithelial excavation. In short, she had an infectious keratitis, presumably of bacterial nature.

After explaining the diagnosis and risk of permanent vision loss, we discussed therapeutic options and the need for potent antibiotics. We felt that a fourth generation fluoroquinolone such as moxifloxacin, gatifloxacin or besifloxacin would be best. However, after discussing the cost of these branded medications, the patient emphatically stated that she would not be able to afford them and could not possibly use them. After a discussion about the need for aggressive treatment, a compromise was reached. We prescribed generic Ocuflox (ofloxacin 0.3%) which cost her about $9.00.

Though Ocuflox is an older generation fluoroquinolone, it eradicated the infection successfully.

Ocuflox and Ciloxan

Ocuflox and Ciloxan (ciprofloxacin 0.3%) are second-generation fluoroquinolone antibiotics FDA approved for the topical treatment of bacterial conjunctivitis and bacterial keratitis caused by susceptible organisms. Ofloxacin and ciprofloxacin are bactericidal at concentrations equal to or slightly greater than inhibitory concentrations. Ofloxacin and ciprofloxacin exert bactericidal effects on susceptible bacterial cells by inhibiting DNA gyrase (which is a type II topoisomerase) or topoisomerase IV, which is an enzyme necessary to separate replicated DNA, thereby inhibiting bacterial cell division, or both.

Topical Fluoroquinolones

The development in the 1990s of topical fluoroquinolones offered a highly effective alternative to fortified antibiotics for patients with bacterial keratitis. For the first time, commercially available antibiotics were scientifically compared with the standard treatment of fortified antibiotics. Historically, bacterial keratitis was treated with compounded, fortified solutions of aminoglycosides and cefazolin. Often, this involved diluting antibiotics originally designed for intravenous use. The results of the Bacterial Keratitis Study Group, as well as
Therapeutic REVIEW OF OPTOMETRY

more recent studies, showed that ofloxacin and ciprofloxacin were equivalent to fortified cefazolin and tobramycin solutions in the management of patients with bacterial keratitis.1-5 The reduced frequency of ocular toxic effects and the relative ease of use of these fluoroquinolones was seen as additionally beneficial in these studies.

Efficacy

However, bacteria have grown increasingly resistant to second generation fluoroquinolones, and some researchers suggest that later generation fluoroquinolones, such as gatifloxacin 0.3%, provide superior coverage.6 Inspection of at least one medication pricing website, www.goodrx.com, indicates that the cost of gatifloxacin 2.5ml is $131.28 for the branded form and $50.72 for the generic version. In comparison, 5ml of generic ofloxacin is $9.37 and generic ciprofloxacin is $11.74. Unquestionably, the fourth generation fluoroquinolones are outstanding in terms of antibiotic efficacy and superior choices for sight-threatening infections. But the most effective medication will not work if the patient can’t afford it. Don’t discount older generation fluoroquinolones. They remain effective and accessible to most patients.

Non-infectious Keratopathy

In another case, a 30-year-old female contact lens wearer presented with a red, moderately irritated right eye. She had diffuse injection of her bulbar conjunctiva and a non-staining peripheral corneal infiltrate. Additionally, she had a mild diffuse epitheliopathy. We diagnosed a contact lens-related hypoxia and non-infectious keratopathy. We told her to temporarily discontinue contact lens wear. We wanted to reduce inflammation while providing some degree of antibiotic coverage and recommended a combination antibiotic-steroid medication.

When she reported that she had no insurance, we again consulted www.goodrx.com to get an idea of her anticipated costs. We looked at two branded steroid-antibiotic combination agents and found that a 5ml bottle would cost her from $158.88 to $197.97. Her facial expression gave us her answer. We prescribed an alternative, generic Maxitrol solution, which she purchased for $4.00. Within a week, her condition resolved.

Maxitrol

Maxitrol is an antibiotic-steroid combination of neomycin sulfate 3.5mg, polymyxin B sulfate 10,000 units, and dexamethasone 0.1%. As with most antibiotic-steroid combinations, it is appropriate for steroid-responsive inflammatory ocular conditions for which a corticosteroid is indicated and where bacterial ocular infection or a risk of infection exists. Polymyxin B is an antibiotic used for resistant gram-negative infections, altering bacterial outer membrane permeability by binding to a negatively charged site in the lipopolysaccharide layer, which results in a destabilized bacterial outer membrane.

Neomycin is an aminoglycoside antibiotic that, like others in the category, has excellent activity against gram-negative bacteria, and has partial activity against gram-positive bacteria. It is relatively toxic. Much has been said about the potential allergic responses that patients have to neomycin, making it a lesser choice for clinicians. However, the majority of allergic reactions that we have seen have involved over-the-counter dermatologic preparations where patients used it off-label for the eyes. We cannot recall any instance where Maxitrol caused a toxic ocular reaction, quite possibly due to the steroid combating any allergic responses. Lately, we have seen more cataract surgeons providing Maxitrol in postoperative kits, likely due its efficacy and price. While branded antibiotic-steroid combinations are excellent choices, we never hesitate to prescribe Maxitrol.

Ocular Hypertension

The final patient is a 58-year-old male with ocular hypertension, also with no prescription drug coverage. His untreated IOP was 25mm Hg OD and 26mm Hg OS and his central cornea was thin at 456µm OD and 464µm OS. He mentioned his mother lost vision due to glaucoma. After a detailed discussion about the risks and benefits of prophylactic IOP reduction, we prescribed generic latanoprost, which gave him an unacceptable degree of hyperemia.

We discontinued the latanoprost and instead prescribed generic timolol 0.5%, which was tolerable and affordable at $4.00 a bottle. Using timolol, this patient achieved adequate IOP reduction.

Timolol

Since the advent of the prostaglandin medications, clinicians’ reliance on topical beta blocker therapy has significantly reduced. However, prior to the development of prostaglandins, doctors used beta blockers extensively with few problems. Like prostaglandin analogs, the earlier introduction of branded Timoptic (timolol maleate 0.5%) revolutionized glaucoma management.

While specific contraindications exist, it appears that many of the propagated fears about topical beta blockers stem from anecdotal case reports rather than evidence-based
sources. These drugs are actually quite safe for the vast majority of patients, and virtually no confirmation shows they directly induce clinical depression, sexual dysfunction, claudication, prolonged hypoglycemia or hypoglycemic unawareness. However, practitioners must remain aware of potential complications and be alert for any unusual systemic complaints in patients using beta blocker therapy. Always obtain a thorough health history, check in-office pulse rate, and refer patients for medical evaluation when indicated prior to initiating topical beta blockers. With these minimal precautions appropriately taken, timolol is an excellent first-line medication, which gives impressive IOP reduction at a low cost.

The field has developed a number of ophthalmic medications in the last several years with unprecedented efficacy. However, it is important to remember medications we used successfully in the past. Just because something is old doesn’t mean it isn’t good.

The authors would like to thank Dr. Amanda Brown for suggesting this column.

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Cataract surgery is a once in a lifetime opportunity to provide our patients with greatly improved vision. As the comanaging OD, any information we can gather prior to the surgical referral will help determine the procedure and outcome. For instance, if a patient has corneal astigmatism and a toric IOL would be appropriate, send the corneal topographies along with the referral notes, so the surgeon can look for consistency in the readings.

This month, let’s review how pre-op testing can aid surgical planning.

**Biometry**

Precise biometry is essential in cataract surgery. Simply put, outcomes depend on it. Measurement of axial length by ultrasound has been the gold standard for many years. The introduction of optical biometry has increased accuracy and added the ability to measure anterior chamber depth, lens thickness, pachymetry, pupillometry, keratometry and white-to-white distance.

Popular optical biometers include the Lenstar LS900 (Haag-Streit) and the IOLMaster 500 (Zeiss). Both measure corneal curvature; the IOLMaster makes six measurements at 3mm of the central cornea and the Lenstar performs 16 central corneal readings—eight at 1.7mm and eight at 2.3 mm. All the standard IOL prediction formulas (Holladay I and II, SRK-T, Haigis and Hoffer Q) are built into the software.

If optical biometry cannot be performed, use applanation biometry. Compared to traditional biometry, A-scan biometry by immersion has better reproducibility, which leads to an overall increase in accuracy.

**Corneal Measurements**

Accurate corneal measurements are critical in IOL calculations and surgical planning, and they also determine which patients would benefit from limbal relaxing incisions or toric IOLs. Several technologies can provide this important data, and many practices capture corneal measurements on a number of devices to ensure consistency and accuracy of the measurements. Options include:

- **Keratometry.** A mainstay of optometry, this reflection-based test measures four points over a central zone of 2.88mm to 4mm, two at the steep axis and the other two 90 degrees away.

- **Placido disk topography** reflects a series of concentric rings, or mires, off the cornea. Placido disk systems have certain advantages over the keratometer in that they are able to measure more points over the corneal surface and over an infinite corneal power range.

- **Scanning slit topography** such as the Orbscan (Bausch + Lomb) is a projection-based method that uses a series of slit-beam images to generate data regarding anterior surface curvature, posterior surface curvature and pachymetry.

- **Scheimpflug-based systems.** The Pentacam (Oculus) is a combination device consisting of a slit illumination system and a Scheimpflug camera that rotates around the eye. Rotating around the central corneal axis, the slit-camera device generates a series of radially oriented images of the anterior eye chamber. Results can be used to generate data on elevation, curvature, pachymetry and depth of the anterior eye chamber.

- **Color LED Topography.** The Cassini (i-optics) technology uses ray-tracing principles to measure the relative position of each point, using the three different colors as ‘triangulation’ points to determine elevation, depression and curvature.

If we are unable to take accurate pre-op measurements—e.g., in patients who could not easily access the diagnostic technology due to physical limitation—many surgeons rely on average corneal readings and approximations of axial length.

**Additional Diagnostic Tests**

- **Endothelial cell count.** Assessing endothelial cell counts and density is useful in spotting patients at higher risk of corneal decompensation.

- **Pachymetry** determines corneal thickness and helps identify patients at risk of corneal decompensation; it also aids in glaucoma management.

- **B-scan ultrasound** is indicated for mature cataracts and to detect any posterior segment pathology prior to surgery.

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**Product Review**

### Diagnostic Equipment

**New Portable Autorefractor**

If space in your exam lane is hard to come by or you do a lot of clinical exams at various locations in and out of the office, the new SVOne autorefractor from Smart Vision Labs might help. The handheld device, which measures refractive error using a wavefront sensor, connects to an iPhone and uploads information to a HIPAA-compliant cloud. Its open-field design reduces instrument-induced myopia, the company says. The device also allows for instantaneous smartphone-based digital prescriptions. The portability and long-lasting battery—which is said to last through 36 hours of continuous refracting—helps optometrists work in limited space and non-clinical conditions.


### Ophthalmic Lenses

**Next Generation of Unbreakable Lenses**

Athletic and other outdoorsy patients will appreciate hearing about new photochromic lenses called ImpactX-2 that provide faster color transition and enhanced contrast in an unbreakable lens, according to manufacturer Rudy Project.

ImpactX-2 lenses have the ability to automatically lighten and darken from a semitransparent tone to a specific color according to lighting conditions. ImpactX-2 comes in five variations that transition from clear to either black, laser black, red, laser red or laser brown.

These lenses offer a wider photochromic range of up to 65% light transmission difference, Rudy Project says. This photochromic activation occurs in all natural light within seconds, including behind surfaces that screen UV rays such as windows or car windshields.

For patients with refractive error, ImpactX-2 can be crafted with their specific correction parameters; backside surfacing applies the prescription to the back of each lens.

Visit [www.e-rudy.com](http://www.e-rudy.com).

**“Change Your View” Sweepstakes**

Want to win a sightseeing trip to Alaska, or other fun prizes like a GoPro camera or a Microsoft Surface tablet? You can enter the “Change Your View” sweepstakes from Essilor of America, running through April 30, 2015, to register. Doctors who complete a training session on the use of Transitions photochromic lenses are eligible for the contest.

Prizes are awarded weekly and monthly. The grand prize winner will receive round-trip airfare for two to Fairbanks and Anchorage, accommodations in both cities, an expedition to the Spencer Glacier and a rafting adventure in Anchorage. The winner will also receive a digital camera, photography classes and a voucher redeemable for a pair of Transitions lenses and frames.

Monthly prizes include an HD camera, a 32GB tablet and an airlines gift card. Weekly prizes include Amazon, Teavana and iTunes gift cards.

Visit [www.essilortransitions.com](http://www.essilortransitions.com).

### Eyewear

**Shauns California** recently debuted its Spring 2015 collection, which incudes five new sun styles and four ophthalmic styles.

The women’s Rona frame and Spey optical style have a metal temple design with recessed enamel color fill. The women’s Tacit boasts an asymmetrical front with a raised right brow, and the unisex More style has a front-on silhouette. Featuring a high metal bridge frame between two separate pieces of acetate, the absence of a traditional nose bridge brings a completely new perspective to a timeless shape.

Spring 2015 offers a selection of tortoise shell acetates (Rona, Hoy, Annan and Luss styles). Graduated acetate in a deep black cherry makes its debut in this collection as well, flowing from a near black to a cherry color (Carron, Hoy and Spey styles). This gradation technique is also used in a Bistre to topaz blue (Spey, Annan and Hoy styles). The popular mirrored lens is back in multiple colors, as well as in two different treatments. The “Super” family of mirrors is a multi-layered mirror for a strong, highly reflective effect (seen in blue and silver in the collection). A lighter “flash” application is used for a subtle mirror effect with a greater degree of translucency.

Visit [www.shaunscalifornia.com](http://www.shaunscalifornia.com).
March 2015


April 2015

- **15-17.** World Cornea Congress VII. San Diego Convention Center, San Diego, CA. Hosted by: ASCRS. To register, go to: http://corneacongress.org.
- **17-19.** NOA Spring Conference-CE Event. Embassy Suites, Lincoln, NE. Hosted by: Nebraska Optometric Association. To register, email ajohnson@assocoffice.net.
- **29-May 7.** Annual Educational Conference and Exposition. Red Lion Colonial Hotel, Helena, MT. Hosted by: Montana Optometric Association. To register, call (406) 443-1160 or email sweingartner@msmangement.com.
- **30-May 1.** Spring 2015 Convention. Pierre Ramkota, Pierre, SD. Hosted by: South Dakota Optometric Society. To register, email Deb Mortenson at deb.mortenson@pie.midco.net or go to southdakota.aoa.org.

May 2015

- **2-3.** 8th Annual Evidence Based Care in Optometry Conference. Turf Valley Conference Center and Resort, Ellicott City, MD. Hosted by: Maryland Optometric Association & John Hopkins-Wilmer Eye Institute. To register, call Annie Phan at (410) 486-9662 or email aphar@marylandoptometry.org.
- **3.** OptoWest Regional Conference. Anaheim Marriott Suites, Anaheim, CA. Hosted by: California Optometric Association. CE Hours: 6. Key Faculty: Steven Ferrucci, Bruce Onofrey, Mary Schmidt. To register, go to www.optowest.com, call Sarah Harbin at (916) 266-5022 or email sharbin@coavision.org.
- **3.** NECO Sunday Seminar Series CE. New England College of Optometry, Boston, MA. Hosted by: New England College of Optometry Alumni Association. CE Hours: 5. Key Faculty: Mark Dunbar, Michael Springer. To register, go to www.neco.edu/academics/continuing-education/sunday-series, call Margery Warren at (617) 587-5687 or email ce@neco.edu.
- **3-5.** CE in Italy. Hotel Silla, Florence, Italy. Hosted by: James Fanelli. CE Hours: 12. Key Faculty: James Fanelli, Carlo Pelino. To register, call James Fanelli at (910) 452-7225, go to www.CEinItaly.com or email jamesfanelli@CEinItaly.com.
- **3-7.** ARVO 2015. Colorado Convention Center, Denver, CO. Hosted by: The Association for Research in Vision and Ophthalmology. For information and registration, go to www.arvo.org/Annual_Meeting.
- **7-9.** CE in Italy. Palazzo Al Valabro, Rome, Italy. Hosted by James Fanelli. CE Hours: 12. Key Faculty: James Fanelli, Carlo Pelino, Joseph Pizzimenti. To register, call James Fanelli at (910) 452-7225, go to www.CEinItaly.com or email jamesfanelli@CEinItaly.com.

June 2015

- **5-7.** June “Summer” Conference. Harborside Hotel & Marina, Bar Harbor, ME. Hosted by: Maine Optometric Association. To register, call (207) 288-5033 or toll-free (800) 328-5033.
- **12-14.** 2015 Annual Meeting. Myrtle Beach, SC. Hosted by: North Carolina State Optometry Society. To register, email Adrienne Drollette at adrianne@nceyes.org.

26-July 5. A Comprehensive Update on Contemporary Eye Care. Northern European Capitals Cruise, departs Copenhagen, Denmark. Hosted by: Dr. Travel Seminars/The New Jersey Society of Optometric Physicians. Key Faculty: Randall Thomas. CE Hours: 12. To register, call Robert Pascal at (800) 436-1028, email info@DrTravel.com or visit DrTravel.com.

July 2015


10-12. 21st Conference on Clinical Vision Care. Southern College of Optometry, Memphis. Hosted by: OEP Foundation. CE Hours: 17. For information and registration, call Theresa Krejci at 800-447-0370, email theresakrejci@oep@verizon.net or go to www.oep.org.


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Diagnostic Quiz

Salt and Pepper Fundus?

By Andrew S. Gurwood, OD

History
A 37-year-old female presented to the office with a chief complaint of blurry vision at near. Her systemic history was remarkable for arrhythmia and hypertension for which she was properly medicated. She had no known allergies.

Her last eye exam was estimated at being seven years ago with no reported abnormalities or unusual diagnoses.

Diagnostic Data
The patient’s best-corrected acuity was 20/20 OU at distance and 20/25 OU at near. Her external examination was normal and there was no afferent pupillary defect.

Refraction uncovered mild hyperopia measuring 0.50 DS. The cornea and internal ocular health examination was normal, OU. Goldmann intraocular pressures measured 14mm Hg, both eyes. The pertinent dilated fundus findings are demonstrated in the photograph.

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

To find out, please visit Review of Optometry online at www.reviewofoptometry.com. Click on the cover icon for this month’s issue and select “Diagnostic Quiz” from the table of contents.

Retina Quiz Answers (from page 99): 1) b; 2) a; 3) a; 4) c.

Next Month in the Mag
April’s is Review of Optometry’s “Corneal Disease Report” issue.

Topics include:
• Corneal Harbingers of Systemic Disease (earn 2 CE credits)
• Slit Lamp Essentials: Anterior Stromal Puncture
• Comprehensive Evaluation and Management of Keratoconus/Ectasia
• Corneal Complications of Ocular Rosacea
• 10 Tips for Obtaining Better Visual Fields
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Fundus photographs of a 37-year-old female with a chief complaint of blurry vision at near.
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