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Leadership in clinical care

KEEPING UP WITH KERATITIS
Page 62

CORNEOSCLERAL CONCERNS:
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Fit your patients in PRECISION1® for Astigmatism.

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Reference: 1. In a study where n=78 eyes; Alcon data on file, 2020.
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NEW CONSENSUS ON KERATOCONUS
— EARN 2 CE CREDITS
Page 86
THE HORSEPOWER YOU NEED
TO LOWER IOP

Powerful IOP reduction with excellent tolerability¹,²
VYZULTA delivered up to 9.1 mmHg mean IOP reduction from baseline in pivotal trials.¹,²*

INDICATION

VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

• Increased pigmentation of the iris and periorbital tissue (eyelid) can occur. Iris pigmentation is likely to be permanent
• Gradual changes to eyelashes, including increased length, increased thickness, and number of eyelashes, may occur. These changes are usually reversible upon treatment discontinuation
• Use with caution in patients with a history of intraocular inflammation (iritis/uveitis). VYZULTA should generally not be used in patients with active intraocular inflammation
• Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. Use with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema
• There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products that were inadvertently contaminated by patients
• Contact lenses should be removed prior to the administration of VYZULTA and may be reinserted 15 minutes after administration
• Most common ocular adverse reactions with incidence ≥2% are conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%)

For more information, please see Brief Summary of full Prescribing Information on adjacent page.


*Pivotal study designs: Two Phase 3, randomized, multicenter, parallel-group studies, APOLLO and LUNAR, evaluating noninferiority of once-daily VYZULTA vs twice-daily timolol maleate 0.5% in patients with open-angle glaucoma or ocular hypertension. Primary endpoint was IOP measured at 9 assessment time points in study eye. APOLLO (VYZULTA, n=284; timolol, n=133) and LUNAR (VYZULTA, n=278; timolol, n=136).¹,²
BRIEF SUMMARY OF PRESCRIBING INFORMATION
This Brief Summary does not include all the information needed to use VYZULTA safely and effectively. See full Prescribing Information for VYZULTA.

VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024%, for topical ophthalmic use.
Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE
VYZULTA® (latanoprostene bunod ophthalmic solution) 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

2 CONTRAINDICATIONS
None

5 WARNINGS AND PRECAUTIONS
5.1 Pigmentation
VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% may cause changes to pigmented tissues. The most frequently reported changes with prostaglandin analogs have been increased pigmentation of the iris and peribulbar tissue (eyelid).

Pigmentation is expected to increase as long as latanoprostene bunod ophthalmic solution 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 VYZULTA, pigmentation of the iris is likely to be permanent, while pigmentation of the peribulbar tissue and eyelash changes are likely to be reversible in most patients. Patients who receive prostaglandin analogs, including VYZULTA, should be informed of the possibility of increased pigmentation, including permanent changes. 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 nevi nor freckles of the iris appear to be affected by treatment.

While treatment with VYZULTA® (latanoprostene bunod ophthalmic solution), 0.024% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly [see Patient Counseling Information (17) in full Prescribing Information].

5.2 Eyelash Changes
VYZULTA may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and the number of lashes or hairs. Eyelash changes are usually reversible upon discontinuation of treatment.

5.3 Intraocular Inflammation
VYZULTA should be used with caution in patients with a history of intraocular inflammation (iritis/uveitis) and should generally not be used in patients with active intraocular inflammation as it may exacerbate this condition.

5.4 Macular Edema
Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. VYZULTA 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.

5.5 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.

5.6 Use with Contact Lens
Contact lenses should be removed prior to the administration of VYZULTA because this product contains benzalkonium chloride. Lenses may be reinserted 15 minutes after administration.

6 ADVERSE REACTIONS
The following adverse reactions are described in the Warnings and Precautions section: pigmentation (5.1), eyelash changes (5.2), intraocular inflammation (5.3), macular edema (5.4), bacterial keratitis (5.5), use with contact lens (5.6).

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

VYZULTA was evaluated in 811 patients in 2 controlled clinical trials of up to 12 months duration. The most common ocular adverse reactions observed in patients treated with latanoprostene bunod were: conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%). Approximately 0.6% of patients discontinued therapy due to ocular adverse reactions including ocular hyperemia, conjunctival irritation, eye irritation, eye pain, conjunctival edema, vision blurred, punctate keratitis and foreign body sensation.

8 USE IN SPECIFIC POPULATIONS
8.1 Pregnancy
Risk Summary
There are no available human data for the use of VYZULTA during pregnancy to inform any drug associated risks. Latanoprostene bunod has caused miscarriages, abortion, and fetal harm in rabbits. Latanoprostene bunod was shown to be abortifacient and teratogenic when administered intravenously (IV) to pregnant rabbits at exposures ≥ 0.28 times the clinical dose. Doses > 20 µg/kg/day (23 times the clinical dose) produced 100% embryofetal lethality. Structural abnormalities observed in rabbit fetuses included anomalies of the great vessels and aortic arch vessels, domed head, sternal and vertebral skeletal anomalies, limb hypertension and malrotation, abdominal distension and edema. Latanoprostene bunod was not teratogenic in the rat when administered IV at 150 mcg/kg/day (87 times the clinical dose) [see Data]. The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2% to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies.

Data
Animal Data
Embryofetal studies were conducted in pregnant rabbits administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 19, to target the period of organogenesis. The doses administered ranged from 0.24 to 80 mcg/kg/day. Abortion occurred at doses > 0.24 mcg/kg/day latanoprostene bunod (0.28 times the clinical dose, on a body surface area basis, assuming 100% absorption). Embryofetal lethality (resorption) was increased in latanoprostene bunod treatment groups, as evidenced by increases in early resorptions at doses ≥ 0.24 mcg/kg/day and late resorptions at doses ≥ 6 mcg/kg/day (approximately 7 times the clinical dose). No fetuses survived in any rabbit pregnancy at doses of 20 mcg/kg/day (23 times the clinical dose) or greater. Latanoprostene bunod produced structural abnormalities at doses ≥ 0.24 mcg/kg/day (0.28 times the clinical dose). Malformations included anomalies of sternum, coarctation of the aorta with pulmonary trunk dilation, retroesophageal subclavian artery with absent brachiocephalic artery, domed head, forepaw hyperextention and hindlimb malrotation, abdominal distention/edema, and missing/fused caudal vertebrae.

An embryofetal study was conducted in pregnant rats administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 17, to target the period of organogenesis. The doses administered ranged from 150 to 1500 mcg/kg/day. Maternal toxicity was produced at 1500 mcg/kg/day (870 times the clinical dose, on a body surface area basis, assuming 100% absorption), as evidenced by reduced maternal weight gain. Embryofetal lethality (resorption and fetal death) and structural anomalies were produced at doses ≥ 300 mcg/kg/day (174 times the clinical dose). Malformations included anomalies of the sternum, domed head, forepaw hyperextention and hindlimb malrotation, vertebral anomalies and delayed ossification of distal limb bones. A no observed adverse effect level (NOAEL) was established at 150 mcg/kg/day (87 times the clinical dose) in this study.

8.2 Lactation
Risk Summary
There are no data on the presence of VYZULTA in human milk, the effects on the breastfed infant, or the effects on milk production. The developmental and health benefits of breastfeeding should be considered, along with the mother’s clinical need for VYZULTA, and any potential adverse effects on the breastfed infant from VYZULTA.

8.4 Pediatric Use
Use in pediatric patients aged 16 years and younger is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

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

13 NONCLINICAL TOXICOLOGY
13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility
Latanoprostene bunod was not mutagenic in bacteria and did not induce micronuclei formation in the in vivo rat bone marrow micronucleus assay. Chromosomal aberrations were observed in vitro with human lymphocytes in the absence of metabolic activation.

Latanoprostene bunod has not been tested for carcinogenic activity in long-term animal studies. Latanoprost acid is a main metabolite of latanoprostene bunod. Exposure of rats and mice to latanoprost acid, resulting from oral dosing with latanoprost in lifetime rodent bioassays, was not carcinogenic.

Fertility studies have not been conducted with latanoprostene bunod. The potential to impact fertility can be partially characterized by exposure to latanoprost acid, a common metabolite of both latanoprostene bunod and latanoprost. Latanoprost acid has not been found to have any effect on male or female fertility in animal studies.

13.2 Animal Toxicology and/or Pharmacology
A 9-month toxicity study administered topical ocular doses of latanoprostene bunod to one eye of cotonomylous monkeys: control (vehicle only), one drop of 0.024% bid, one drop of 0.04% bid and two drops of 0.04% per dose, bid. The systemic exposures are equivalent to 4.2-fold, 7.9-fold, and 13.5-fold the clinical dose, respectively, on a body surface area basis (assuming 100% absorption). Microscopic evaluation of the lungs after 9 months observed pleural/subpleural chronic fibrosis/inflammation in the 0.04% dose male groups, with increasing incidence and severity compared to controls. Lung toxicity was observed at the 0.024% dose.


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Missed Neuro Diagnoses Lead to Patient Harm

About a quarter of mistaken cases experienced some kind of adverse impact on health.

Neuro-ophthalmologists commonly encounter high rates of diagnostic error in the cases referred to them, which could lead to unnecessary or even inappropriate tests and treatments. To assess the impact, researchers decided to evaluate the extent to which patients were misdiagnosed prior to the neuro-ophthalmology referral and whether they suffered harm as a result. The findings offer a sobering look at the gap between generalist and specialist care in neuro-ophthalmic cases.

In this prospective cross-sectional study, researchers collected data from 496 patients regarding demographics, prior care, referral diagnosis, final diagnosis, diagnostic testing, treatment, patient disposition and impact of the neuro encounter. Referral diagnosis was incorrect in almost half the cases—49%, which is consistent with prior studies’ results. Furthermore, 26% of misdiagnosed patients suffered harm, which could have been prevented by earlier referral to neuro-ophthalmologists. Patients experienced inappropriate laboratory testing, diagnostic imaging or treatment prior to referral in 23% of cases, with higher rates for patients misdiagnosed prior to referral (34% of patients vs. 13% with a correct referral diagnosis.)

Seventy-six percent of inappropriate referrals were misdiagnosed, compared to 45% of appropriate referrals. The most common reasons for referral were optic neuritis or optic neuropathy, papilledema, diplopia or cranial nerve palsies and unspecified vision loss.

The most common sources of diagnostic error involved the physical exam, history taking, use or interpretation of diagnostic testing, and the generation and consideration of the differential diagnosis.

“These results emphasize the value of subspecialty-trained neuro-ophthalmologists in diagnosing and managing these potentially devastating conditions,” the authors noted in their study, published in Ophthalmology.

But there’s one silver lining to the study: optometrists were no worse than general ophthalmologists at neuro assessment. The study notes that there was “no meaningful difference between rates of misdiagnosis or rates of harm” between the two professions.

In Brief

Tear film breakup patterns in patients with thyroid eye disease appear to be different than those found in individuals with dry eye alone, new research published in Scientific Reports suggests. The incidence of each breakup pattern was similar in patients with simple dry eye without thyroid eye disease. On the other hand, among those with the condition, line breaks were the most frequently observed pattern (52%), followed by random patterns (23%), spots (19%) and dimple breaks (7%). These findings indicate that thyroid eye disease frequently induces aqueous-deficient dry eye, probably due to lacrimal gland involvement. But, it was found to be relatively mild. However, the severity of thyroid eye disease was not associated with tear film breakup pattern.


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Dra. Paulina Ramirez Neria

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Telemedicine was emerging in optometry long before COVID, but the spring 2020 lockdown prompted even some virtual-visit naysayers to rethink their position in order to ensure continuity of care.

One year later, practices are back open for business, prompting the question of whether telemedicine will retain as strong a role in optometry going forward, especially concerning ocular diseases such as glaucoma that often require in-person attention.

Still, others have already been sold on the advantages of virtual formats and are launching long-term telemedicine initiatives.

**Academia Test-drives Telemed Glaucoma Screenings**

In an effort to better engage at-risk and vulnerable populations and those least likely to have access to eye care, the CDC recently provided funding to Columbia University, the University of Michigan and the University of Alabama at Birmingham for five-year pilot investigations, collectively known as the Screening and Interventions for Glaucoma and Eye Health Through Telemedicine (SIGHT) studies.

The investigators are assessing the usefulness of community health workers in conducting on-site vision screenings, “patient navigators and glaucoma coaches” and telehealth to ensure follow-up eye care for those diagnosed with glaucoma and other eye diseases.

MI-SIGHT, the University of Michigan’s phase of the study, seeks to identify whether telemed eye health screening programs in local community clinics that serve higher risk glaucoma populations can detect the condition at a higher rate than the 2% found in the general population, explains researcher Paula Anne Newman-Casey, MD, MS, education director at the Kellogg Eye Center for eHealth and assistant professor and interim associate chair for research in the Department of Ophthalmology and Visual Sciences at the University of Michigan.

Through MI-SIGHT, ophthalmic techs will conduct vision screenings at two community clinics—the Hope Clinic, a free facility in Ypsilanti, and the Hamilton Community Health Network, a federally qualified health center in Flint. Each testing room is equipped with a vision chart, autorefractor, phoropter, trial lens set, Finhoff transilluminator, pachymeter, iCare tonometer and fundus/SD-OCT camera.

Once the tech has completed the eye health history and taken all of the requisite measurements, the data is securely sent to a university-based ophthalmologist for review. Ophthalmologists will then send their recommendations back to the tech, who will educate patients about their diagnoses and dispense and fit glasses as needed.

If a participant screens positive for glaucoma or suspected glaucoma, they will be randomized to either receive standard education or personalized glaucoma coaching to determine which approach helps more patients return for their recommended follow-up.

Fifty percent of people with glaucoma do not know they have the condition and are currently undiagnosed, Dr. Newman-Casey says. Adding to that sobering statistic, the need for glaucoma screening in underserved and at-risk populations, including those of African American descent and lower incomes, is great, she adds.

“The public health need to detect and treat glaucoma more effectively among people of African ancestry is critical in mitigating needless vision loss from glaucoma. In the MI-SIGHT program, we are testing whether using a telemedicine approach to embed glaucoma screening programs in trusted community clinics can help bridge this critical gap,” she says.

The MI-SIGHT study has great replication potential in other community clinics, Dr. Newman-Casey believes.

The purpose of using telemedicine is to provide eye health and glaucoma screening in trusted community clinics, since trust is one important barrier to engaging underserved populations in glaucoma screening, she says.

**Private Practice Poses Glaucoma Screening Challenges**

“I think telemedicine can play a part in glaucoma screening and management, which was particularly evident during the early days of the COVID-19 pandemic,” says Ian Gaddie, OD. However, he predicts that post-pandemic, telemedicine for glaucoma management will decrease as in-person visits return to normal.

“There are limits today governing the feasibility of telemedicine in glaucoma,” he explains. “How do you take a threshold field and have comparative data? How do you administer an OCT online? Equally important, how do you measure IOP by telemedicine?”

“Telemed could be a viable approach for glaucoma screening if a trained technician performs the appropriate testing from a remote location and the images are then sent to an eye care practitioner for a diagnosis,” says James Fanelli, OD.

(Continued on page 9)
INDICATIONS AND USAGE
ZERVIATE® (cetirizine ophthalmic solution) 0.24% is a histamine-1 (H1) receptor antagonist indicated for treatment of ocular itching associated with allergic conjunctivitis.

IMPORTANT SAFETY INFORMATION
ADVERSE REACTIONS
The most commonly reported adverse reactions occurred in approximately 1%-7% of patients treated with either ZERVIATE or vehicle. These reactions were ocular hyperemia, instillation site pain, and visual acuity reduced.

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

ZERVIATE™ (cetirizine ophthalmic solution) 0.24%

Brief Summary

INDICATIONS AND USAGE
ZERVIATE (cetirizine ophthalmic solution) 0.24% is a histamine-1 (H1) receptor antagonist indicated for treatment of ocular itching associated with allergic conjunctivitis.

DOSEAGE AND ADMINISTRATION
Recommended Dosing: Instill one drop of ZERVIATE in each affected eye twice daily (approximately 8 hours apart). The single-use containers are to be used immediately after opening and can be used to dose both eyes. Discard the single-use container and any remaining contents after administration. The single-use containers should be stored in the original foil pouch until ready to use.

CONTRAINDICATIONS
None.

WARNINGS AND PRECAUTIONS
Contamination of Tip and Solution: As with any eye drop, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle or tip of the single-use container to avoid injury to the eye and to prevent contaminating the tip and solution. Keep the multi-dose bottle closed when not in use. Discard the single-use container after using in each eye.

Contact Lens Wear: Patients should be advised not to wear a contact lens if their eye is red.

ZERVIATE should not be instilled while wearing contact lenses. Remove contact lenses prior to instillation of ZERVIATE. The preservative in ZERVIATE, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 10 minutes following administration of ZERVIATE.

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

In 7 clinical trials, patients with allergic conjunctivitis or those at risk of developing allergic conjunctivitis received one drop of either cetirizine (N=511) or vehicle (N=329) in one or both eyes. The most commonly reported adverse reactions occurred in approximately 3%–7% of patients treated with either ZERVIATE or vehicle. These reactions were ocular hyperemia, instillation site pain, and visual acuity reduced.

USE IN SPECIFIC POPULATIONS
Pregnancy
Risk Summary
There were no adequate or well-controlled studies with ZERVIATE in pregnant women. Cetirizine should be used in pregnancy only if the potential benefit justifies the potential risk to the fetus.

Data
Animal Data
Cetirizine was not teratogenic in mice, rats, or rabbits at oral doses up to 96, 225, and 135 mg/kg, respectively (approximately 1300, 4930, and 7400 times the maximum recommended human ophthalmic dose (MRHOD), on a mg/m² basis).

Lactation
Risk Summary
Cetirizine has been reported to be excreted in human breast milk following oral administration. Multiple doses of oral dose cetirizine (10 mg tablets once daily for 10 days) resulted in systemic levels (Mean Cmax = 311 ng/mL) that were 100 times higher than the observed human exposure (Mean Cmax = 3.1 ng/mL) following twice daily administration of cetirizine ophthalmic solution 0.24% to both eyes for 1 week. Comparable bioavailability has been found between the tablet and syrup dosage forms. However, it is not known whether the systemic absorption resulting from topical ocular administration of ZERVIATE could produce detectable quantities in human breast milk.

There is no adequate information regarding the effects of cetirizine on breastfeeding infants, or the effects on milk production to inform risk of ZERVIATE to an infant during lactation. The developmental and health benefits of breastfeeding should be considered along with the mother’s clinical need for ZERVIATE and any potential adverse effects on the breastfed child from ZERVIATE.

Pediatric Use: The safety and effectiveness of ZERVIATE has been established in pediatric patients two years of age and older. Use of ZERVIATE in these pediatric patients is supported by evidence from adequate and well-controlled studies of ZERVIATE in pediatric and adult patients.

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Carcinogenicity
In a 2-year carcinogenicity study in rats, orally administered cetirizine was not carcinogenic at dietary doses up to 20 mg/kg (approximately 550 times the MRHOD, on a mg/m² basis). In a 2-year carcinogenicity study in mice, cetirizine caused an increased incidence of benign liver tumors in males at a dietary dose of 16 mg/kg (approximately 220 times the MRHOD, on a mg/m² basis). No increase in the incidence of liver tumors was observed in mice at a dietary dose of 4 mg/kg (approximately 55 times the MRHOD, on a mg/m² basis). The clinical significance of these findings during long-term use of cetirizine is not known.

Mutagenesis
Cetirizine was not mutagenic in the Ames test or in an in vivo micronucleus test in rats. Cetirizine was not clastogenic in the human lymphocyte assay or the mouse lymphoma assay.

Impairment of Fertility
In a fertility and general reproductive performance study in mice, cetirizine did not impair fertility at an oral dose of 64 mg/kg (approximately 875 times the MRHOD, on a mg/m² basis).

PATIENT COUNSELING INFORMATION
Risk of Contamination: Advise patients not to touch dropper tip to eyelids or surrounding areas, as this may contaminate the dropper tip and ophthalmic solution. Advise patients to keep the bottle closed when not in use. Advise patients to discard single-use containers after each use.

Concomitant Use of Contact Lenses: Advise patients not to wear contact lenses if their eyes are red. Advise patients that ZERVIATE should not be used to treat contact lens-related irritation. Advise patients to remove contact lenses prior to instillation of ZERVIATE. The preservative in ZERVIATE solution, benzalkonium chloride, may be absorbed by soft contact lenses. Lenses may be reinserted 10 minutes following administration of ZERVIATE.

Administration: Advise patients that the solution from one single-use container is to be used immediately after opening. Advise patients that the single-use container can be used to dose both eyes. Discard the single-use container and remaining contents immediately after administration.

Storage of Single-use Containers: Instruct patients to store single-use containers in the original foil pouch until ready to use.

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ZER-04-20-MS-44


(Continued from page 6)

“Glaucoma is amenable to telemedicine as far as screening is concerned, but it’s a bit different with management because there are instances where you need to see the eye in vivo,” he says.

On the other hand, out of the plethora of conditions seen in a primary care clinic, glaucoma is one of the more amenable diseases for telemedicine management, although not exclusively, Dr. Fanelli adds. “A patient can’t be solely managed by telemedicine. Glaucoma screening lends itself to telemedicine because of the imaging capabilities we have. That would be a very different answer, for example, if we were talking about corneal ulcers.”

A patient sitting at home can’t be managed long-term for glaucoma, nor is there any screening capability from a home use perspective, he notes.

Unless the practice gives the patient a home tonometer or the practice has access to an online visual field instrument, Dr. Gaddie doesn’t see much utility in telemedicine for glaucoma. The exception is to see how the patient is tolerating medications or if refills are needed, he notes.

As for screening high-risk populations and those without access to eye care, Dr. Gaddie believes telemedicine could have some impact in environments specially designed with glaucoma testing capabilities. “This would be great in theory, but we have significant gaps to close to make this a mainstream reality,” he says.

Telemed Extends Beyond Glaucoma in Private Practice

Telemedicine can be supplemental to a practice, but it shouldn’t be used in isolation, says optometrist Kelsey Moody Mileski. In certain instances, the modality could be ideal for follow-up visits with established patients, and this approach may be more personal than a telephone call if a video chat format is used, she says. However, telemedicine isn’t practical if the evaluation requires equipment that isn’t available in a virtual format, she explains.

A traditional video-based telemed visit would be an option for minor ocular emergencies, such as a hordeolum or a subconjunctival hemorrhage.

Personally, Dr. Moody Mileski has found telemedicine visits to be helpful for dry eye patients after they have started a new therapy. If a fundus camera is available, additional screenings can be performed during a telemedicine visit, she says. “This is a wonderful option for patients who are seen in the emergency department (ED) or in another provider’s office, like neurology or endocrinology practices, where an eye care provider is not available,” she adds.

In this setting, an ED provider could obtain information that would not otherwise be available on a traditional video-based exam, such as visual acuity, pupil assessment and IOP. In this scenario, patients could be quickly diagnosed with more concerning and life-threatening conditions such as papilledema, retinal artery occlusion and/or diabetic retinopathy, she explains.

Still, telemedicine poses challenges, including time constraints, since virtual visits need to be scheduled, just like in-person exams. She suggests scheduling virtual visits at the beginning or end of the day, and for the same amount of time as an in-office visit. However, if the clinic is running behind, a patient may only wait a few minutes before leaving the video call, in which case the entire exam may be missed.

Another telemedicine challenge is technology. Although patients are becoming more familiar with Zoom and FaceTime during the pandemic, technology can still pose difficulties, particularly in older patients. Also, internet accessibility has to be considered for patients in underserved areas, she adds.

Dr. Moody Mileski offered telemedicine exams when her clinics were closed for routine care at the beginning of the pandemic. Still, she found few patients opted for a video-based exam, and instead preferred an initial phone consult.

Even though she currently isn’t using telemedicine, as she believes the exams are too limiting, Dr. Moody Mileski thinks virtual visits could play a secondary role in patient care.

A future directive for telemedicine in optometry could include eye care providers engaged in virtual consultations with other healthcare providers, such as those in the ED. In this modality, exam information and imaging such as fundus photography could be sent to the eye care provider for review.”

Myopic Progression Accelerated During Lockdown

The COVID-19 lockdown last spring prompted many individuals across the globe to pause routine health care. Looking into how this impacted eye care, a new study found an acceleration of myopia progression in Chinese children and teenagers during this time period; however, the trend was reversed after lockdown, suggesting that both accommodative spasm and structural change may have been contributing factors.

The investigation included 59,000 eyes of 30,000 participants. Refractive error was estimated through noncycloplegic autorefraction, and the spherical equivalent refraction (SER) was calculated as a sphere power of +0.5 cylinder power. Myopia of one eye was defined as SER of -0.5D or less, and high myopia was defined as SER of -6.0D or less. Myopic progression was defined as SER of -0.5D or less, +0.5 cylinder power. Myopia of one eye and high myopia was defined as SER of -0.5D or less, +0.5 cylinder power. Myopia of one eye and high myopia was defined as SER of -0.5D or less, +0.5 cylinder power.

Increased screen time may have caused accommodative spasm during this period.

The mean rates of SER change during the first three periods were -0.030D, -0.074D and 0.016D per month, respectively, which reflected negative deviations in the first two and a positive shift in the last.

Considering age and gender, the differences in myopic proportion and SER were significant in rounds three and four but not in round two. In round three, a greater risk of high myopia was noted; however, younger age and male gender were linked to a lower risk of myopia progression. Rounds one and two showed equal prevalence rates for myopia and SER, while round three curves were notably more myopic. The differences between rounds were more pronounced in younger students.

Additionally, the difference in SER change across the three survey periods was more remarkable in younger students, which indicated these individuals were more sensitive to myopic progression during the lockdown.

Also, the rate of myopic progression was greater during period two, followed by a hyperopic progression during period three. “We speculate that this may be explained by accommodative spasm during lockdown,” the researchers wrote in their paper. They suggest the impact of restricted outdoor time, increased screen time and limited indoor space on accommodative spasm.

At round four, about half a year after the lockdown was lifted, the accommodative spasm reversed, and the refractive state consequently became more hyperopic.

Exercise caution when interpreting evidence in other populations that live in different areas, the investigators suggested.

The mean rates of SER change during the first three periods were -0.030D, -0.074D and 0.016D per month, respectively, which reflected negative deviations in the first two and a positive shift in the last.

Increased screen time may have caused accommodative spasm during this period.

AOA Moves Annual Meeting to Denver

The American Optometric Association (AOA) announced today plans to move forward with an in-person format for Optometry’s Meeting this summer, but in a new location. Rather than host the annual conference in Anaheim, CA, as originally scheduled, the meeting will now take place in Denver, CO.

The dates will remain the same, June 24 through 26.

The AOA chose Denver due to the health and safety protocols put in place there and accreditations received from several certifying bodies, “all of which go beyond what is required by public health experts,” AOA President William Reynolds, OD, and American Optometric Student Association (AOSA) President Alex Bennet wrote in a letter to members. “Rest assured, we will continue to work closely with the convention center, government and local health officials leading up to the event and communicate any updates accordingly,” their letter stated.

For over a year, the optometry profession has had to be nimble in the face of changing circumstances, and the AOA feels this move is in keeping with that larger trend.

“The optometry community faced the growing challenge of the COVID-19 pandemic in 2020 through a dedicated approach of adapting, overcoming and advancing, always delivering the highest level of safe care for patients nationwide throughout the crisis,” the AOA statement said.

The AOA reaffirmed the organization’s commitment to hosting an in-person event. “It’s finally time to come together, and we are taking the same approach to convene our optometry community and leaders for AOA/AOSA Optometry’s Meeting in 2021, to celebrate the incredible resilience and compassion demonstrated by our members, as well as look forward and plan for an even brighter future.”

Pre-registration is now open online. The AOA says it asks for patience as it continues to work through details and ensure a safe meeting for all.
As one of the few contact lenses available in two base curves, 8.5mm and 8.8mm — Unity BioSync® with HydraMist® continues to provide patients exceptional comfort, high oxygen supply, outstanding vision and the most convenient daily wearing schedule. Choose the optimal fit for your patients with the only contact lens exclusive to the VSP network, and provide them the freedom to focus on everyday’s best moments.

To learn more, scan the code or visit unitybiosync.com.
Delay Between Cataract Surgeries Impacts Mental Health

The visual debilitation brought on by cataract development has been linked to depression, and the authors of a recent study wanted to dive deeper and investigate the effect of surgery on the need for medical interventions to alleviate psychological distress. A team from Taiwan assessed usage of mental health consults for depression and anxiety associated with both first- and second-eye surgeries, and sought to determine whether the time interval between procedures affects outcomes of psychological treatment.

A national database of claims was the basis of a cohort study spanning 10 years on 585,422 patients who received cataract surgeries for both eyes. The rates of mental health inpatient and outpatient consultations were analyzed with different time intervals between surgeries (less than three months, three to six months, six to 12 months and more than 12 months).

With patients who had previously sought mental health treatment before their first eye surgery, the number of mental health care consultations decreased postoperatively. This study also found that the decrease in number of mental health consults was less pronounced in patients who had not yet received second-eye surgery and those who received their second procedure more than three months after their first.

The number of mental health consultations was lowest among patients with a time interval of three months or less. For patients with an interval of more than 12 months, the predicted number of mental health consultations increased.

“In our results, mental health care utilization was most reduced in the group with time intervals of less than three months, suggesting that for patients more active in seeking medical help, the benefits of cataract surgery for reducing mental health care consultations are notable,” the authors noted in their paper.

“The authors also note there were some limitations of this study. For example, increases in the number of mental health consultations are not necessarily equivalent to a greater severity of clinical mental illness. “Although considering the actual number of mental health service consultations has merits, future studies should analyze the frequency of mental health consultations in combination with the actual mental health severity on a single study population,” the authors concluded.


ACNE MAY AFFECT MEIBOMIAN GLANDS

It’s not just an unfortunate rite of passage for many teenagers—acne may make people who suffer from the condition more susceptible to meibomian gland damage and tear instability, a recent paper published in Clinical and Experimental Optometry suggests. The research team from Turkey hypothesized that since acne vulgaris is a disease of the sebaceous glands, it could have potential effects on the ocular surface and tear homeostasis.

The investigation enrolled the right eyes of 70 individuals (34 patients with acne vulgaris and 36 healthy controls). Testing included tear breakup time (TBUT), Schirmer’s test, impression cytology from conjunctiva samples and meibography of the upper and lower eyelid meibomian glands.

The researchers found that TBUT was significantly lower in the study group, but the conjunctival impression cytology results were similar between both the subjects with acne and the healthy volunteers. Additionally, the investigators did not observe any grade three cytological changes in either group.

Meibomian gland loss in the upper eyelid of patients with acne vulgaris was about 19% compared with roughly 9% in the controls. Meibomian gland loss in the lower eyelid of the individuals with acne was also greater at approximately 16% compared with about 8% in the healthy participants. Individuals with acne vulgaris had a collective gland loss of 35% in both eyes.

In light of the study’s findings, a detailed ophthalmologic exam should be performed in patients with acne, the researchers suggested. Additionally, optometrists should remain vigilant for ocular surface pathologies when prescribing glasses or contact lenses for the visual rehabilitation of these young patients, they added.

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38%

For topical ophthalmic use

Initial U.S. Approval: 1998

INDICATIONS AND USAGE

LOTEMAX® SM is a corticosteroid indicated for the treatment of postoperative inflammation and pain following ocular surgery.

DOSAGE AND ADMINISTRATION

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

CONTRAINDICATIONS

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

WARNINGS AND PRECAUTIONS

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

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

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

Bacterial Infections: Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections.

In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection.

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

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

Contact Lens Wear: Contact lenses should not be worn when the eyes are inflamed.

ADVERSE REACTIONS

Because clinical trials are conducted under widely varying conditions, adverse reactions observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice. Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera. There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

USE IN SPECIAL POPULATIONS

Pregnancy: Risk Summary: There are no adequate and well controlled studies with loteprednol etabonate in pregnant women. Loteprednol etabonate produced teratogenicity at clinically relevant doses in the rabbit and rat when administered orally during pregnancy. Loteprednol etabonate produced malformations when administered orally to pregnant rabbits at doses 4.2 times the recommended human ophthalmic dose (RHOD) and to pregnant rats at doses 106 times the RHOD. In pregnant rats receiving oral doses of loteprednol etabonate during the period equivalent to the last trimester of pregnancy through lactation in humans, survival of offspring was reduced at doses 10.6 times the RHOD. Maternal toxicity was observed in rats at doses 1066 times the RHOD, and a maternal no observed adverse effect level (NOAEL) was established at 106 times the RHOD. The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2 to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies. Data Animal Data. Embryofetal studies were conducted in pregnant rabbits, and loteprednol etabonate by oral gavage on gestation days 6 to 18, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations at 0.1 mg/kg (4.2 times the recommended human ophthalmic dose (RHOD) based on body surface area, assuming 100% absorption). Spina bifida (including meningoecele) was observed at 0.1 mg/kg, and enencephaly and craniofacial malformations were observed at 0.4 mg/kg (17 times the RHOD). At 3 mg/kg (128 times the RHOD), loteprednol etabonate was associated with increased incidences of abnormal left common carotid artery, limb flexures, umbilical hernia, scoliosis, and delayed ossification. Abortion and embryofetal lethality (resorption) occurred at 6 mg/kg (256 times the RHOD). A NOAEL for developmental toxicity was not established in this study. The NOAEL for maternal toxicity in rabbits was 3 mg/kg/day. Embryofetal studies were conducted in pregnant rats administered loteprednol etabonate by oral gavage on gestation days 6 to 15, to target the period of organogenesis. Loteprednol etabonate produced fetal malformations, including absent innominate artery at 5 mg/kg (106 times the RHOD); and cleft palate, agnathia, cardiovascular defects, umbilical hernia, decreased fetal body weight and decreased skeletal ossification at 50 mg/kg (1066 times the RHOD). Embryofetal lethality (resorption) was observed at 100 mg/kg (2133 times the RHOD). The NOAEL for developmental toxicity in rats was 0.5 mg/kg (10.6 times the RHOD). Loteprednol etabonate was maternally toxic (reduced body weight gain) at 50 mg/kg/day. The NOAEL for fetal toxicity was 5 mg/kg. A peri-postnatal study was conducted in rats administered loteprednol etabonate by oral gavage from gestation day 15 (start of fetal period) to postnatal day 21 (the end of lactation period). At 0.5 mg/kg (10.6 times the RHOD), reduced survival was observed in live-born offspring. Doses ≥ 5 mg/kg (106 times the RHOD) caused umbilical hernia/incomplete gastrointestinal tract. Doses ≥ 50 mg/kg (1066 times the RHOD) produced maternal toxicity (reduced body weight gain, death), decreased number of live-born offspring, decreased birth weight, and delays in postnatal development. A developmental NOAEL was not established in this study. The NOAEL for maternal toxicity was 5 mg/kg.

Lactation: There are no data on the presence of loteprednol etabonate in human milk, the effects on the breastfed infant, or the effects on milk production. The developmental and health benefits of breastfeeding should be considered, along with the mother’s clinical need for LOTEMAX® SM and any potential adverse effects on the breastfed infant from LOTEMAX® SM.

Pediatric Use: Safety and effectiveness of LOTEMAX® SM in pediatric patients have not been established.

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic in vitro in the Ames test, the mouse lymphoma tk assay, or in the chromosomal aberration test in human lymphocytes, or in vivo in the mouse micronucleus assay. Treatment of male and female rats with 25 mg/kg/day of loteprednol etabonate (533 times the RHOD based on body surface area, assuming 100% absorption) prior to and during mating caused preimplantation loss and decreased the number of live fetuses/live births. The NOAEL for fertility in rats was 5 mg/kg/day (106 times the RHOD).

Distributed by: Bausch + Lomb, a division of Bausch Health US, LLC, Bridgewater, NJ 08807 USA

Manufactured by: Bausch & Lomb Incorporated, Tampa, FL 33637 USA

U.S. Patent Number: 10,596,107

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Based on 9669601 (Folded) 9669701 (Flat) Revised: 4/2020

LSM.0032.USA.21 Issued: 2/2021
**SM TECHNOLOGY™**
- Engineered with SM Technology for efficient penetration at a low BAK level (0.003%)\(^1,3\)
- ~2× greater penetration to the aqueous humor than LOTEMAX GEL (loteprednol etabonate ophthalmic gel) 0.5%\(^3\)

Clinical significance of these preclinical data has not been established.

**SMALL & MIGHTY SUBMICRON PARTICLES**

**PROVEN STRENGTH**
- 30% of LOTEMAX® SM patients had complete ACC resolution vs vehicle (15%) at Day 8 (N=371, P<0.0001)\(^1,2\)
- 74% of LOTEMAX® SM patients were completely pain-free vs vehicle (49%) at Day 8 (N=371, P<0.0001)\(^1,2\)

\(^1\)Pooled analysis of Phase 3 clinical studies. Study 1: 29% LOTEMAX® SM (N=171) vs 9% vehicle (N=172). Study 2: 31% LOTEMAX® SM (N=200) vs 20% vehicle (N=199), P<0.05 for all.

\(^2\)Pooled analysis of Phase 3 clinical studies. Study 1: 73% LOTEMAX® SM (N=171) vs 48% vehicle (N=172). Study 2: 76% LOTEMAX® SM (N=200) vs 50% vehicle (N=199), P<0.05 for all.

**Indication**
LOTEMAX® SM (loteprednol etabonate ophthalmic gel) 0.38% is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

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

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

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

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

- Contact lenses should not be worn when the eyes are inflamed.

- There were no treatment-emergent adverse drug reactions that occurred in more than 1% of subjects in the three times daily group compared to vehicle.

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

**References:**

**Discover more at**
[www.LOTEMAXSM.com](http://www.LOTEMAXSM.com)
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Amniotic membranes could be the best corneal treatment option in a number of cases.
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More than just an annoyance, this condition can alter refractive status in ways that complicate outcomes for surgical patients.
Paul M. Karpecki, OD

When the epithelium is compromised, sometimes the best approach is to let the eye start over with a clean slate.
Ethan Zimmerman, OD, Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

Doing a thorough job with corneal abrasions helps greatly.
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Using Photrexa® Viscous (riboflavin 5’-phosphate in 20% dextran ophthalmic solution), Photrexa® (riboflavin 5’-phosphate ophthalmic solution), and the KXL® system, the iLink™ corneal cross-linking procedure from Glaukos is the only FDA-approved therapeutic treatment for patients with progressive keratoconus and corneal ectasia following refractive surgery.*

**GET THERE IN TIME**

When you see patients with signs of keratoconus, don’t hesitate. Refer them for iLink™— the only FDA-approved cross-linking procedure that slows or halts disease progression and is eligible for commercial insurance coverage with over 95% of commercially covered lives.

**INDICATIONS**

Photrexa® Viscous (riboflavin 5’-phosphate in 20% dextran ophthalmic solution) and Photrexa® (riboflavin 5’-phosphate ophthalmic solution) are indicated for use with the KXL System in corneal collagen cross-linking for the treatment of progressive keratoconus and corneal ectasia following refractive surgery. Corneal collagen cross-linking should not be performed on pregnant women.

**IMPORTANT SAFETY INFORMATION**

Ulcerative keratitis can occur. Patients should be monitored for resolution of epithelial defects.

The most common ocular adverse reaction was corneal opacity [haze]. Other ocular side effects include punctate keratitis, corneal striae, dry eye, corneal epithelial defect, eye pain, light sensitivity, reduced visual acuity, and blurred vision.

These are not all of the side effects of the corneal collagen cross-linking treatment. For more information, go to www.livingwithkeratoconus.com to obtain the FDA-approved product labeling.

You are encouraged to report all side effects to the FDA. Visit www.fda.gov/medwatch, or call 1-800-FDA-1088.

*Photrexa® Viscous and Photrexa® are manufactured for Avedro. The KXL® System is manufactured by Avedro. Avedro is a Glaukos company.


MA-02164A
PM-US-0427
Presbyopia is a prevalent condition that affects nearly everyone at some point, typically beginning around age 40 and worsening with age. About 128 million Americans are currently affected, and the numbers continue to increase with the aging population. A progressive condition that reduces the eye’s ability to focus on near objects, presbyopia impacts patients’ daily activities and health-related quality of life. Patient frustration is also prevalent. Modern technology and extended screen time have changed the demands and needs of presbyopia patients. Despite the variety of surgical and nonsurgical treatment options, the results of a survey of 1339 patients, aged 40 to 55, found that 90% were frustrated or irritated with presbyopia. The survey also showed that 79% of presbyopes who saw an eye care professional (ECP) initiated a discussion about their near vision loss symptoms, but only half got the information they needed. Clearly, there is an opportunity to enhance interactions with these patients to improve satisfaction.

### Awareness Gap
Presbyopes are more frustrated with their vision and current treatment options than is often realized.

### Surprising results of recent patient dialogue research
With the objective of uncovering opportunities for better engagement with patients, 42 in-office dialogues were analyzed to understand the nature of discussions between ECPs and patients with presbyopia. The research focused on topics either associated with presbyopia as a condition or treatment options and expectations.

### Insight #1: Presbyopia was often downplayed in many ways
- Short/sparse conversations, with the average presbyopia discussion lasting less than 2 minutes
- Lack of probing beyond the initial expression of impaired near vision
- Minimizing language used regarding reading glasses
- Humor used to dispel any notion of pathology, but with the unintended consequence of presenting presbyopia as a nonserious burden patients must bear

### Insight #2: Patient education on presbyopia was limited
- There was little uniformity in explaining presbyopia, and ECPs rarely talked about the physical changes in the eye that lead to the condition

### Only 1 out of 5 nonspecific dialogues* involved an ECP explaining presbyopia in depth.

- Presbyopia was described as an unavoidable, natural part of aging that is progressive
- ECPs and patients struggled to find vocabulary to describe vision changes, with metaphors like “trouble reading” or “needing reading glasses” consistently used instead of “presbyopia”

*Dialogue not specific to presbyopia or not with a currently known presbyope.

ECP: “You are right on time...welcome to the club.”
ECP: “The only thing that causes this is the candles on your birthday cake.”
Patient: “I really don’t do anything. I have a little magnifying glass.”

Continued on back.
Insight #3: Quality-of-life (QoL) concerns were not fully addressed

ECPs tended to present the need for reading glasses as a simple fact of aging, whereas dependency on glasses was a common QoL concern for patients. Framing the discussion around “reading glasses” may prevent patients from thinking about or addressing presbyopia in the context of other activities where close distance/near vision is needed, such as screen usage, fishing, or threading a needle.

Insight #4: OTC reading glasses recommended the most

Reading glasses were the default treatment option for presbyopia, but limited support was provided. Most ECPs suggested specific magnification levels, but usually a range and rarely a single strength. Although reading glasses are OTC products, patients still wanted guidance. When other treatment options were presented, the advantages of reading glasses were reinforced: cost, simplicity, and noninvasiveness.

Patient: “So, what’s the best way to pick reading glasses… is it just trial and error?”

ECP: “It’s totally trial and error.”

Best practices to improve patient engagement

<table>
<thead>
<tr>
<th>Conduct deeper presbyopia discussions</th>
<th>Increase the educational content of presbyopia discussions</th>
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</thead>
<tbody>
<tr>
<td>More meaningful discussions can raise the importance of presbyopia to a level worth treating.</td>
<td>Explaining presbyopia in a way that is easily understood can empower patients to make informed treatment decisions.</td>
</tr>
<tr>
<td>• <strong>Initiate intentional conversations</strong> about presbyopia</td>
<td>• <strong>Use clear, consistent language</strong> and specific keywords</td>
</tr>
<tr>
<td>• <strong>Probe patients</strong> to share their concerns</td>
<td>• <strong>Describe the aging eye</strong> and the cause</td>
</tr>
<tr>
<td>• <strong>Avoid minimizing</strong> and downplaying presbyopia</td>
<td>• <strong>Assure patients that, while unavoidable, presbyopia can be treated</strong></td>
</tr>
</tbody>
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<table>
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<tr>
<th>Make QoL concerns a key part of the discussion</th>
<th>Explore and discuss treatment options in depth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gaining a full understanding of patients’ concerns can help guide treatment decisions.</td>
<td>Setting realistic expectations about treatment can increase patient satisfaction.</td>
</tr>
<tr>
<td>• Discuss the <strong>day-to-day impact</strong> on patients’ lives</td>
<td>• <strong>Ask questions</strong> to identify the best option for each patient’s lifestyle</td>
</tr>
<tr>
<td>• <strong>Acknowledge frustrations</strong> patients are experiencing</td>
<td>• <strong>Guide patients in making an informed decision</strong></td>
</tr>
<tr>
<td>• <strong>Ask patients about activities</strong> beyond reading where close distance/near vision is needed</td>
<td>• <strong>Discuss potential pros and cons</strong> of each treatment option</td>
</tr>
</tbody>
</table>

Enhancing patient interactions can help minimize frustrations

The presbyopia population and its needs are shifting, presenting both challenges and opportunities for raising the bar in patient satisfaction. Adopting the presbyopia dialogue strategies presented here can create better engagement with patients and minimize the frustration factor.

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I was very discouraged to see one of the major optometric journals publish an article about amblyopia (“The Generalist’s Guide to Amblyopia,” January 2021) without mentioning vision therapy as a treatment option. While VT may not be common practice for most eyecare professionals (though it should be the first line of treatment), it should be worth mentioning in an article about amblyopia.

It concerns me that optometry has aligned itself with the medical model and vision has been simplified to no more than a concern for visual acuity. We appear to have ignored our history and the principles that our unique profession was founded on; namely, the function of vision and the pinnacle of vision function, binocularity. Current research clearly shows that functional amblyopia exists because the two eyes cannot be fused in the brain. To truly solve amblyopia, one must solve the binocular problem at the brain level. How is patching a child with amblyopia (making them monocular) going to improve binocular vision? It cannot be that training an amblyopic eye independently rather than the visual system as a unit constitutes best practices.

It astounds me that optometry will consult and/or refer to ophthalmology but we do not refer (or even recognize) those within our own profession who successfully treat amblyopia daily. When will mismanagement of amblyopia be held to the same standards as mismanagement of glaucoma or a retinal condition?

Therapy for amblyopia has advanced much further than managing the condition with an eye patch alone. Let’s stop simplifying vision and thereby degrading our profession. The profession has expanded but let us not move the profession away from our roots: the diagnosis and treatment of functional vision problems.

When will mismanagement of amblyopia be held to the same standards as mismanagement of glaucoma or a retinal condition?

—Megan Lott, OD, FCOVD
Belle Vue Specialty Eye Care, Hattiesburg, MS

**CORRECTIONS**

**Eso or exo?**

A figure in the February article, “An Action Plan for Assessing Double Vision,” incorrectly described the results of Maddox rod testing as an eso deviation when it was an exo. The correct caption appears in the online version.

**Visual field not so bad**

A perimetry report in the February article, “Breaking Down Visual Fields in Glaucoma,” printed incorrectly, suggesting more end-stage disease than was present in the case. The online version has been updated and the correct result is reproduced here as well.

—George E. White, OD, FAAO
Residency director (ret.), Pennsylvania College of Optometry

**YOUNG ODs CAN REINVIGORATE ESTABLISHED PRACTICES**

I read with interest the February editorial (“Can’t Get There From Here”) on the plight of new graduates struggling to find positions that use their skills to the fullest.

There are—or should be—plenty of opportunities for young doctors to expand private practices by offering their more robust skill set to established offices. Many older ODs did not have the outstanding education that’s offered today in our training programs. These senior doctors can bring newer services into their practices simply through a hire rather than extensive training on their own part.

In addition to your example of glaucoma, newly minted optometrists have the potential to offer an established practice skills in any of the following realms: AMD, night vision complaints, diabetes and diabetic retinopathy, dry eye and MGD, computer-related visual symptoms, concussion and head trauma, cataract comanagement, eyelid lumps and bumps, blepharochalasis and ptosis, identification of retinal lesions, ocular safety of systemic meds, neurological conditions like MS or Parkinson’s, and specialty contact lenses, which are enjoying a renaissance thanks to sclerals.

Established ODs who say they can’t afford to pay the new grads should consider the tremendous increase in revenue these services will bring to their practices just among the established patients already being seen in their offices, let alone new ones. Some creative salary structuring can minimize the liability, such as a compensation plan giving the new OD a somewhat smaller base salary and 30% to 50% of the new fees that are collected. Both parties win! Finally, the established OD finds an eventual buyer for their practice—something they frequently complain is not available.

For the young OD, benefits are equally desirable, including income growth potential that’s absent in corporate positions, the career satisfaction of using all the skills and training that they invested their time in developing, and the security in knowing that an equity share provides a valued asset when the time comes to retire.

—George E. White, OD, FAAO
Residency director (ret.), Pennsylvania College of Optometry
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A Case of Nerves

ODs may hesitate to embrace neuro-ophthalmic cases, but you can be an essential player in care without shouldering it all.

It’s a white-knuckle situation for sure. A patient presents with what appears to be a swollen optic disc and complaints of nausea. Or maybe it’s a case of double vision that doesn’t fit the mold for strabismus. Perhaps, instead, an OCT scan suggests you have optic neuritis on your hands.

In any of these—and a dozen more—the pressure’s on.

So, it’s no surprise that neuro-ophthalmic diagnoses by generalists often don’t go well. As reported in this month’s news section, a prospective study of 496 patients sent to neuro-ophthalmologists found the referral diagnosis was incorrect 49% of the time. Misdiagnosed patients suffered harm in 26% of cases, and unfortunately these adverse effects could have been prevented by earlier referral to neuro-ophthalmology in nearly every case.

Those harms were as serious as it gets, including death due to delay in tumor diagnosis, failure to recognize TIA, progression of permanent vision loss, spontaneous CSF leak, irreversible strabismus and others of equal magnitude.

With all that in the air while a patient’s in your chair, the urge to refer out immediately has to be strong. And, indeed, in many cases that’s exactly what you should do. Not everyone can or should try to be a neuro-ophthalmic specialist—that seems to be underlying message of this study.

But an equally strong message is that there’s a dearth of those specialists out there. “Improving access to neuro-ophthalmologists has the potential to prevent patient harm, which is made challenging by the current shortage of neuro-ophthalmologists,” the researchers wrote in their paper on the study. “Improving incentives to attract trainees to subspecialize in neuro-ophthalmology will allow expanded access to patients who need care for these complex conditions.”

That’s not your problem—leave it for ophthalmology teaching institutions to sort out. What you can do, though, is hone your skills in triaging these cases to make sure they get a timely and accurate referral. The assessment stage, patient counseling and write-up of referral notes all seem like fertile ground for improvement of clinical and communication skills.

In the above-mentioned study, the most common sources of error involved deficiencies in the physical exam (36%), generation of a complete differential diagnosis (24%), history taking (24%) and use or interpretation of diagnostic testing (13%). That’s all bread-and-butter optometry at this point.

You may have noticed that we’re in the middle of a six-part series on comanagement (see this month’s article about cornea care on page 80). The connective tissue among the whole series is optometric leadership in screening patients and directing the effort.

As luck would have it, next month’s topic is neuro-ophthalmic care. I encourage you to make time for that one, and even to send us any thoughts on what could help you do a better job in neuro care out there in the trenches. We’ll work it into our coverage for later this year and beyond. Kindly drop me a line at editor@reviewofoptometry.com with any anecdotes, frustrations or longstanding problems you experience when conducting neuro workups. As the kids say, my DMs are open.
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New Options, Front to Back

Take a look at promising treatments for the cornea and retina, ready now or on the horizon.

This month’s issue showcases recent strides in cornea care, a somewhat sleepy category that doesn’t always get the attention it deserves. Among the other topics covered in this issue, dry AMD stands out as another perhaps overlooked area where optometrists can really shine if we put our minds to it.

Cornea-copia
Keratoconus is such a difficult condition because of its progressive nature, effects on vision and higher failure rates after transplants. Having new, more patient-friendly treatment options is greatly needed.

Imagine being able to treat keratoconus topically or crosslink the eye with just a scleral lens. Both concepts are in clinical trials. The first, a drug called IVMed-80 (iVeena), showed in its Phase II data a 1.8D mean reduction in K readings compared to placebo, an 11.3 letter improvement in BCVA over baseline and no treatment-related adverse events. The second concept, a scleral lens known as CXLens (TecLen), includes a built-in transducer that allows energy emission to crosslink the cornea. Riboflavin would be placed in the bowl of the lens and procedure would be completed in the optometrist’s office.

Although a very sound and comprehensive clinical study known as SCUT showed that using topical steroids in bacterial keratitis did not show greater vision loss, and actually improved outcomes in more severe cases, there are safeguards to keep in mind. First, be sure the patient has bacterial keratitis—avoid if there is a chance it could be fungal or HSV. Second, steroids are not recommended within 48 hours (preferably 72) if the infection may be caused by Pseudomonas (e.g., in contact lens wearers). Next, confirm the antibiotic you are using is working by clinical improvement, re-epithelialization or culture results that confirm the pathogen is susceptible to the antibiotic.

Optometrists see 85% of all comprehensive eye exams in the US, and patients frequently ask us what options exist for dry AMD.

Limbal stem cell deficiency, (LSCD), although rare, can be somewhat easy to overlook. Damage to the limbal stem cells or to their microenvironment leads to LSCD. As a result, the corneal epithelium is replaced with conjunctival epithelial cells. A good history that identifies a previous trauma—such as a chemical burn, contact lens overwear, Stevens-Johnson Syndrome or aniridia—will help in the diagnosis.

Presentations vary but LSCD typical manifests superiorly with corneal staining and sometimes haze, epithelial irregularity, poor healing and recurrent erosion. Treatments range from biologics like amniotic membrane (Prokera, BioTissue; Apollo, Atlas Ocular) and autologous serum (Vital’ Tears) to cytokine extract drops (Regener-Eyes), lubricants and inflammation control. Scleral lenses can be effective. In severe cases, a limbal stem cell transplant can deliver unbelievable results.

Owning AMD
Shifting gears from cornea, I can’t state enough how much I feel optometry needs to be the primary provider of dry AMD management. Optometrists see 85% of all comprehensive eye exams in the United States, and patients frequently ask us what options exist for this devastating condition. Five potential treatments are going through FDA trials for geographic atrophy, but much can also be done now.

Early detection can help a patient begin carotenoid supplementation. The most illuminating diagnostic for early AMD, with over 90% sensitivity and specificity, is dark adaptometry, which can now be performed with a device that resembles a virtual reality headset (AdaptDx Pro, Maculogix) and doesn’t require a separate room. If the patient has progressed to intermediate stage AMD (i.e., a least one large druse), AREDS2 formulations are proven to slow progression.

To monitor the disease, at-home testing (Notal Vision) can help encourage patient buy-in due to its convenience. Don’t forget to recommend high quality sunglasses, as well as a healthy diet and avoidance of smoking. Dry AMD treatments involving the complement system could be on their way, but now is the time to be active in management, as this condition is more common than glaucoma and DR combined.

It’s an exciting time to be in practice with so many new tools in the development pipeline, but waiting for FDA approvals before you begin managing these conditions will leave you behind, as there are many treatment and management options available today.
As you adapt to the changes our industry is facing, you’ll find there is also opportunity.

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Let’s Take a Gander at Gaps

We all have them in our schedules—worse now than ever. Don’t deny it.

Don’t lie. Each of you also has COVID-related gaps in your schedule. It’s inevitable. And if you claim you don’t, then just whom exactly did you pre-appoint during the shut down when the pandemic blew up?

No appointments for the larger part of 2020 must have impacted your ability to pre-appoint. And if you didn’t pre-appoint to begin with, I will never understand why. I can think of a few reasons though:

• It doesn’t work.
• It results in no-shows.
• Patients don’t know what their schedule will be a whole year from now.
• I myself don’t know what my schedule will be a whole year from now.
• There may be a pandemic.

That last one hits a little too close to home, I guess.

But if any of those reasons rings a bell, I guess you are actually used to gaps in your schedule, unless you work for the prison system or VA. Both have similar scheduling software, or so it seems.

I myself never had gaps in my appointment book when, as a student at the Pennsylvania College of Optometry, I volunteered to work in the eye clinic at Holmesburg Prison in Philadelphia. All the inmates were so bored that when the “screws” (prison lingo, you wouldn’t understand) asked on “the block” (again, don’t try to understand) if anyone had an eye problem, guess what? They all said they did, even if they had to scratch their eye with their fingernail on purpose. Hope you’re feeling better, Bob.

They’d come in wearing their “Jailhouse Schoolboys,” which were glasses they made in metal shop with windowpanes as lenses. Not good during riots—don’t ask me how I know that. I still regret not buying a pair for posterity, but $20 was a lot of money to a starving student in 1977. Still is.

Anyway, gaps exist. So, the question: what should you do about them? The answer: something that will fill them as quickly as possible. I want to present some good ideas and some bad ones. Hope these help:

1. Ask any patient who schedules an appointment if they would like to go ahead and get their kids in since they missed their annual exam last year. Good idea.
2. Ask any patient who schedules an appointment if they would be willing to buy a bunch of stuff when they come in to your office. Bad idea.
3. Make sure your office hours are convenient for your patients (especially for homebound workers). Offer some appointments early in the morning, at lunchtime and in the evening. Maybe even consider the weekend. Yuck! Good idea, even the last part.
4. Make sure you are open 24/7 just in case a patient happens to wander in. Bad idea.
5. Have an open bar. My task force is still debating the goodness or badness of this idea. Stay tuned; we’ll get back to you.
6. Office drive-through? Good idea.
7. Petting zoo? Bad idea.
8. Free contact lens delivery? Good idea.
10. Block scheduling? Great idea. (I feel the need to remind you, and by “you” I mean my staff members, that no doctor in his or her right mind wants to see an appointment at eight in the morning when the next appointment isn’t until 11:30. Not that this happened to me last Tuesday or anything. Block scheduling means all appointments must be physically touching one another, with respect of course, on the schedule.)
11. Blaming staff members for stuff? Bad idea, even if it’s literally all their fault. Just sayin’.

So, anyway, you get the picture. You have to get after your patients. They are past due because of a worldwide pandemic. At least that’s a better excuse than, “Umm, I forgot.”
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I recently saw a 38-year-old woman with a long history of recurrent corneal erosion (RCE) well-controlled with Muro 128 drops (sodium chloride 5%, Bausch + Lomb). A month ago, she began experiencing excruciating pain again. What’s going on?

“Severe eye pain upon waking is a symptom with a relatively small differential, and first on that list is RCE,” says Aaron Bronner, OD, of Pacific Cataract and Laser Institute. It’s thought that RCE is caused by irregularities in the extracellular architecture that keeps normal epithelial cells firmly adherent to their neighboring cells. Abrupt and absent anchoring junctions make the corneal epithelium more prone to sloughing. This results in both macroform RCE—large erosions that typically require medical management—and the smaller and self-limiting microform RCE.

Vicious Cycle

Though shearing forces to the epithelium occur with each blink, epithelial sloughing with RCE is most common at night. This is likely due to a combination of subtle epithelial edema from lid closure during sleep and decreased tear turnover at night. Then, during REM sleep or upon waking, the epithelium is eroded and the patient experiences marked pain, photophobia and lacrimation.

“This process creates a vicious cycle where the epithelium heals within hours or days, but anchoring filament reorganization takes as long as a few months,” Dr. Bronner says. “This results in periods where the patient may feel normal, but the epithelium can slough again without warning, and the process begins again.” In cases caused by scars or dystrophies, the anchoring filaments may never fully recover without surgical intervention.

If the patient goes through a window of two to three months without recurrence, they may never have another episode. But for those who recur or whose case arises from a traumatic or dystrophic source, the potential for recurrence persists for years.1

Management Options

To heal the acute epithelial defect, consider debridement and either a bandage soft contact lens (BSCL) or an amniotic membrane (AM). “Pair either of these with topical antibiotics while the epithelial defect is present,” Dr. Bronner says. Once the defect heals, he recommends continuing BSCL use for as long as three months.

Given its role in promoting corneal wound healing and its anti-inflammatory effects, an AM may be even more effective in the acute stage of the disease than a BSCL.2 However, it would probably need to be replaced with a bandage lens following initial AM removal when the epithelium has healed to support anchoring junction development.

Our patient had stopped the Muro 128 a month prior because she no longer found it available. A few weeks later, she began experiencing the same pain that first brought her in. “Eventually many patients will, whether directed to or not, discontinue the ‘salt water,’” Dr. Bronner warns. “Some of these patients may never have another episode, but in cases like our patient, RCE recurs shortly after cessation.” While she was in the office, we confirmed that both the Muro drops and ointment were available online. She restarted therapy and has since experienced total relief.

For those patients wishing to come off conservative therapy, anterior stromal micropuncture (ASP), debridement with diamond burr polishing (DBP) and phototherapeutic keratectomy (PTK) may be considered in select cases. “My personal experience is that ASP and DBP have higher rates of recurrence following their use,” Dr. Bronner says. “So, I currently recommend PTK for cases not curable with conservative management.”

As with all problems, there is no one RCE treatment that’s right for everyone. For those considering surgical intervention, the risks of pain, infection, poor wound healing and corneal scarring will have to be balanced with how they perceive conservative therapy’s effectiveness or inconvenience.


Dr. Ajamian is the center director of Omni Eye Services of Atlanta. He currently serves as general chairman of the education committee for SECO International. He has no financial interests to disclose.
INDICATION
Upneeq® (oxymetazoline hydrochloride ophthalmic solution), 0.1% is indicated for the treatment of acquired blepharoptosis in adults.

IMPORTANT SAFETY INFORMATION
WARNINGS AND PRECAUTIONS
• Alpha-adrenergic agonists as a class may impact blood pressure. Advise Upneeq patients with cardiovascular disease, orthostatic hypotension, and/or uncontrolled hypertension or hypotension to seek medical care if their condition worsens.
• Use Upneeq with caution in patients with cerebral or coronary insufficiency or Sjögren’s syndrome. Advise patients to seek medical care if signs and symptoms of potentiation of vascular insufficiency develop.
• Upneeq may increase the risk of angle closure glaucoma in patients with untreated narrow-angle glaucoma. Advise patients to seek immediate medical care if signs and symptoms of acute narrow-angle glaucoma develop.
• Patients should not touch the tip of the single patient-use container to their eye or to any surface, in order to avoid eye injury or contamination of the solution.

ADVERSE REACTIONS
Adverse reactions that occurred in 1-5% of subjects treated with Upneeq were punctate keratitis, conjunctival hyperemia, dry eye, blurred vision, instillation site pain, eye irritation, and headache.

DRUG INTERACTIONS
• Alpha-adrenergic agonists, as a class, may impact blood pressure. Caution in using drugs such as beta blockers, anti-hypertensives, and/or cardiac glycosides is advised. Caution should also be exercised in patients receiving alpha adrenergic receptor antagonists such as in the treatment of cardiovascular disease, or benign prostatic hypertrophy.
• Caution is advised in patients taking monoamine oxidase inhibitors which can affect the metabolism and uptake of circulating amines.

To report SUSPECTED ADVERSE REACTIONS or product complaints, contact RVL Pharmaceuticals at 1-877-482-3788. You may also report SUSPECTED ADVERSE REACTIONS to the FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

Please see next page for Brief Summary of full Prescribing Information.
UPNEEQ® (oxymetazoline hydrochloride ophthalmic solution), 0.1%, for topical ophthalmic use

*Each mL of UPNEEQ contains 1 mg of oxymetazoline hydrochloride, equivalent to 0.09 mg (0.09%) of oxymetazoline free base.

BRIEF SUMMARY: The following is a brief summary only; see full Prescribing Information at https://www.upneeq.com/Upneeq-PI.pdf for complete information.

1 INDICATIONS AND USAGE

UPNEEQ is indicated for the treatment of acquired blepharospasm in adults.

2 DOSAGE AND ADMINISTRATION

Contact lenses should be removed prior to instillation of UPNEEQ and may be reinserted 15 minutes following its administration. If more than one topical ophthalmic drug is being used, the drugs should be administered at least 15 minutes between applications.

3 CONTRAINDICATIONS

None.

4 WARNINGS AND PRECAUTIONS

5.1 Potential Impacts on Cardiovascular Disease

Alpha-adrenergic agonists may impact blood pressure. UPNEEQ should be used with caution in patients with severe or unstable cardiovascular disease, orthostatic hypotension, and uncontrolled hypertension or hypotension. Advise patients with cardiovascular disease, orthostatic hypotension, and/or uncontrolled hypertension/hypotension to seek immediate medical care if their condition worsens.

5.2 Potentiation of Vascular Insufficiency

UPNEEQ should be used with caution in patients with cerebral or coronary insufficiency, or Sjögren’s syndrome. Advise patients to seek immediate medical care if signs and symptoms of potentiation of vascular insufficiency develop.

5.3 Risk of Angle Closure Glaucoma

UPNEEQ may increase the risk of angle closure glaucoma in patients with untreated narrow-angle glaucoma. Advise patients to seek immediate medical care if signs and symptoms of acute angle closure glaucoma develop.

5.4 Risk of Contamination

Patients should not touch the tip of the single patient-use container to their eye or to any surface, in order to avoid eye injury or contamination of the solution.

6 ADVERSE REACTIONS

6.1 Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice. A total of 360 subjects with acquired blepharospasm were treated with UPNEEQ once daily in each eye for at least 6 weeks in three controlled Phase 3 clinical trials, including 203 subjects treated with UPNEEQ for 6 weeks and 157 subjects treated with UPNEEQ for 12 weeks. Adverse reactions that occurred in 1-5% of subjects treated with UPNEEQ were punctate keratitis, conjunctival hyperemia, dry eye, blurred vision, instillation site pain, eye irritation, and headache.

7 DRUG INTERACTIONS

7.1 Anti-hypertensives/Cardiac Glycosides

Alpha-adrenergic agonists, as a class, may impact blood pressure. Caution in using drugs such as beta-blockers, anti-hypertensives, and/or cardiac glycosides is advised.

Caution should also be exercised in patients receiving alpha adrenergic receptor antagonists such as in the treatment of cardiovascular disease, or benign prostatic hypertrophy.

7.2 Monoamine Oxidase Inhibitors

Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available data on UPNEEQ use in pregnant women to inform a drug-associated risk for major birth defects and miscarriage. In animal reproduction studies, there were no adverse developmental effects observed after oral administration of oxymetazoline hydrochloride in pregnant rats and rabbits at systemic exposures up to 7 and 278 times the maximum recommended human ophthalmic dose (MRHOD), respectively, based on dose comparison. [see Data]. The estimated background risks of major birth defects and miscarriage for the indicated population are unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2-4% and 15-20%, respectively.

Data

Animal Data

Effects on embryo-fetal development were evaluated in rats and rabbits following oral administration of oxymetazoline hydrochloride during the period of organogenesis. Oxymetazoline hydrochloride did not cause adverse effects to the fetus at oral doses up to 0.2 mg/kg/day in pregnant rats during the period of organogenesis (28 times the MRHOD, on a dose comparison basis). Oxymetazoline hydrochloride did not cause adverse effects to the fetus at oral doses up to 1 mg/kg/day in pregnant rabbits during the period of organogenesis (278 times the MRHOD, on a dose comparison basis). Maternal toxicity, including decreased maternal body weight, was produced at the high dose of 1 mg/kg/day in pregnant rabbits and was associated with an increase in pup mortality and reduced pup body weights. Delayed sexual maturation was noted at 0.1 mg/kg/day (14 times the MRHOD, on a dose comparison basis). Oxymetazoline hydrochloride did not have any adverse effects on fetal development at a dose of 0.05 mg/kg/day (7 times the MRHOD, on a dose comparison basis).

8.2 Lactation

Risk Summary

No clinical data are available to assess the effects of oxymetazoline on the quantity or rate of breast milk production, or to establish the level of oxymetazoline present in human breast milk post-dose. Oxymetazoline was detected in the milk of lactating rats. The developmental and health benefits of breastfeeding should be considered along with the mother’s clinical need for UPNEEQ and any potential adverse effects on the breastfed child from UPNEEQ.

8.4 Pediatric Use

Safety and effectiveness of UPNEEQ have not been established in pediatric patients under 13 years of age.

8.5 Geriatric Use

Three hundred and fifteen subjects aged 65 years and older received treatment with UPNEEQ (n = 216) or vehicle (n = 99) in clinical trials. No overall differences in safety or effectiveness were observed between subjects 65 years of age and older and younger subjects.

10 OVERDOSAGE

Accidental oral ingestion of topical intended solutions (including ophthalmic solutions and nasal sprays) containing imidazoline derivatives (e.g., oxymetazoline) in children has resulted in serious adverse events requiring hospitalization, including nausea, vomiting, lethargy, tachycardia, decreased respiration, bradycardia, hypotension, hypertension, sedation, somnolence, mydriasis, stupor, hypothermia, drooling, and coma. Keep UPNEEQ out of reach of children.

PATIENT COUNSELING INFORMATION

Advising the patient to read the FDA-approved patient labeling (Instructions for Use).

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PM-US-UPN-0203 01/21
Focus on refraction

By Marc B. Taub, OD, MS, Paul Harris, OD, and Alicia Groce, OD

Over the past five years, this column has covered many topics related to lenses and prisms, but it has not yet discussed yoked prisms in detail. As we all learned in optometry school, a prism shifts light, and the person shifts their eye in response. Specifically, yoked prisms shift images the same direction and amount in both eyes. If you stick with the basic understanding of prisms as chiefly ray shifters (1.00 cm for every 1.00 m traveled per 1.00 D of prism), you will miss their true power. Besides their ability to shift light and eyes, yoked prisms initiate spatial shifts, or shifts in a person’s center of gravity, causing an observed postural change, which triggers a shift and/or rotation in the pelvis. These changes have the potential to alter behavior and attention.

In the field of neuro-optometric rehabilitation, we use horizontal yoked prisms in the treatment of cases of homonymous hemianopsia, midline shift, neglect and sometimes unilateral spatial inattention. In patients with nystagmus who have a null point that requires a head turn, yoked prisms sometimes allow for the patient to maintain a straighter head position, alleviating future spinal issues.

Vertically oriented yoked prisms are used most often to improve behavior and attention. While the exact mechanisms are not fully understood, we know these prisms transform space beyond the shifts in the chief ray in the direction of the apex of the prism. Other spatial transformations are complex but understood. We find that patients on the autism spectrum, as well as those with attention difficulties, often respond positively to vertical yoked prisms.

The goal with prism is to improve and enhance looking behavior and to extend how long a patient is able to pay attention. This column focuses on the use of vertically oriented yoked prisms and includes two stories of success.

Case One

A six-year-old girl had an eye exam several weeks prior, but her father was concerned that she was still moving her body instead of her eyes while writing. The patient’s medical history included ADHD and autism. She uses both Focalin (dexmethylphenidate, Novartis) and guanfacine. She was born on time and has been meeting all developmental milestones but is mildly delayed. She is not currently reading and receives speech and occupational therapy several times per week. Her father described her as “very clumsy” and said she bumps into things a lot at home.

The patient was able to see 20/15 OD, OS and OU, had 20 seconds of stereo and was orthophoric at distance and near. Retinoscopy was +0.50 -0.50x090 OU. The monocular estimated method response through plano was +0.50 with fluctuations. Through +0.50, the response was plano with fewer fluctuations. A test of gross eye movement demonstrated significant issues on both pursuits and saccades, showing significant body overflow and head movement. When we were able to get her to sit down, the patient was fidgety in the chair, and she was jumpy otherwise.

We decided to trial 2.00 D of base-up yoked prism. Interacting with the hanging Marsden ball, the patient’s attention was more focused with the yoked prism and she was better able to catch the ball. Her father commented that he had never seen his daughter so focused.

This option has improved the lives of many patients; two are highlighted here.

About Drs. Taub and Harris

Dr. Taub is a professor, chief of the Vision Therapy and Rehabilitation service and co-supervisor of the Vision Therapy and Pediatrics residency at Southern College of Optometry (SCO) in Memphis. He specializes in vision therapy, pediatrics and brain injury. Dr. Harris is also a professor at SCO. Previously, he was in private practice in Baltimore for 30 years. His interests are in behavioral vision care, vision therapy, pediatrics, brain injury and electrodiagnostics. They have no financial interests to disclose.
and with such good hand-eye coordination.

We prescribed +0.25D of sphere with the 2.00D of base-up yoked prism OU and initiated a course of vision therapy focusing on eye and body movements. The patient is currently on her 10th session and progressing wonderfully.

**Case Two**

A minimally verbal four-year-old boy with autism was referred by his occupational therapist for a visual evaluation due to difficulty with visual attention to tabletop tasks and moving targets, as well as visual motor integration concerns. He receives occupational therapy services for sensory processing, visual motor integration, fine motor skills, bilateral integration, gross motor skills and difficulty with daily activities. He also receives speech therapy.

The patient had a limited attention span, so only the basic examination components and some modified visual perceptual tests were performed. He was able to see 20/20 OU at distance and 20/25- OU at near with Lea symbols. He did not like either eye being covered, so monocular acuities were not evaluated. His eye movements showed no restrictions but were jerky in nature and consisted of small microsaccades instead of a smooth saccade or pursuit movement.

His visual attention to our target was very limited (about one to two seconds at a time). His cover test revealed orthophoria at distance and near, and his near point of convergence was to the nose on two attempts; after that, he lost interest and attention. His distance retinoscopy was plano OD and OS, and his just-look retinoscopy showed bright, equal reflexes with a near retinoscopy finding of +0.50D of sphere OD and OS for the brightest, most stable reflex.

We chose to trial a higher amount of prism than usual to see if there was a positive response, since the patient’s visual attention was so fleeting. He wore 5.00D of base-up yoked prism and then 5.00D of base-down yoked prism while walking and interacting with the Marsden ball and other various objects around him. He walked in a straighter line and was more attentive to his surroundings with the base-up yoked prism; however, his attention was still limited. With the base-down yoked prism, he had an immediate negative response, was very anxious and ripped the glasses off.

At the following visit, we attempted testing again but did not get the response we had hoped for. During basic perceptual testing, the patient looked at each object for one to two seconds before looking away and then had to be redirected to where the object was on the table several times before he would visually attend.

We trialed a lower amount of prism this time (2.00D OU), and the response was instantaneous. The patient’s attention improved and he completed tasks that were previously challenging. We prescribed +0.25D of sphere with the 2.00D of base-up yoked prism OU.

The patient was re-evaluated two months later. His mom noted that he had been wearing his glasses at school, during occupational therapy and occasionally on the weekends at home when doing near tasks. He was able to perform several perceptual tests that he previously could not complete. Since he was already involved in so many other therapies, we decided to hold off on vision therapy. We monitor his progress every three to six months.

**Takeaways**

In patients with ADHD or autism who may benefit from yoked prism, we often try smaller amounts (1.00D to 5.00D) of base-up and base-down yoked prism per eye. The goal is to provide the smallest amount of yoked prism to create a positive change in awareness and a subsequent response. Higher amounts can also be trialed, but the goal remains the same. Typically, 1.00D to 3.00D of prism per eye is prescribed.

While seeing these patients can be challenging, you have the skill and knowledge to succeed. Not only can you improve the clarity of sight, but you can also enhance vision and encourage development of the visual process through the use of vertically oriented yoked prism.

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Biological Alternative
Amniotic membranes could be the best corneal treatment option in a number of cases.

In today’s clinical conversations, we can’t talk about treating the cornea without discussing amniotic membranes as a clinical option. While the code for amniotic membranes for ocular use has gone through a few iterations as far as definition is concerned, it currently is defined as:

- 65778: Placement of amniotic membrane on the ocular surface; without sutures.

It is considered a surgical code and application, even though the definition indicates “without sutures.” This category follows a different set of rules when creating the medical record and coding for application and follow-up visits.

There are additional considerations to keep in mind. All amniotic membranes may not be created equally, as the CMS has noted in a recent Local Coverage Determination (LCD):

“Amnion can be prepared for implantation a number of ways. Heat- or air-dried amniotic membrane loses some of its biologic properties and is not ideal for ocular surface rehabilitation.”

However, “the cryopreservation method allows for greater retention of the membrane’s structural, physiological and biochemical properties responsible for its dramatic healing and easier handling intraoperatively.”

Covered Indications
Amniotic membrane transplant for ocular conditions is considered medically reasonable and necessary for the following indications (the full list can be found in the online version):

- Failure of standard therapy for severe ophthalmological conditions demonstrated by ocular surface cell damage or failure and/or underlying inflammation, scarring or ulceration of the underlying stroma.

This category follows a different set of rules when creating the medical record and coding for application and follow-up visits.

- Circumstances where there is a severe condition requiring acute treatment with amniotic membrane such as chemical, thermal or radiation injuries, or Stevens-Johnson syndrome or limbal stem cell failure.
- Band keratopathy after treatment with other therapy such as surgery, topical medications, bandage contact lens or patching.
- Bullous keratopathy associated with an epithelial defect.
- Scleral melting.
- Corneal ulcer following initiation of anti-infective therapy and demonstration of clinical response for the purpose of healing the persistent epithelial defect.

While I realize that using an amniotic membrane for dry eye is a commonplace occurrence, many carriers will not cover dry eye as a diagnosis, but will cover the corneal sequelae caused by moderate to severe dry eye.

Other Considerations
Coding for a minor surgical procedure is not difficult, but realize that, in accordance with minor surgical rules, an office visit (either 920XX or 992XX) is generally not separately billable when performed on the same date of service as CPT code 65778. Reimbursement for the 65778 code itself already includes compensation for the office visit related to the decision to perform this minor surgical procedure. So, it would be the rare occasion to append modifier -25 to an E/M office visit performed on the same day as the application of an amniotic membrane.

Make sure that your medical record contains an operative report that specifies the details of the procedure and discharge instructions for the patient.

The global period for 65778 is zero days, so the postoperative period expires at the end of the day the service was performed. All follow-up examinations beginning on the day after the procedure was performed are separately billable. Another notable characteristic of the code is:

**Bilateral/unilateral status.** As with many surgical codes, this is a bilateral 150% procedure, meaning if you perform it bilaterally on the same day, you will get 100% for the first procedure and 50% for the fellow eye.

While reimbursement looks enticing, don’t let it guide your clinical decisions. Having amniotic membranes as part of your treatment arsenal is a big boon to your practice. Establish appropriate and proper medical necessity for the procedure to ensure that you survive an audit.

Send your coding questions to rcodingconnection@gmail.com.
I’m looking for a reputable solution for a digital acuity system – any suggestions?

- Pat Hamilton: Our office loves Acuity Pro. Six lanes.
- Andrew Hart: Great customer support when you have questions.
- Wayne Gilmore: Acuity Pro is developed and owned by an OD. Highly recommend.
- Ted Bryant: Another vote for Acuity Pro!
- Mindy Seaman: Have used Acuity Pro for over 10 years; love it!
Myopia control has been growing in popularity over the last two decades worldwide. In 2002, only 10 randomized control trials were published.1 To date, there are now over 170 peer-reviewed articles on myopia control.1 The growing interest in this topic, more so in certain populations and countries around the world than others, likely reflects the increasing incidence and prevalence of myopia. In 2000, there were an estimated 1.406 billion people in the world with myopia.2 By 2050, predictions estimate that number will grow to 4.758 billion.2 With global numbers on the rise, the myopia epidemic is quickly increasing in severity.

Although global numbers are increasing overall, myopia disproportionately affects some ethnicities more than others. For example, the Multi-Ethnic Pediatric Eye Disease Study (MEPDES) group showed small variations in myopia prevalence among preschool-aged children in the United States (1.2% non-Hispanic whites, 3.7% Hispanics, 3.98% Asians, 6.6% African Americans).3 In comparison, school-aged children in Hong Kong had a myopia prevalence of 18.3% in six-year-olds and 61.5% in 12-year-olds.4 Another paper illustrates that in east and southeast Asian countries, 80% to 90% of young adults are myopic.4 Even within the same country, differences exist in the prevalence of myopia between urban and rural settings.

There are many risk factors that affect the global trend of myopia, including high educational pressures and limited time outdoors.5 A recent cross-sectional study in China showed a myopic shift after home confinement due to COVID-19. The myopia prevalence increased 1.4-fold to three-fold in 2020 compared with the previous five years.6 Why should we be concerned about increasing levels of myopia? High myopia is associated with an increased risk of retinal detachment, cataract, glaucoma and myopic degeneration. Rhegmatogenous retinal detachment (RRD) results from axial elongation, vitreous liquefaction, posterior vitreous detachment and vitreoretinal degeneration, and affects 6.3 to 17.9 per 100,000 people.7 Independent of age, an eye with a spherical equivalent refractive error of -1.00D to -3.00D has a 4x increased risk of RRD compared with an emmetropic eye.7

Ortho-K is one lens option for patients undergoing myopia management.
An eye with a refractive error greater than -3.00D has a 10x increased risk. Patients with a longer axial length are more likely to have denser nuclear sclerosis and need cataract surgery. Myopia is associated with an increased prevalence of all forms of open-angle glaucoma and ocular hypertension. Myopic degeneration can lead to choroidal neovascularization and macular edema. Due to the increased risk of developing these sight-threatening conditions, researchers and clinicians alike have sought to more thoroughly understand myopia, including its process and progression.

**The Basics**

Myopia is caused by a complex, multifactorial interplay between many genetic and environmental factors. The extent of the influence of genetics is not fully understood. Myopia is highly heritable and has a 60% to 80% chance of being passed down. Additionally, almost 200 genetic loci have been identified to date as causing refractive error and myopia. Despite the scientific discoveries and advances we have made in the area of myopia genetics, genetic epidemiology and epigenetics, there is still much more research and knowledge to be gained.

The Homeostasis of Eye Growth and the Question of Myopia and the IMI—Report on Experimental Models of Emmetropization and Myopia. Studies provide a comprehensive and exhaustive review of many key animal studies that have established how we currently understand how the eye develops. It is assumed, like many other organs in the human body, that the eye undergoes both visually and non- visually guided regulations regarding eye growth. The axial length of the eye changes by either increasing the rate of growth of the entire eye or remodeling the sclera at the posterior pole. Additionally, the retina pulls back within the eye through choroidal thinning.

**Local mechanisms.** In animal studies, myopia is commonly induced by two mechanisms. The first involves creating an optical defocus with a minus lens. When a minus lens is placed in front of the eye, the image focuses behind the retina, causing the eye to grow. Conversely, when a plus lens is placed in front of the eye, the image focuses in front of the retina, causing the eye to inhibit its growth. This phenomenon is true of many animal species.

The second mechanism to induce myopia is through form deprivation. One study demonstrated that if only half of the visual field is occluded, myopia and axial elongation occur only in the hemiretinal area that is form-deprived. The same was true with full-field lenses. In addition, if diffusers or negative lenses cover half of the retina, only that half of the eye will become myopic and enlarged. Conversely, if positive lenses cover half of the retina, only that half will show inhibited eye growth. Even in eyes with a severed optic nerve or blocked action potential of the nerve, myopia can still be induced through form deprivation. These studies revealed that local ocular mechanisms can also be responsible for eye growth, rather than a neurological feedback loop involving the brain.

**Peripheral defocus.** The central 10° of our visual field contributes to 50% to 60% of the striate cortex. Researchers hypothesize whether the macula has more of an influence on the peripheral retina for eye growth. When the central retina was photoablated by a laser, and negative lenses were placed in front of the eye to induce hyperopic defocus, the magnitude of myopia produced in animal models was only slightly smaller in the foveal-ablated group compared with the control group. Therefore, the fovea is only responsible for a small amount of refractive development, and peripheral defocus likely plays a larger role.

**Light levels.** This factor plays an important part in emmetropization and decreasing the incidence of myopia. Outdoor light levels range from 30,000 lux to 130,000 lux, while indoor light levels are typically lower than 500 lux. Even on cloudy days, lux levels can measure between 10,000 and 20,000. Raising light levels in animal studies has been shown to increase retinal dopamine activity, which alters gene expression in the retina and reduces signals that produce axial elongation. Chicks that were raised in cages with 10,000 lux on a 12-hour light/12-hour dark cycle emmetropized normally. However, those raised in 500 lux became slightly myopic by 90 days (0.03D), and those raised in 50 lux became fully myopic by 90 days (-2.40D).

Human studies support the findings in animal studies when it comes to sunlight exposure. One report studied Chinese children in grades one through 12. The study compared six schools that added a 40-minute period...
of outdoor activities with six schools that continued with a normal schedule. The three-year cumulative incidence of myopia was significantly different. The children who had more outdoor activity had an incidence of myopia of 30.4% compared with 39.5% for the children who did not.24 When it comes to light exposure and myopia progression, the data has not yet arrived at a clear consensus.

**Treatment Options**

To date, the most viable myopia control options are atropine drops, soft multifocal contact lenses and orthokeratology (OK) lenses.

**Atropine.** These drops arguably have the most randomized control trial evidence for myopia control, namely from the landmark study Atropine in the Treatment of Myopia (ATOM).25,26 Initially, atropine was thought to slow the progression of myopia due to its cycloplegic effect on the smooth muscle fibers in the ciliary muscle. However, this theory no longer holds, as atropine is also capable of exhibiting myopia control effects on chicks. Atropine is a nonspecific muscarinic, cholinergic antagonist. Accommodation and light response in chicks are mediated by nicotinic receptors, rather than muscarinic receptors.15 Additionally, atropine may have a nonmuscarinic mode of action based on: “(1) the generally high dose of atropine required to prevent myopia in animal models, (2) its continued effect following ablation of cholinergic amacrine cells and (3) its effectiveness in inhibiting proteoglycan synthesis in isolated scleral cells.”12

In summary, atropine affects eye growth through cellular mechanisms that do not involve accommodation or ciliary muscle activity. The precise mechanism of action of muscarinic and nonmuscarinic pathways are not fully known or understood at this point.

**Low-dose atropine has shown clinical efficacy in myopia management.**

The ATOM studies demonstrated that atropine eye drops reduce myopia progression and axial elongation in children in a dose-related way.25,26 Atropine drops have no serious adverse or systemic effects. High doses of atropine (1%), however, have been shown to cause pupil dilation, accommodation loss and near vision blur to the point where the treatment was not previously accepted in the US. Additionally, a significant rebound effect has been seen with higher concentrations. Low-dose atropine (0.01%) has the best therapeutic index, with clinically few, relatively insignificant amounts of pupil dilation and near vision and accommodation loss.

More recently, the Low-Concentration Atropine for Myopia Progression (LAMP) study evaluated the efficacy and safety of 0.05%, 0.025% and 0.01% atropine eye drops over two years to determine the optimal concentration for longer-term myopia control. Over the study period, 0.05% atropine was more effective at slowing myopia progression compared with 0.01% atropine. It is important to note that the 0.01% atropine in the ATOM 2 study had similar effects to the 0.05% atropine in the LAMP study with regard to changes in spherical equivalent. Additionally, only 0.63D of accommodation was lost in 0.01% atropine patients in the LAMP study compared with 6.41D loss of accommodation at the 24-month mark with the 0.01% atropine in the ATOM 2 study.

The vast difference in accommodation loss between the two studies at the same 0.01% atropine dose calls into question the efficacy of the atropine drops used in the LAMP study. In my opinion, I would start off with 0.01%, and if the patient demonstrates progression, increase dosage to 0.025% or 0.05%.27,28

**Contact lenses.** Numerous multifocal contact lens and OK studies have been published to date. The most recent multifocal study was the Bifocal Lenses in Nearsighted Kids (BLINK) randomized clinical trial. The objective of the study was to determine whether center-distance soft multifocal contact lenses (Biofinity, CooperVision) slow myopia progression in children compared with single-vision lenses and whether high add power (+2.50D) slows myopia progression more than medium add power (+1.50D) over a three-year period.29 The study concluded that high add power multifocal contact lenses significantly reduce the rate of myopia progression compared with single-vision contact lenses of the same power and medium add power multifocal lenses.29

The results of two OK meta-analyses confirm the effectiveness of this modality for myopia control.30,31 A recent study specifically compared multifocal OK with conventional OK and demonstrated that patients who were treated with the former modality showed significantly less axial length progression.32
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Although various studies have shown myopia control efficacy for both multifocal contact lenses and OK lenses, each used different multifocal and OK lenses. To date, there hasn’t been a study comparing the different soft multifocal contact designs within either lens category.

These lenses create an optical defocus in the peripheral retina of the myopic patient. Multifocal contact lenses do so by additional correction incorporated into the lens, which is typically a center-distance design. OK lenses temporarily flatten the central cornea, allowing patients to see clearly during the day without correction. In addition, the peripheral cornea creates an optical defocus on the retina to control the patient’s myopia.

**Management Approaches**

My myopia treatment strategy includes encouraging patients to spend more time outdoors (at least two hours each day), place limits on screen time and take frequent breaks from electronic screens. As far as atropine drops go, I start my patients on 0.01% atropine and increase to 0.025% or 0.05% if progression is demonstrated. My patients who are ready for contact lenses are fitted into soft multifocal contact lenses (center-distance design) or OK lenses depending on patient and parent preference. The type of soft multifocal contact lens used depends on the patient’s refractive error and whether or not they have astigmatism.

Treatment-specific tests and frequency of follow-ups are often determined by the modality of treatment. For example, a patient on atropine would have their pupil size, pupil function and accommodation evaluated; whereas, a patient using an OK lens would have corneal topography performed.

In my opinion, cycloplegic refraction and axial length measurements must be performed at least once a year regardless of treatment modality. Subjective dry refraction often yields varying results, but autorefraction is more repeatable and objective. It may be worthwhile to conduct both. Accommodation must be neutralized in order to obtain an accurate reading of the refractive error. The only way to truly quantify progression is to obtain objective cycloplegic refractions and axial length measurements. In the case of OK lenses, it is difficult to gauge myopia progression via refractive error. For these patients, axial length measurements are your best guide in determining rate of progression and effectiveness of treatment. In all cases, it is helpful to know a patient’s rate of progression before treatment so the patient can serve as their own control.

For treatment with contact lenses, most practitioners opt to continue wear until the patient is out of the myopia progression phase, which typically occurs during their late teens or early twenties. Reasons for early discontinuation include adverse ocular health events or the patient’s desire to stop treatment. The World Health Organization currently recommends atropine drops be used for two years. Personally, I keep my patients on atropine until they’re out of the myopia progression phase.

Treatment should be stopped or switched if it is not sufficiently controlling progression as expected over the course of a year. While treatment does not halt myopia progression, I would consider a 50% reduction in progression to be sufficient. Additionally, treatment should be altered if there is a problem with compliance, safety or tolerance of visual side effects.

It is possible to augment contact lens treatment with low-dose atropine or vice-versa, as long as it is at the benefit of the patient. A recent study looking into the efficacy of combined OK lenses and 0.01% atropine found that axial length elongation was more prevalent in the OK group compared with the combination therapy group. The combination group was also 28% more effective in slowing myopia progression.33

**Clinical Takeaways**

In the coming years, research and studies will lead to an increase in science- and evidence-based medicine that will serve as the guiding principles to help solidify clinical guidelines for myopia control. Above all, we should always act in the best interests of our patients, which means finding the most optimal treatment, while also ensuring health and safety.
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Indications and Usage
For steroid responsive inflammatory ocular conditions of the palpebral and bulbar conjunctiva, cornea, and anterior segment of the globe and chronic anterior uveitis, corneal injury from chemical, radiation or thermal burns, or penetration of foreign bodies for which a corticosteroid is indicated and where the risk of superficial bacterial ocular infection is high or where there is an expectation that potentially dangerous numbers of bacteria will be present in the eye.

Important Safety Information
CONTRAINDICATIONS:
Most viral disease of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal disease of ocular structures. Hypersensitivity to any components of the medication.

WARNINGS & PRECAUTIONS:
- IOP increase – Prolonged use may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. IOP should be monitored.
- Aminoglycoside sensitivity – Sensitivity to topically applied aminoglycosides may occur.
- Cataracts – Posterior subcapsular cataract formation may occur.
- Delayed healing – May delay healing and increase the incidence of bleb formation. Perforations of the cornea or sclera have occurred. Slit lamp biomicroscopy, and fluorescein staining should be conducted.
- Bacterial infections – May suppress host response and increase secondary ocular infections. In acute purulent conditions, steroids may mask infection or enhance existing infection. If signs and symptoms fail to improve after 2 days, the patient should be re-evaluated.
- Viral infections – Use with history of herpetic simplex requires great caution. The course and severity of many viral infections of the eye (including herpes simplex) may be exacerbated.
- Fungal infections – Fungal infections of the cornea may occur and should be considered in any persistent corneal ulceration.
- Use with systemic aminoglycosides – Total serum concentration of tobramycin should be monitored.

ADVERSE REACTIONS:
The most frequent adverse reactions (<4%) to topical ocular tobramycin are hypersensitivity and localized ocular toxicity, including eye pain, eyelid pruritus, eyelid edema, and conjunctival hyperemia. The reactions due to the steroid component are increased intraocular pressure with possible development of glaucoma, and infrequent optic nerve disorder, subcapsular cataract, and impaired healing.

The development of secondary infection has occurred. Fungal infections of the cornea may occur. Secondary bacterial ocular infection following suppression of host responses also occurs.
Non-ocular adverse events (0.5% to 1%) included headache and increased blood pressure.

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

References:
TOBRADEX® ST (tobramycin/dexamethasone ophthalmic suspension) 0.3%/0.05%

Brief Summary

This Brief Summary does not include all the information needed to use TOBRADEX ST safely and effectively. Please see Full Prescribing Information for TOBRADEX ST at MyTobraDexST.com.

INDICATIONS AND USAGE

TOBRADEX ST is a topical antibiotic and corticosteroid combination for steroid-responsive inflammatory ocular conditions for which a corticosteroid is indicated and where superficial bacterial ocular infection or a risk of bacterial ocular infection exists.

Ocular steroids are indicated in inflammatory conditions of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe where the inherent risk of steroid use in certain infective conjunctivitides is accepted to obtain a diminution in edema and inflammation. They are also indicated in chronic anterior uveitis and corneal injury from chemical, radiation or thermal burns, or penetration of foreign bodies.

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

DOSAGE AND ADMINISTRATION

Recommended Dosing: Instill one drop into the conjunctival sac(s) every four to six hours. During the initial 24 to 48 hours, dosage may be increased to one drop every 2 hours. Frequency should be decreased gradually as warranted by improvement in clinical signs. Care should be taken not to discontinue therapy prematurely.

CONTRAINDICATIONS

Nonbacterial Etiology: TOBRADEX ST is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

Hypersensitivity: Hypersensitivity to any component of the medication.

WARNINGS AND PRECAUTIONS

IOP increase: Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. IOP should be monitored.

Aminoglycoside sensitivity: Sensitivity to topically applied aminoglycosides may occur.

Cataracts: May result in posterior subcapsular cataract formation.

Delayed healing: May delay healing and increase the incidence of bleb formation after cataract surgery. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids.

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

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

Fungal infections: Fungal infections of the cornea are particularly prone to develop with long-term use. Fungal invasion must be considered in any persistent corneal ulceration.

Use with systemic aminoglycosides: Use with systemic aminoglycoside antibiotics requires monitoring for total serum concentration of tobramycin.

ADVERSE REACTIONS

The most frequent adverse reactions to topical ocular tobramycin (TOBREX®) are hypersensitivity and localized ocular toxicity, including eye pain, eyelids pruritis, eyelid edema, and conjunctival hyperemia. These reactions occur in less than 4% of patients. Similar reactions may occur with the topical use of other aminoglycoside antibiotics.

Non-ocular adverse events occurring at an incidence of 0.5% to 1% included headache and increased blood pressure.

The reactions due to the steroid component are: increased intraocular pressure (IOP) with possible development of glaucoma, and infrequent optic nerve disorder; subcapsular cataract; and impaired healing.

Secondary Infection.

The development of secondary infection has occurred. Fungal infections of the cornea are particularly prone to develop with long-term use. Fungal invasion must be considered in any persistent corneal ulceration. Secondary bacterial ocular infection following suppression of host responses also occurs.

USE IN SPECIFIC POPULATIONS

Pregnancy and Nursing Mothers

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A NEW WAY TO EXPERIENCE REVIEW OF OPTOMETRY

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FIVE QUESTIONS ON DRY AMD MONITORING AND MANAGEMENT

Address these concerns and observing these patients will be more effective.

BY JESSICA HAYNES, OD
MEMPHIS

A
dge-related macular degeneration (AMD) is the leading cause of vision loss in patients over the age of 65.1 As there is no cure for AMD, current management focuses on reducing risk of conversion to advanced stages with formation of geographic atrophy (GA; advanced non-exudative AMD) or choroidal neovascularization (CNV; advanced exudative AMD). In addition, early detection of CNV is crucial in obtaining best visual outcomes for those who develop exudative AMD.

Development of advanced-stage disease poses the greatest threat of acuity reduction and significant loss of central vision, but even those without advanced disease may suffer visual deficits such as reduction in dark adaptation and loss of contrast sensitivity.2-6

Optometrists can manage patients with dry AMD from a medical perspective while also providing optical and low vision services to meet their visual needs. This article will focus on the medical management of those with non-exudative AMD by answering five common questions.

How Can I Tackle Compliance?
This perennial problem in many spheres of care is certainly a factor in AMD as well.

Knowledge is key. Patients who understand their condition and reason for being monitored are more apt to adhere to a mutual agreement. Those who are completely asymptomatic may not understand why they are monitoring their vision at home or why they are having periodic follow-ups when they have no visual symptoms.

On the other hand, those with reduced visual function may not understand why they are required to have office visits periodically when nothing is being done to "cure" their disease. They must understand the limitations that exist when managing AMD and the reason why we are monitoring them and making certain recommendations in order for them to adhere to follow-up.

A conversation about AMD and CNV may start off like this:

"AMD is a degenerative condition of the retina where the retina ages more quickly than it should. This accelerated aging can lead to loss of visual function as the retina structures weaken and no longer function normally."

"There is no cure for AMD, but I am going to make specific recommendations that will decrease your risk of vision loss. Many patients with AMD maintain functional vision for a lifetime, and while AMD can affect your central vision, it does not make you go completely blind because peripheral vision is still intact."

Visualize the problem. It is very useful for patients to be able to visualize alterations to their macula. Show them their fundus photographs or optical coherence tomography (OCT) images and walk them through the abnormalities that exist. Consider comparing them with normal photos or OCT scans.

I show patients with drusen their OCT images and point out that AMD affects the bottom layer of the retina (showing them the hyper-reflective band that is the retinal pigmented epithelium [RPE]), causing buildup of waste products (showing them areas of drusen). I explain that this layer of the retina is a barrier between the retina above and blood vessels (the choroid) underneath. This layer is weakened in patients with AMD, and these blood vessels can grow through and start to leak or bleed, causing a sudden and severe decline in vision (Figure 1).

About the author
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REFERENCES


Although this is very simplified pathophysiology, it can help patients understand why they must be monitored daily for conversion to wet AMD, even though they may be asymptomatic.

In patients who have GA, fundus autofluorescence (FAF) can demonstrate how certain areas of the retina have atrophied, causing the vision to have “good spots” and “bad spots” (Figure 2). Patients can understand visual deficits better and see that, while they may see 20/20 on the acuity chart, there are areas of the vision in with scotomas. On FAF, regions of GA will appear dark or hypo-autofluorescent.7

In addition to traditional diagnostic imaging, the AdaptDx Pro is an FDA-approved instrument from Maculogix that can demonstrate altered function of the retina.8,9 This may help both the patient and the physician understand how the retina is functioning in those with good visual acuity but significant visual complaints. It can also convince those who mistakenly feel that their vision is fine. Dark adaptation may also be altered prior to any clinical findings.8

Keep an eye on them. Until recently, we had no way of monitoring our patients outside of their scheduled follow-ups. We could send them home with an Amsler grid but couldn’t determine whether they were using it or if they understood how to use it. Now there is an FDA-approved at-home monitoring system for those who have intermediate AMD, the ForeseeHome system from Notal Vision. It takes the guesswork out of vision monitoring. Patients test their vision at home daily with a physician-prescribed monitoring device. Algorithms use hyperacuity to detect visual changes consistent with conversion to exudative AMD.

One report showed that 94% of eyes using ForeseeHome maintained 20/40 vision or better after conversion to exudative AMD vs. only 62% using current standard-of-care methods such as the Amsler grid.10 This is due to earlier detection with ForeseeHome. If conversion is detected, the prescribing physician will be alerted to contact the patient immediately. Physicians can access testing history through a portal or an app on their phone to know how often patients are testing, keeping them accountable (Figure 3).

When Should I Employ an Amsler Grid or Other At-home Monitoring Options?
Conversion to wet AMD remains the greatest risk for severe vision loss in those with AMD. Even with anti-VEGF injections, earlier detection of CNV leads to better visual outcomes. We rely heavily on home monitoring techniques and self-reporting to catch these conversions that can happen between visits. Educate patients with any stage of AMD about at-home vision monitoring with tools such as the Amsler grid. Those with intermediate AMD also qualify for the at-home vision monitoring system, ForeseeHome. Heavily consider that option for those with intermediate AMD.

Patients may ask, “Do I really need this device at my stage?” This is a major checkpoint to determine whether patients truly understand why they are being monitored. Introduce the program as AMD insurance. You hope to never use it, but it is there if you need it.

Figure 4 shows conversion to wet AMD that occurred less than three weeks after a scheduled monitoring appointment in a patient being followed for advanced non-exudative AMD. This well-educated patient noticed new-onset visual distortion of her Amsler grid and called our office. She was immediately brought in that afternoon where OCT confirmed...
presence of exudation, and treatment with anti-VEGF was initiated.

Anecdotal stories and images may be useful to share with other AMD patients. By sharing your personal experiences in managing AMD, it makes the disease seem much more relatable and less abstract for the newly diagnosed patient. It also emphasizes the importance of at-home monitoring.

When Should I Decide to Discuss Lifestyle Interventions?
Simply put, the sooner the better. Recommending healthy diet, exercise, smoking cessation and UV protection is generally a positive thing for any patient, but especially those with risk factors such as family history of AMD, systemic vascular disease, poor nutrition and history of smoking. Discuss with them how such factors increase risk of macular degeneration and how lifestyle modifications can decrease it.

Smoking remains the most important modifiable risk factor for development and progression of AMD. Many patients know that smoking can be bad for their heart or for their lungs, but it often comes as a surprise that it can also increase their risk of losing vision.

Making dietary recommendations can be daunting for those who are not used to this type of conversation. Even simple recommendations can spark a change for patients. One easy recommendation for patients can be to eat more naturally sourced food. Real, whole, unprocessed foods with a focus on brightly colored fruits and vegetables is a good place to start.

Additional suggestions may include observing a Mediterranean-type diet that involves intake of fatty fish rich in omega-3 oils at least twice per week and incorporating whole grain forms of rice, pasta and bread. For those with complicated systemic health, consider comanagement with the primary care physician or a nutritionist to facilitate dietary improvements.

Educate patients on the correlation between AMD and a sedentary lifestyle, obesity and vascular diseases such as hypertension, diabetes and hyperlipidemia. Consider making or using a quick reference sheet of the AMD lifestyle do’s and don’ts to give to your patients as shown in Figure 5.

When Should I Recommend Vitamin Supplementation?
Nutritional supplementation remains controversial. So, unfortunately, there is not an easy or straightforward answer. For a disease with no cure and very limited treatment options, there is a desire to do everything possible for a patient. This has to be appropriately balanced with current evidence. Recommendations for vitamin supplementation must always consider potential adverse effects alongside potential benefits.

The following is quoted from the American Academy of Ophthalmology’s Preferred Practice Patterns: “Antioxidant vitamin and mineral supple-

Fig. 3. On this ForeseeHome system report, a patient was testing regularly with almost six tests performed per week in the last month (red circle). I felt more comfortable then to extend his follow-up to six months.

Fig. 4. A patient with advanced dry AMD was seen with 20/20 OS. Less than three weeks later, the patient self reported visual changes and was brought into the clinic. OCT confirmed conversion to wet AMD OS and 20/25 vision (bottom).
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the optometrist’s choosing to patients who have intermediate AMD may go like this:

“There is evidence, for your stage of macular degeneration, that vitamin supplements do help to reduce the risk of developing more advanced stages of macular degeneration by 25%. I am going to recommend a supplement for you. This does not take the place of a healthy diet and lifestyle that involves wearing sunglasses and discontinuing smoking, which are equally important!”

What about those with early AMD or at-risk patients? While there is no substantial proof that vitamin supplementation reduces the risk of AMD progression or development in those with early-stage disease or at-risk patients such as those whose family members have AMD, there are multiple trials that suggest benefit to macular pigment optical density as well as both objective and subjective measures of visual function including visual acuity, contrast sensitivity and electoretinography with various types of vitamin supplementation.21-29

Supplementation with carotenoids such as lutein, zeaxanthin and mesozeaxanthin seems reasonable in this patient population, particularly in those with poor nutrition or visual limitations such as difficulty seeing at night.

The discussion with this patient population about vitamin supplementation may go as follows:

“While there is limited evidence that vitamin supplementation slows down the development/progression of macular degeneration at your stage, there is evidence to show that these vitamins may help the retina function more normally. I am going to recommend a supplement for you.”

How Should I Modify the Follow-up Schedule in Response to Presentation?

To plan follow-ups for your AMD patients, determine which clinical, OCT and FAF findings suggest increased risk for conversion to advanced AMD.

A suggested follow-up and management depending on the stage of disease is shown in Table 1.

It has been well documented that patients with any large-sized drusen 125nm or larger or with pigmentary changes on clinical exam are at increased risk for conversion to advanced AMD. This is approximately the width of a major retinal vein as it crosses the disc margin (Figure 6a). Those with both of these findings in each eye have a 50% chance of converting to advanced AMD in the next five years.30 The presence of either of these findings automatically puts a patient in the intermediate AMD category. It is highly important to make this distinction for three reasons.31 First, this is the point at which vitamin supplementation with full AREDS II-type vitamins is well accepted. Second, this is also when to use an at-home monitoring system. Third, it is pertinent to educate these patients and monitor them more carefully due to their increased risk of developing advanced AMD.

OCT imaging can be useful in visualizing small- to large-sized drusen and getting a sense of the size of the drusen deposits. In addition, it can help to detect a particular high-risk phenotypical variant of drusen deposition called reticular pseudodrusen (RPD), also known as subretinal drusenoid deposits. This variation is difficult to distinguish clinically and presents as small- to intermediate-sized drusen. On OCT, they appear as hyper-reflective deposits sitting on top of the RPE. Although small in size, patients with these deposits are even more likely than patients with large-sized drusen to develop advanced AMD.32-38

Also, these patients tend to be more symptomatic with worse visual function than other AMD phenotypes.39-41

Fig. 6. Look out for the following clinical findings: large soft drusen (A) and RPD (B). Soft drusen are the width of a major retinal vein as it crosses the disc (black arrow). RPDs are more evident on FAF and OCT. On OCT, they present as hyper-reflective deposits on top of the RPE (red arrows).
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Patients with RPD should be more heavily educated regarding increased risk of progression, need for nutritional and lifestyle modifications and need for more careful monitoring. FAF can also help to identify RPD (Figure 6b).

**Key Clinical Takeaways**

Optometrists often have the training, the knowledge and the access to all the tools necessary to manage patients with non-exudative AMD. Proper patient education, appropriate lifestyle, nutritional and nutraceutical recommendations, identification of risk factors and emphasis of both in-office and at-home monitoring can all lead to better visual outcomes for our patients with AMD. ■

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**TABLE 1. AMD FOLLOW-UP AND MANAGEMENT DEPENDING ON STAGE**

<table>
<thead>
<tr>
<th>AMD Stage</th>
<th>Fundus Findings</th>
<th>Management</th>
</tr>
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</table>
| Age-related changes/ at-risk patient | None, visible or only rare small drusen | • Reduce systemic and environmental risks.  
• Monitor yearly. |
| Early AMD       | Significant small drusen or any medium-sized drusen  | • Reduce systemic and environmental risks.  
• Recommend home monitoring with techniques such as the Amsler grid.  
• Discuss option of carotenoid supplementation.  
• Monitor six to 12 months, depending on risk. |
| Intermediate AMD| Significant medium-sized drusen, any large drusen or pigmented alterations | • Reduce systemic and environmental risks.  
• Discuss benefits of AREDS II.  
• Consider home monitoring device.  
• Monitor every four to six months, depending on risk. |
| Advanced AMD    | Presence of GA or CNV                                 | • Reduce systemic and environmental risks.  
• Discuss benefits of nutritional supplementation.  
• Recommend home monitoring.  
• Immediate referral for possible anti-VEGF injections for those with CNV.  
• Monitor GA every six to 12 months, depending on risk.  
• Consider low vision referral if appropriate. |

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A GLAUCOMA STARTER KIT: THE PATIENT IN YOUR CHAIR

Applying personalized medicine to this chronic disease is critical to ensure the best outcomes.

Glaucoma is a significant public health problem but represents an opportunity for doctors to make a positive impact in long-term eye care. In Part 1 last month, we outlined some of the reasons why an OD would pursue a glaucoma-focused clinic. New technologies and a renaissance of established techniques provide doctors with an incredible arsenal of testing to comprehensively care for patients, to prevent vision loss and quality of life reduction arising from glaucoma.

Part 2, we take a step into the consulting room and describe the diagnostic and management processes involved in glaucoma.

The Diagnosis Paradigm

The process of identifying glaucoma is complex, with no single cut-off criterion for diagnosis, unlike other lesion-based diagnoses like age-related macular degeneration and diabetic retinopathy. Although glaucoma is the most common optic neuropathy, it shares several features with other conditions that also manifest with visual field (VF) defects, loss of neural tissue or changes at the optic nerve. Glaucoma remains a diagnosis of exclusion when assessing suspected optic nerve head disease. Careful inspection of the nerve and surrounding features can help differentiate these conditions.

The common mimickers of glaucoma and their clinical features that overlap with or differentiate each entity from glaucoma are shown in Table 1.

To aid this process, we recommend four key considerations in the differential diagnosis of glaucoma:

1. **Patient profile, ocular and medical history.** For example, consider a history of trauma in the context of a pale but intact neuroretinal rim with neural tissue thinning and a VF defect, which points toward a traumatic optic neuropathy rather than glaucoma. Similarly, the patient’s age is also helpful, as several optic neuropathies mimicking glaucoma often manifest in younger patients and may point clinicians toward a hereditary or congenital cause instead.

2. **Longitudinal data.** Glaucoma is a progressive optic neuropathy; thus documented stability in the presence of untreated structural or functional loss precludes glaucoma as a diagnosis. In cases such as myopic optic neuropathy where there is significant overlap in the clinical presentation, longitudinal data is key for differentiating between these disease entities and can prevent unnecessary treatment.


3. “**Characteristic**” changes to the optic nerve and peripapillary region. A defining feature of glaucoma is presence of neuroretinal rim thinning. As such, loss of neural tissue (e.g., nerve fiber layer thinning) in the absence of visible optic nerve head changes points towards a non-glaucomatous etiology. Pallor of the nerve head is also not a classic feature of glaucoma and warrants investigation into other causes of optic atrophy. These signs should signal the need for further neuro-ophthalmic assessment, such as color vision.

Optical coherence tomography angiography (OCT-A) is also useful in evaluating non-glaucomatous optic neuropathies, as it can highlight vascular changes that are not visible using traditional OCT or funduscopically. Although glaucoma has been shown

About the authors

Dr. Phu is a clinician-scientist with academic and clinical positions at the Centre for Eye Health in Kensington, New South Wales, Australia, and the School of Optometry and Vision Science, University of New South Wales. He is a Fellow of the American Academy of Optometry and a Diplomate in glaucoma. Dr. Wang is a research and clinical staff optometrist at the Centre for Eye Health. She is a Fellow of the AAO. They have no financial interests to disclose.
to result in a reduction in OCT-A parameters such as vessel density, this typically does not occur until later in the disease process. A marked reduction in vessel density in the presence of shallow neuroretinal rim loss points to an ischemic or congenital condition rather than glaucoma (Figure 1).

4. The pattern of structural and functional loss. Retrograde degeneration presents with classic patterns of both structural and functional loss, which allow for retinotopic mapping to the location of insult. Carefully scrutinize patients presenting with midline respecting structural or functional loss for a non-glaucomatous etiology and consider neuroimaging to investigate other diseases of the visual pathway.3

Paradigm shifts in glaucoma diagnosis have arisen with the changing availability of specific devices or techniques such as fast VF testing and progression analysis, high resolution structural examination and intraocular pressure (IOP) profiling. Improved understanding of the multifactorial pathophysiology of glaucoma has also underscored the need to perform other non-ocular assessments to rule out contributions from systemic and non-ocular comorbidities.

Personalized Medicine

After a diagnosis is made, the doctor should then develop a management plan with the patient and the rest of the health care team.4 A commonly deployed treatment algorithm in the past is shown in Figure 2A, which demonstrates the concept of treatment escalation with increasingly invasive IOP-lowering therapies. A more recent stepwise algorithm that we have suggested is shown in Figure 2B. In the updated algorithm, several significant changes to clinical care are highlighted:

1. Determining the need for treatment.
2. Preservative-free drops as primary therapy.
3. Primary laser (or non-drop) therapy.

Does the patient even require treatment? One of the biggest changes in recent years is the concept of the need for therapy. The trajectory of glaucoma differs for each patient. The seminal Early Manifest Glaucoma Trial gave clinicians important information about the natural history of glaucoma.5 From those results we know that pseudoexfoliative glaucoma tends to progress the quickest, followed by high-tension glaucoma and then normal-tension glaucoma as the slowest. However, even within the separate groups of glaucoma subtypes, there is significant variability. Therefore, it is incumbent on the clinician to determine the trajectory of their individual patient.

The importance of disease trajectory lies in the fundamental reasons for treating glaucoma: to preserve vision and maintain quality of life. In cases where the patient’s disease trajectory is not likely to significantly

Fig. 1. Funduscopic and OCT findings for three contrasting patients: (A) A patient with classic primary open-angle glaucoma. This disc appears cupped with a gradient of retinal nerve fiber layer loss and a shallow reduction in perfusion on OCT-A. (B) A patient with superior segmental optic nerve hypoplasia. This disc has a sharply delineated absence of neural tissue in the superior rim, with a marked loss of nerve fiber layer, perfusion and visual function. The sectoral nature of the loss in addition to the attenuation and absence of retinal vasculature is indicative of a congenital vascular anomaly rather than glaucoma. (C) A patient with an ischemic retinal nerve fiber layer defect. Unlike in patient A, the neuroretinal rim appears intact and is thus incompatible with glaucoma. Supplementary imaging using OCT-A confirms a very deep and focal loss of perfusion that is not typical in early glaucoma as implied by the shallow VF defect and little neural loss.
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TABLE 1. CLINICAL FEATURES OF OPTIC NEUROPATHIES THAT MIMIC THE APPEARANCE OF GLAUCOMA

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>OVERLAPPING FEATURES WITH GLAUCOMA</th>
<th>DIFFERING FEATURES WITH GLAUCOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myopic optic neuropathy</td>
<td>- Loss of the neuroretinal rim with associated nerve fiber layer and ganglion cell thinning</td>
<td>- Nonprogressive</td>
</tr>
<tr>
<td></td>
<td>- Concordant VF defect(s)</td>
<td></td>
</tr>
<tr>
<td>Traumatic optic neuropathy</td>
<td>- Typically unilateral nerve fiber layer and ganglion cell thinning</td>
<td>- History of trauma</td>
</tr>
<tr>
<td></td>
<td>- Concordant VF defect(s)</td>
<td>- Intact but pale neuroretinal rim</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Non-progressive</td>
</tr>
<tr>
<td>Ischemic optic neuropathies</td>
<td>- Optic disc cupping (common in anterior ischemic optic neuropathy)</td>
<td>- Current or previous disc edema</td>
</tr>
<tr>
<td></td>
<td>- Nerve fiber and ganglion cell layer thinning can be focal or diffuse</td>
<td>- Neuroretinal rim pallor or chalky white disc</td>
</tr>
<tr>
<td></td>
<td>- Concordant VF defect(s)</td>
<td>- Marked reduction in capillary density using OCT-A</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Nonprogressive</td>
</tr>
<tr>
<td>Hereditary optic neuropathies</td>
<td>- Bilateral nerve fiber and ganglion cell layer thinning</td>
<td>- Positive family ocular history</td>
</tr>
<tr>
<td></td>
<td>- Concordant VF defect(s)</td>
<td>- Early onset–atypical patient profile</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Neuroretinal rim pallor</td>
</tr>
<tr>
<td>Congenital optic neuropathies</td>
<td>- Loss of the neuroretinal rim with associated nerve fiber layer and ganglion cell thinning</td>
<td>- Nonprogressive</td>
</tr>
<tr>
<td></td>
<td>- Concordant VF defect(s)</td>
<td>- Topless disc appearance</td>
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<tr>
<td></td>
<td></td>
<td>- Sharp demarcation between normal and abnormal VF</td>
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<tr>
<td></td>
<td></td>
<td>- Marked reduction in vessel density using OCT-A</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Non-progressive</td>
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<tr>
<td>Retrograde degeneration</td>
<td>- Nerve fiber and ganglion cell layer thinning</td>
<td>- Vertical midline respecting pattern of loss</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Neuroretinal rim pallor</td>
</tr>
<tr>
<td>Compressive/ infiltrative</td>
<td>- Nerve fiber and ganglion cell layer thinning</td>
<td>- Current or previous disc edema</td>
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<tr>
<td></td>
<td></td>
<td>- Peripapillary hemorrhages or exudates</td>
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<td></td>
<td></td>
<td>- Neuroretinal rim pallor</td>
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<td></td>
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<td>- Meningeal and optic nerve enhancement with orbit MRI</td>
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<td>Inflammatory</td>
<td>- Nerve fiber and ganglion cell layer thinning</td>
<td>- Neuroretinal rim pallor</td>
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<td></td>
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<td>- Current or previous disc edema</td>
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<td>- Relative afferent papillary defect</td>
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</table>

Affect their quality of life, any activated treatment may conversely lead to a reduction in quality of life. Other considerations include the costs of treatment. In such cases, there may be a reason to monitor without treatment to mitigate these issues. **Preserved or non-preserved?** Traditionally, topical therapy has been the mainstay, first-line treatment for glaucoma, with prostaglandin analogs preferred due to their convenient once-daily dosing and favorable safety profile. However, benzalkonium chloride in glaucoma drugs is known to detrimentally affect the ocular surface, especially with long-term use.** Preservative-free formulations have been suggested as an alternative. Recent studies have shown that preservative-free medications are similarly effective.** Therefore, clinicians should weigh the potential advantages and disadvantages of both formulations before proceeding. **SLT as first-line.** In suitable patients, selective laser trabeculoplasty (SLT) may be a viable first-line treatment that addresses fundamental issues with topical therapy such as compliance and adverse effects. While the benefits of SLT are clear, doctors should remember that, in patients with IOP lower than 21mm Hg (i.e., normal-tension glaucoma), the amount of IOP reduction achieved using SLT was lower. Some studies have shown that at pressure levels below 15mm Hg, the probability of success is less than 30%.** Compared with medical therapy, SLT runs the risk of being an episodic point-of-care that may lead to patient non-adherence to follow-up.** Remind patients of the importance of review appointments, even if everything seems fine from their perspective. **Referrals and Communication.** A successful glaucoma practice involves effective communication intra-professionally and inter-professionally. Clinicians who are just starting off may find it useful to obtain feedback from their optometry or ophthalmology peers, especially with unusual presentations or complex cases. Establishing a glaucoma clinic requires forging relationships with glaucoma specialists and understanding the services that they may offer. Even the patient’s primary care physician requires regular updates on the patient’s ocular status. Our top tips for referrals and communications within and between professions are shown in Table 2. **Monitoring Visits.** Typically, review periods may range from three months to one year, depending on the individual’s risk of progression. Rubrics for assessing risk or risk calculators (such as from the Ocular Hypertension Treatment Study) are accessible, but remember that individuals do not necessarily fall into the typical mold. Examples of considerations for clinicians in risk titration include:
**Demographics:** Age, ethnicity and gender may not match the clinical trial or guideline that you are deploying.

**Financial constraints:** Are patients able to financially afford frequent review visits?

**Travel/distance constraints:** Are patients able to practically attend appointments during typical work hours?

**Clinic workflow and capacity constraints:** Does the clinic have the capacity to review these patients on a more frequent basis?

**Risk of glaucoma-related consequences:** What is the patient’s risk of vision loss and impairment of quality of life? Where does this stand relative to other factors such as morbidity and mortality?

**Medication prescriptions:** Typically, six-month review periods are recommended in many clinics due to the need for repeat prescriptions—however, can this be done in other ways?

The goal of these follow-up visits is to identify significant disease progression. There needs to be a balance between the anticipated diagnostic yield (more likely to see the “right patient at the right time”) and having potentially wasteful and costly review appointments.

**Follow-up Consultation**

Tests that identify markers of progression should include both structural and functional components, as patients may show progression on either or both indices. Be prepared to take an updated medical history to identify contributory risk factors for glaucoma. These may include new diagnoses of other concurrent systemic disease, new meds and newly discovered family history of glaucoma.

IOP measured at follow-up visits may provide additional data regarding the patient’s IOP profile. Fluctuations or significant elevations may signal the need for closer monitoring or earlier intervention.

Guidelines for repeating gonioscopy or anterior chamber angle assessment also provide a broad range of recommendations. It is fairly well-established that the anterior chamber angle narrows with age, increasing the risk of angle closure. Pseudoxefoliation, the most common identifiable cause of secondary open-angle glaucoma, also has a tendency to increase in prevalence with age. Since it is unlikely that these issues manifest within a short time frame, it would be appropriate to repeat gonioscopy about every two years, with routine screening at visits in between.

Be vigilant for any changes that may signal the need for treatment plan modifications. Examples could include:

**Unreliable VF results at the last visit:** The key is to identify repeatable VF progression at the next visit.

**A disc hemorrhage at the previous visit:** The next visit will specifically target the identification of consequent structural or functional loss.

**IOP was not quite meeting target, but there was no structural or functional progression:** The next visit may be to reassess to see if target pressure is met.

**Progression Analysis**

An advantage of quantitative assessment techniques such as standard automated perimetry and OCT is the ability to quantify changes and perform regression analyses to identify statistically significant progression over time. Although quantitative values are a useful indicator to identify trends, as with all similar measurements there are

---

**Fig. 2.** (A) The current framework used in the management of patients with glaucoma. (B) Our new proposed framework for the management of patients with glaucoma.
sources of variability that may confound the final result. Many clinicians complain about VF testing for being highly subjective and therefore variable. Sources of variability include intra-session fatigue, inter-session learning and the patient’s mental state. However, although OCT may be more objective than perimetry, it also has sources of variability that manifest in the form of artifacts: either instrument-related (internal noise), acquisition-related or patient-related.

Aside from these sources of variability, spacing out tests and monitoring of a patient over a long period of time introduces aging to the progression analysis. The clinician needs to be able to identify a significant change that overcomes the normal age-related decline in both VF sensitivity and neural thickness. One strategy is to use the output rate of change and put it in the context of the patient's age: at this rate of progression, will the patient likely lose significant vision in their lifetime, and when might this happen?

Aside from quantitative analyses of VF sensitivity and retinal thickness data, comparisons of serial fundus photos may also be useful for qualitative identification of structural change. Fundus photography may also reveal the presence or absence of a disc hemorrhage.

Whilst disc hemorrhages have been considered to be a marker for glaucoma progression, disc hemorrhages have been recognized to occur spontaneously or as part of the natural history of glaucoma, not necessarily representing a more aggressive course of the disease. Thus, these patients require more monitoring for identifying realized structural and functional loss.

Remember, structural and functional examination results indicate the endpoints for glaucoma. IOP control is best described as a predictor for risk of future progression. IOP reductions that have not met target, or are in isolation, are not indicators of disease progression, but they must be considered in the context of structure and function. In some cases, over-aggression of IOP control may not be required in a stable glaucoma patient.

### TABLE 2. TOP TIPS FOR REFERRALS AND COMMUNICATION WITHIN AND BETWEEN PROFESSIONS

<table>
<thead>
<tr>
<th>TIP</th>
<th>RECOMMENDATION</th>
<th>EXAMPLE OF EFFECTIVE COMMUNICATION</th>
<th>EXAMPLE OF POOR COMMUNICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Write clearly.</td>
<td>Avoid ambiguity in handwriting. Having proformas with highlighted relevant sections and bolded headings can make it easier to read, with subsequent clinicians being able to target specific information.</td>
<td>Clear headings at the beginning of your letter: “Diagnosis: Glaucoma. Management plan: Initiated therapy with latanoprost both eyes. Review period: Six weeks.”</td>
<td>Buried text somewhere in the middle or bottom of the letter: “The patient has high pressures, so I have initiated therapy with latanoprost BE. I will review in 6/52.”</td>
</tr>
<tr>
<td>Include all relevant investigations.</td>
<td>Sometimes a patient will have long historical records. Have a concise presentation of records for subsequent clinicians. Initially, it may include the progression analysis and the latest cross-sectional data up to that point, but a clinician may request older data sets.</td>
<td>Including the progression analysis of the OCT circumpapillary retinal nerve fiber layer scans, as well as those from today’s visit.</td>
<td>Not including any scan results because printing is expensive, especially with color.</td>
</tr>
<tr>
<td>Referral and communications should be contemporaneous.</td>
<td>Clinicians are busy, but clinical records are best when they are contemporaneous. Communications should be reflective of these investigations.</td>
<td>Sending letters within a few days of the consultation, as well as ensuring that the content is reflective of the day’s results.</td>
<td>Waiting months to send a letter, and forgetting important information because it has been so long since the consultation.</td>
</tr>
<tr>
<td>For referrals, include a comanagement arrangement.</td>
<td>For early-career optometrists, it is sometimes tempting to discharge or be wary about being proactive in communication. To enhance your glaucoma clinic have a proactive approach in developing a plan for patients.</td>
<td>At the bottom of the referral letter: “I have discussed this diagnosis with Patient X and have recommended SLT as a treatment option. I would be happy to review their pressures, discs and fields after they undergo the procedure with you.”</td>
<td>At the bottom of the referral letter: “I have discussed this diagnosis with Patient X and hence I am referring them to you for treatment.”</td>
</tr>
<tr>
<td>Avoid unnecessary abbreviations or jargon for non-ophthalmic practitioners.</td>
<td>The health care team may include non-ophthalmically-trained practitioners. Instead of defaulting to abbreviations that may be eye-specific, it may be more effective to spell out communications where relevant, so there are fewer ambiguities.</td>
<td>“Diagnosis: right primary open-angle glaucoma.” “The right neuroretinal rim was thin, superiorly worse than inferiorly.”</td>
<td>“Dx: POAG RE” “The OD NRR was thin, sup&gt;inf.”</td>
</tr>
<tr>
<td>Promote evidence-based practice in communications.</td>
<td>As the field is rapidly evolving, it may be useful to include references to the latest and/or pertinent literature to demonstrate your attentiveness and adherence to evidence-based practice.</td>
<td>“The results of the recently published ZAP trial suggest that this primary angle-closure suspect can be monitored instead of treated at this stage.”</td>
<td>“I feel that this patient can be monitored at this stage.”</td>
</tr>
</tbody>
</table>
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Feature GLAUCOMA CARE

Similar to diagnosis, progression analysis requires a combinatorial approach to identify the “tipping point” at which a patient converts from a glaucoma suspect to manifest glaucoma, or is identified as a glaucoma progressor. Unfortunately, identification of progression may be as much an art as it is a science. Often, a single test within a suite is insufficient to convince a clinician that a change in treatment plan is required, especially when that test is known to have variable results. Several research groups have suggested that changes occurring in glaucoma may appear in different tests, depending on the stage of the disease.31

Broadly, in early glaucoma OCT has been suggested to be superior to perimetry, and in late glaucoma perimetry, especially 10-2, may be more useful. However, there is an overall lack of consensus regarding what constitutes significant change.

However, if a clinician considers that the goal of glaucoma treatment is to prevent irreversible blindness and impairment to quality of life, there may be a rationale for emphasizing perimetric changes over structural loss. This philosophy stems from the notion that VF results are better reflective of the patient’s functional vision and quality of life, and thus represents the “real-world” impact of the disease.

In the present clinical environment, how might one confidently identify perimetric loss, given its notoriety for variability? Recent developments in automated perimetry have targeted more efficient ways of testing, with strategies such as reduced test time, scotoma targeting and seeding with structural data.32-34 Some of these strategies have arrived in clinical practice, and fast testing methods allow clinicians to perform more examinations per visit, thereby overcoming the variability arising from having fewer data points.

Changing Management Strategy
Once change has been satisfactorily identified using the tools available in your office, you might wonder what the next step for the patient is. Many authoritative guidelines have provided guidance on management algorithms. For example, a typical flowchart approach might begin with either a first-line topical medication or SLT; treatment escalation would be followed by topical medications in a step-wise approach of intensity, before arriving at surgical intervention.

Clinicians need to be aware of the side effect profiles of each treatment paradigm, as the decision to escalate should involve a balancing of the potential threat to vision against detriments to quality of life potentially arising from more intense therapy.

Takeaways
In this two-part article, we have provided an overview of the steps required for the beginner to start a glaucoma clinic. Hopefully, this has demonstrated that whilst there are a lot of considerations in glaucoma, it is also potentially intellectually stimulating. Most importantly, the doctor becomes an integral caregiver for the patient, who in turn can become a long-term, loyal attendee of the practice. ■

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Δ Dr. Kannarr is a paid consultant of Johnson & Johnson Vision.

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‡ Euromonitor International Limited: based on research conducted in August 2020; “world” and “globally” represent markets accounting for 76% of total daily disposable contact lenses in 2019 (retail sales).
Corneal infections move fast—and you should, too. Here’s advice on how to mobilize a defense of the eye that yields swift results.

**Micronetal Keratitis**

Microbial keratitis is a generally painful and sight-threatening condition often associated with ocular trauma, ocular surface disease and contact lens wear.1,2 While the spectrum of pathogens and predisposing factors varies with geography and climate, the condition remains a prominent cause of ocular morbidity.1,3,4 Approximately 30,000 cases occur annually in the US, the preponderance of which are of bacterial origin.5 Fungal and Acanthamoeba-based infections are more rare but can be even more debilitating. Viral corneal infections are predominantly herpetic in origin and more easily recognized clinically.

Traditional first-line therapy of infectious ulcers involves the use of empiric treatment with topical antibiotics.4-8 The role of microbial culture remains perplexing for many practitioners who wonder if they should withhold therapy until results are in, and the use of certain adjunctive therapies, such as topical corticosteroids, is controversial.4,8

**Diagnosing Infectious Keratitis**

When a patient presents with a corneal ulcer, judicious consideration of clinical signs and symptoms is paramount in order to discern the etiology and begin appropriate therapy. Clinical signs associated with bacterial origin may include epithelial defects overlying a single stromal infiltrate, indistinct infiltrate edges, corneal edema with white cell infiltration of nearby stroma, anterior chamber reaction, hypopyon, inferior corneal location, older patient age and—though rarely seen—wreath infiltrates and epithelial plaques.6,9-11

According to a review of 300 bacterial keratitis cases, risk factors for bacterial corneal ulcers include, in order of descending importance: contact lens wear, ocular surface disease, acute corneal trauma and corneal surgery.1 Additional risk factors include systemic diseases, immunosuppression and lack of prior topical antibiotic use.1,12 Gram-positive microbes are predominantly cultured from bacterial ulcers, particularly in instances of ocular surface disease and corneal trauma.1,12 Eye care practitioners, however, identify gram-negative Pseudomonas keratitis more readily, which is often associated with less defined, suppurative infiltrates.1,12 Further defining characteristics of gram-negative keratitis include severe anterior chamber inflammation, greater size of infiltrates and more rapid progression.1

While proper classification of other bacterial keratitis strains has been modest, practitioners accurately discriminate bacterial ulcers from their fungal counterparts 66% of the time.6,12 Clinical features associated with fungal

---

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Ms. Cherny is a 2021 optometry candidate at SUNY and will be completing her ocular disease/contact lens residency at Mass Eye and Ear. They have no financial interests to disclose.
keratitis (AK) relative to bacte-

thamoeba

differentiating features exist for
thamoeba, a greater number of clinically
presence is significantly more indicative
ulcers (as well as herpetic disease), their
note, although ring infiltrates have been

keratitis include the following:6,9,10
• satellite lesions
• infiltrates with feathery, fluffy, ir-
regular or serrated margins
• dry, raised or necrotic infiltrates
• infiltrates pigmented with a color
other than yellow
• endothelial rings
• longer history of symptoms

Compared with bacterial etiology, fungal ulcers show no significant differ-
ence in occurrence of keratic precipi-
tates, immune rings, radial keratoneu-
ritis, endothelial plaques or anterior
chamber reactions.10

Although corneal scrapings rarely
yield parasitic organisms such as Acan-
thamoeba, a greater number of clinically
differentiating features exist for Acan-

thamoeba keratitis (AK) relative to bacte-
rial and fungal etiologies.9 Clinical signs
characteristic of AK include pseudoden-
drites, perineural infiltrates, ring-shaped
infiltrates, a predilection for the corneal
epithelium early in the disease course,
younger age, longer duration of symp-
toms prior to initiation of treatment and
history of prior topical antibiotic use.9 Of
note, although ring infiltrates have been
documented for fungal and bacterial
ulcers (as well as herpetic disease), their
presence is significantly more indicative
of late-presenting Acanthamoeba eti-
ology.9,12 Conversely, satellite lesions—a
feature often noted for fungal kerati-
tis—occur at a similar frequency in both
Acanthamoeba and fungal infections.9

When to Culture

Once an infectious corneal ulcer is
identified, it is imperative to promptly
select an effective treatment in order
to relieve symptoms and ensure best
visual outcomes—and therein lies the
problem. Which one? Matching drug
efficacy to the infectious organism can
yield better outcomes than just choos-
ing a broad-spectrum antibiotic and
hoping for the best.

Studies have shown that clinicians
aren’t as accurate in identifying uncom-
mon organisms or infectious ulcers that
present without the classic character-
istics, with many practitioners dem-
onstrating difficulty in discriminating
fungal from bacterial ulcers solely based
on clinical appearance.10,12 Corneal
specimen culture is the gold standard
for identifying etiological origins of ul-
cers.6,8,12,13 Culturing yields information
that can help modify treatment of ulcers
refractory to empiric therapy, prevent
progression of corneal ulcers, reduce
antibiotic resistance and minimize
medication toxicity from superfluous
medications.8,14 For example, in cases of
AK, proper treatment is often delayed
due to misdiagnosis, and the use of
specimen culture may help avoid a long
and toxic treatment course.9

Although some sources have advo-
cated for culture of all infectious corneal
ulcers, current staining and culturing
methodologies have several limitations,
including low sensitivity in bacterial,
fungal and viral keratitis cases; protract-
ed turnaround time; low bacterial yield
during culture; and time, cost and avail-
ability limitations.7,12,14,15 Furthermore,
in many instances, culture results do not
change clinical management; most cases
of bacterial keratitis resolve with em-
piric broad-spectrum fluoroquinolone
therapy initiated either in the absence of
microbial culture or prior to receipt
culture results.6,7,11,12,26 Given these
limiting factors, a selective approach for
culturing has been suggested.7,11,14

According to the current bacterial
keratitis Preferred Practice Pattern
published by the American Academy of
Ophthalmology, smears and cultures
of infectious corneal ulcers are recom-
manded when any of the following
circumstances are present:11
• size >2mm and centrally located
• accompanied by significant stromal
melting
• unresponsive to empiric antibiotic
therapy
• chronic, multiple or diffuse in num-
ber of infiltrates
• accompanied by characteristics
suggestive of amoebic, mycobacterial or
fungal infection

<table>
<thead>
<tr>
<th>TABLE 1.EXPECTED TRAJECTORY DURING ANTIMICROBIAL THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. aureus, S. pneumonia</td>
</tr>
<tr>
<td>• Rapid improvement after 24 to 48 hours</td>
</tr>
<tr>
<td>• Organisms generally eliminated in 7-10 days</td>
</tr>
</tbody>
</table>
found in eyes with a history of corneal surgery

It is important to note that, although most cases of infectious keratitis are treated empirically, the recovery of organisms via culture may be more difficult to perform if treatment has already been initiated.14

How to Culture

Appropriate culturing technique is critical to maximize yield of organisms (the cornea has a relatively low pathogenic load) and a positive culture. Growth and identification of microorganisms from corneal ulcers may occur in as few as 40% to 60% of cultured cases.6 To increase probability of recovering sufficient microorganisms for a positive culture, multiple samples should be collected and inoculated on various growth media.15

During the procedure, a topical anesthetic is instilled, preferably proparacaine due to its lower bactericidal properties relative to other anesthetics, such as tetracaine.11,14,17 If available, non-preserved topical anesthetic may be used to improve culture yield, and sterile gloves are recommended.11,14,17 The patient should be situated within the slit lamp, as culture is best performed under magnification.11,14,17

A scrape of the corneal ulcer is obtained at the base and advancing edge of the infiltrate from the periphery into the center.11,14,17 Avoid purulent material, as it is less likely to yield sufficient microbial load. However, cultures of contact lenses—cases and solutions—may provide useful information.11,17 Recommended tools for scraping include a 21-gauge needle, a sterilized spud or blade, jeweler’s forceps, a calcium alginate swab or a heat-sterilized Kimura platinum spatula.11,14,17

The material is first smeared onto glass slides if performing gram stain or potassium hydroxide mount, and then inoculated directly onto appropriate agar plates and liquid media (transport media may be used if direct inoculation is not possible).11,14,17 Scrapings should be repeated several times with fresh blades or flame sterilization of the same blade.14,17 Keep cultures at room temperature or incubated if possible, and promptly transport to a laboratory with appropriate labels.11,14,17

Lab reports may come back in as little time as several hours for gram stains, one to two days for bacterial growth or up to two weeks in the case of fungal infections.17 Antimicrobial sensitivity reports may also be obtained from the laboratory.17 Once a culture has been performed and sent out, immediately begin empiric antimicrobial therapy based on clinical signs and history, if you haven’t already.6,17 In the case of a negative culture, cessation of antibiotic therapy for 12 to 24 hours may be considered to increase pathogen yield.11

Treatment of Corneal Ulcers

When initiating treatment of keratitis, corneal infiltrates must first be classified as sterile or infectious in nature.18 Sterile keratitis. These infiltrates are described as minor defects, usually less than 1mm to 1.5mm in size, and are associated with negligible pain, discharge, conjunctival inflammation and anterior chamber reaction, as well as with absent or minimal epithelial involvement.18,19 The majority of sterile infiltrates are subepithelial or anterior stromal and are found in the mid-peripheral to peripheral cornea within approximately 4mm from the limbus.18,19 Sterile infiltrates may be associated with contact lens wear, particularly in cases of extended wear and poor hygiene, and tend to occur in the superior corneal quadrant in the setting of extended contact lens wear.18,19

Initial treatment options for presumed sterile infiltrates include discontinuation of lens wear followed by topical antibiotics, fortified topical antibiotics, antibiotic-steroid combo agents, topical steroids alone or simply observation. Microbial culture is not indicated in cases of sterile infiltrates.18

Topical antibiotic-steroid combinations have been shown to resolve sterile infiltrates in less time than topical antibiotics alone.19 If a distinct epithelial defect develops along with pain and ocular inflammation, a bacterial ulcer may be underlying and requires prompt treatment with antibiotic therapy, with initial avoidance of steroid use.18,19

Infectious keratitis. When you see stromal infiltrates in the presence of epithelial disruption, particularly in cases of bacterial disease, you’re...
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quite likely dealing with an infectious etiology.5,7 Patients with infectious corneal ulcers are typically acutely symptomatic for pain, photophobia, decreased vision, conjunctival injection and mucopurulent discharge.4,7,11 Severe cases may result in stromal thinning, ulceration, corneal perforation, endophthalmitis, vision loss secondary to stromal scarring or even loss of the eye.3,5,11

Prompt empiric treatment is generally necessary when culture and/or sensitivity testing is unavailable or hasn’t yet been performed.3,11 The regimen should consist of topical broad-spectrum antibiotics, such as fluoroquinolones or fortified aminoglycosides, with a cephalosporin or vancomycin.7 Consider geography, and local bacterial prevalence and antibiotic sensitivity, when selecting the antibiotic of choice.3,5

While a large number of clinical trials have been published, there is currently no unanimous treatment regimen detailing which antibiotics should be used for bacterial keratitis.3 Numerous sample regimens have been detailed, and are subdivided based on microorganism characteristics (see Table 1).3 Presently, ciprofloxacin 0.3%, levofloxacin 1.5% and ofloxacin 0.3% are FDA-approved to treat bacterial keratitis.11

In cases of large or sight-threatening ulcers, particularly if accompanied by a hypopyon or deep stromal involvement, fortified topical antibiotics are beneficial, and a loading dose every five to 15 minutes followed by hourly application is recommended.11 Subconjunctival injection, systemic therapy or hospitalization may be necessary in severe cases.3,11 Adjuvant cycloplegics also may be considered in instances of significant anterior chamber reaction and/or severe pain due to their palliative and anti-inflammatory properties.11

Steroid Therapy

While topical antibiotic therapy remains the mainstay treatment of bacterial keratitis, adjunctive corticosteroid use may also prove beneficial for clinical outcomes, although use of topical steroids for microbial keratitis remains controversial.4,5,8 Proponents contend that steroids mitigate tissue damage from the host inflammatory response by decreasing severity of stromal melting, neovascularization and scarring.4,5,9 Furthermore, steroids may improve patient compliance with antibiotic treatment by alleviating pain and discomfort.3 Conversely, steroid therapy may delay epithelial healing and potentiate bacterial keratitis, leading to stromal thinning and melting.4,5,20

Four clinical trials, including one randomized, placebo-controlled, double-masked trial known as SCUT, have compared clinical outcomes in bacterial keratitis treated with antibiotics and steroids vs. antibiotics alone.5,21 While the first three smaller trials evidenced neither harm nor benefit associated with topical steroid use, subgroup analysis within SCUT found that patients with low vision, or deep or central ulcers, at baseline experienced better visual improvement at three months when compared with placebo, as did patients with invasive Pseudomonas strains.5,21 No significant difference in adverse effects was noted between steroid and placebo arms.3,20,21

Timing and dosage are important concerns, as patients who began steroids after only two to three days of antibiotic use, particularly with potent agents administered six times daily, experienced better visual outcomes than those in the placebo arm. Patients with later steroid use experienced neutral or worse acuity vs. placebo.5,20,22

A 12-month follow-up of the SCUT trial also demonstrated superior visual outcomes in patients with ulcers not caused by Nocardia keratitis (NK).23 Patients with NK, however, have been found to experience larger infiltrates and scars with corticosteroid therapy, despite no difference in vision, re-epithelialization or corneal perforation. Poor outcomes with steroid use have also been associated with fungal and Acanthamoeba infections.22,24

To minimize adverse effects, it is prudent to administer topical steroids after 24 to 48 hours of antibiotic therapy, following evidence of ulcer improvement and microorganism identification via culture.4,5,11 Avoidance of topical steroids in cases of atypical keratitis, as well as in NK, fungal and Acanthamoeba etiologies, is critical.3,11

Adjuvant Therapy

In addition to traditional antibiotic therapy, several adjuvant options for bacterial keratitis have been described. A randomized, controlled trial demonstrated that low-concentration topical povidone-iodine 1.25% may perform with equal efficacy as topical antibiotics in the treatment of bacterial keratitis, and has the added benefit of considerably lower cost.25 Collagen crosslinking, a procedure which treats corneal ectatic conditions by strengthening stromal tissue, has been shown to be of potential benefit for recalcitrant infectious keratitis by halting corneal melting, resolving ulcers resistant to treatment and improving patient symptoms.8
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The role of tetracyclines such as doxycycline has been suggested due to their antimalleoproteinase properties, which may lessen the risk of severe complications of infectious keratitis, such as corneal perforation. Other adjunctive therapies proposed for bacterial keratitis include amniotic membranes, antibiotic-soaked soft contact lenses, collagen shields, mitomycin-C, hyperbaric oxygen therapy, autologous serum eye drops and cryotherapy, although the beneficial effects of these therapies are not universally established. When to Change Therapy

Monitoring clinical response to treatment is critical to success—we need to establish if the therapy is working and whether modification is necessary. Lack of clinical response or worsening of signs and symptoms within 48 hours, particularly in cases of vision-threatening ulcers, may warrant initiating a more aggressive antibiotic regimen (e.g., switching to fortified broad-spectrum antibiotics if initial treatment consisted of fluoroquinolone therapy), considering less common microorganisms, performing a re-culture or referring to a cornea specialist.

In cases of unlikely compliance, disease extension into adjacent tissues or threat of corneal perforation, systemic antibiotics or hospitalization may be necessary. Also consider that drug toxicity may be mistaken for lack of clinical improvement, and reduction of therapy may be warranted at times in order to assess healing. Bacterial keratitis remains a prominent cause of vision loss worldwide.

Precise clinical identification of underlying etiology is critical for effective treatment, and may be aided by proper microbial culture. Although antibiotic therapy remains first-line for bacterial ulcers, adjuvant treatments have been described, and corticosteroid therapy may play an essential role in improved visual outcomes.

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CORNEOSCLERAL CONCERNS: TROUBLE AT THE BORDER

The limbal junction is key in protecting and preserving the ocular surface. Here are a few conditions that can disrupt its equilibrium.

The corneoscleral limbus is a unique and critical ocular structure responsible for barrier protection, corneal regeneration and wound repair. These functions are pivotal in maintaining corneal transparency and ocular surface integrity. In addition, the limbus contains vascular and lymphatic vessels that serve to provide nutrients and cytokines to the surrounding structures. This makes the peripheral cornea more susceptible to immune-mediated inflammation and ensuing structural changes.

The corneal epithelium is in a constant state of remodeling. The layer of surface cells, wing cells and basal cells. Surface cells are made up of non-keratinized, stratified squamous cells. The conjunctival epithelium, in contrast, is made up of non-keratinized, stratified columnar cells with mucin-containing goblet cells. As surface corneal epithelial cells are shed, the basal lamina encourages new cell proliferation. Only cells in contact with the basal layer of the epithelium can divide.

The corneal epithelium is thought to repair itself in a centripetal fashion. Differentiation and replenishment of the epithelium from basal cells to surface cells takes approximately seven to 14 days to complete. The limbus marks the junction where the corneal epithelium meets the conjunctival epithelium. In the basal lamina at the limbus, stem cells can be found in the palisades of Vogt. These are critical for cell proliferation and migration following an insult to the corneal epithelium. Limbal stem cells also provide a barrier to conjunctival epithelial cells, keeping them from migrating onto the cornea. When limbal stem cells are damaged, the cornea can become “conjunctivalized.” Conjunctivalization is evident through neovascularization, the presence of goblet cells and an unstable epithelium. This transformation of cell tissue can lead to chronic inflammation, neovascularization, persistent epithelial defects and possible opacification.

The cornea has angiogenic and immune privilege, both of which are necessary to retain transparency. The process of maintaining corneal avascularity is ongoing. When the cornea is injured, anti-angiogenic factors are upregulated, while pro-angiogenic factors are downregulated. This equilibrium ensures no corneal neovascularization ensues. When the limbal tissue is compromised, balance is disrupted and angiogenic factors prevail, leading to new blood vessel growth.

This article highlights several limbal diseases, to which we should pay particularly close attention.

Deficiency Processes
Disease or trauma can bring on a state of limbal stem cell deficiency (LSCD). This condition results from dysfunction, damage or destruction of limbal stem cells. Destruction can be caused by chemical or thermal burns, Stevens-Johnson syndrome, multiple ocular surgeries, contact lens (CL) wear or severe microbial keratitis. Gradual loss of stem cell function may be due to a variety of genetic or acquired conditions. Genetic causes of LSCD include aniridia, congenital erythrokeratodermia and multiple endocrine deficiency. Acquired gradual loss can be stimulated by neurotrophic keratitis, vernal conjunctivitis or peripheral inflammatory keratitis, to name a few.

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When corneal conjunctivalization and persistent epithelial defects are present, consider LSCD. Fluorescein pooling is often evident in areas of conjunctivalization due to thin, irregular tissue with a loss of tight junctions. This staining can take on a whorl-like pattern and be sectoral or more widespread depending on the severity of LSCD. Fibrovascular pannus, scarring and calcification are also often observed. LSCD patients are highly susceptible to epithelial erosions and report chronic pain, blurred vision and photophobia. In advanced disease, corneal melt or perforation is possible.

Topical steroids are considered the first-line therapy in early stages of LSCD. Additionally, frequent administration of preservative-free artificial tears is recommended to treat underlying ocular surface disease. Restasis (cyclosporine, Allergan) or Xiidra (lifitegrast, Novartis) can help decrease inflammation. A bandage lens, scleral lens or amniotic membrane can promote epithelial healing. In cases where some limbal stem cells are still functional, debride the abnormal epithelium. This allows the remaining functional stem cells to repopulate the surface with normal corneal epithelium.

In extensive or complete LSCD, limbal stem cell transplantation (LSCT) is often required, as these patients do not typically respond well to traditional keratoplasty. To improve surgical outcomes, control inflammation prior to transplantation. In unilateral LSCD, autologous limbal tissue can be transplanted from the fellow eye. There is no risk of rejection in an autologous transplant, leading to a higher success rate. Bilateral LSCD, which most commonly occurs secondary to burns, requires allogenic transplantation or cultivated oral mucosal transplantation. Amniotic membranes are used to transplant tissue onto the host cornea. The risk of rejection in allogenic LSCT is a significant concern, necessitating the use of HLA typing and long-term immunosuppression.

Common adverse effects of LSCT include recurrent or persistent epithelial erosions and increased intraocular pressure. Administer the same therapies used in early disease post-transplantation to reduce inflammation, suppress the immune system and optimize the ocular surface.

Mechanical Processes
The ocular surface is continuously buffeted by internal and external forces and can succumb when overtaxed.

CL-mediated issues. Lens wear can lead to limbal dysfunction via mechanical wear, decreased tear exchange and corneal hypoxia. Additionally, injury is possible with improper lens insertion or removal techniques. These factors open the door to possible microbial infection and progressive compromise to the limbal tissue. CL wear and care accounts for 15% of all LSCD cases.

Monitor the superior cornea carefully for staining due to increased friction between the superior lid and lens. Also monitor the periphery for neovascularization, which indicates hypoxia. Ask the patient heed their replacement schedule and consider reduced wear time and/or daily disposables to keep the risk of CL-related limbal complications low. In the case of lens-associated LSCD, discontinue lens wear indefinitely.
**Superior limbic keratoconjunctivitis (SLK).** This is thought to be caused by a poor interaction between the superior eyelid and the superior bulbar conjunctiva.\(^{18-20}\) It may also be influenced by a localized tear deficiency.\(^{20}\) SLK is much more common in females and may be accompanied by concurrent thyroid disease in up to 50% of cases.\(^{21}\)

SLK is marked by superior bulbar injection, especially near the limbus, with thickening or redundancy of the conjunctival tissue. Fine papillae can be observed on the superior tarsus, and punctate erosions can be seen on the superior cornea, limbus and bulbar conjunctiva. Superior filaments often develop, and dry eye disease is a common comorbidity. Patients with SLK often present with conjunctival injection, such as superior lid swelling, blepharoconjunctivitis.

Initial therapy may include frequent lubrication, punctal occlusion and a therapeutic soft CL. Also consider autologous serum, cyclosporine A and topical mast cell stabilizers. SLK responds poorly to topical steroids.\(^{18}\) More aggressive treatments include 0.5% to 1% silver nitrate application to the conjunctiva following a topical anesthetic, which chemically debrides the inflamed conjunctival tissue. Take extreme caution to avoid burning the cornea. Additionally, conjunctival resection or thermal cautery of the superior bulbar conjunctiva can improve the conjunctival interface.\(^{21}\)

**Dellen.** This is a painless area of thinning caused by ocular surface dryness. It often develops adjacent to elevated areas. It can also occur near conjunctival chemosis, episcleritis, pingueculae or pterygia. The presenting location is often temporal near the limbus.

Dellen commonly present following cataract surgery, strabismic surgery or glaucoma-filtering surgery.\(^{21}\) Patients may report mild discomfort or foreign body sensation. Look for a saucer-like depression at the slit lamp. Corneal dellen represent thinning of the epithelium, Bowman’s layer and the anterior stroma. They typically resolve within a few days but in some cases may last weeks.\(^{23}\) Treatment consists of frequent lubrication, patching if necessary and possibly a prophylactic antibiotic ointment. In more resistant cases, reducing the adjacent elevation may be required.

**Immune-mediated Processes**

Aberrations in the immune response can affect the limbal region.

**Phlyctenulosis.** Phlyctenular keratoconjunctivitis is an immune-mediated condition in which the cornea becomes sensitized to a microbial antigen during a delayed hypersensitivity reaction. With repeated exposure to this antigen, phlyctenules can develop on the corneal or conjunctival side of the limbus. *Staphylococcus* is the most common stimulus; however, historically, phlyctenulosis commonly occurred in response to tuberculoprotein.\(^{24}\) As antigens infiltrate the surface, exotoxins are released, causing the corneal epithelium to break down.\(^{25}\) This condition is more common in teenagers and has a higher predilection for females.\(^{24}\)

A phlyctenule is a 1mm to 2mm fleshy white nodule often accompanied by conjunctival injection. Symptoms include foreign body sensation, tearing, increased light sensitivity and possible itching. Phlyctenules on the cornea increase the severity of symptoms and have the ability to ulcerate, causing scarring and neovascularization. Corneal ulceration frequently results in a triangular-shaped anterior stromal scar. Phlyctenules can be recurrent and often occur at the edge of previous neovascularization sites.\(^{24}\)

Some cases of phlyctenular keratoconjunctivitis are self-limiting, but topical steroids dosed QID are the treatment of choice. If a corneal epithelial defect is present, a broad-spectrum antibiotic is recommended prior to steroid initiation. Due to the risk of recurrence, it is critical to treat concurrent blepharitis. This can be achieved with lid hygiene, antibiotic ointment or even a course of oral doxycycline (if the patient is older than eight and not nursing or pregnant).

Differentials of phlyctenulosis include Salzmann’s nodules, Horner-Trantas dots in vernal keratoconjunctivitis, pingueculitis and nodular episcleritis. In episcleritis and pingueculitis, the nodules do not ulcerate. Corneal phlyctenules often resemble infectious ulcers, so it is important to rule out microbial keratitis. If suspicions of an infectious etiology arise, culturing should be performed.

**Staph marginal keratitis.** This com-
mon immune-mediated limbal disorder is considered a type III hypersensitivity reaction to resident *Staphylococcus aureus*, which is often present on the lids and lashes. It occurs peripherally due to its close proximity to limbal lymphatic vessels. Symptoms include pain, light sensitivity, foreign body sensation and conjunctival injection.

The condition is marked by one or often multiple small white subepithelial infiltrates 1mm to 2mm from the limbus, with a clear cornea in between. These infiltrates tend to be located in areas where the lid interacts with the corneal tissue. Ulceration is possible and indicated by sodium fluorescein staining over an infiltrate. Neovascularization can ensue in cases of ongoing inflammation.

It is treated similarly to phlyctenulosis, as it responds quickly to topical steroids. If an epithelial defect is present, add a broad-spectrum antibiotic. Additionally, lid hygiene and possibly topical or oral antibiotics are critical to reduce the likelihood of recurrence.

**Mooren’s ulcer.** This rare, painful peripheral corneal ulceration is marked by a wavy pattern. It is associated with non-perfusion of the superficial vascular plexus. Mooren ulcers are idiopathic in etiology but thought to be immune-mediated. There are three known forms:

- **Type 1,** a unilateral Mooren, is a painful, progressive form found in patients older than 60.
- **Type 2,** a bilateral, aggressive Mooren, occurs in younger patients and progresses circumferentially.
- **Type 3,** a bilateral, indolent Mooren, is marked by slow, progressive peripheral corneal guttering in middle-aged patients.

Behind the slit lamp, a swollen gray area of cornea that tends to furrow rapidly can be visualized. An epithelial defect and stromal thinning can also be observed. In Types 1 and 2, limbal inflammation is significant and marked by swelling and neovascularization. The ulceration often starts focally at the nasal or temporal limbus and spreads circumferentially and centrally.

Therapy depends on the severity and type of ulceration. Start with an aggressive topical steroid course and a prophylactic antibiotic. Use topical cyclosporine therapy QID as adjunctive therapy. In any form of epithelial defect, frequent lubrication is recommended to reduce eyelid friction and inflammatory cytokines.

Type 2 often requires IV immunosuppression with methylprednisolone followed by oral steroids. In any form, the goal of treatment is re-epithelialization and decreased inflammation. Shallow ulceration can be repaired with amniotic membrane transplantation, conjunctival resection or a conjunctival flap. For deeper ulcers, partial or total lamellar keratoplasty may be required.

**Vernal keratoconjunctivitis (VKC).** This severe, bilateral and chronic allergic condition is most prominent in young boys living in warmer climates. Common comorbidities include asthma and allergic rhinitis. VKC is thought to be an IgE-mediated hypersensitivity, but IgG, basophil and cellular delayed-type hypersensitivities may also be involved.

Patients usually report severe itching, photophobia, thick mucus discharge, tearing, burning, foreign body sensation, pain and possibly blurred vision. Symptoms tend to be most common in the spring. On slit lamp exam, giant cobblestone papillae can be observed on the superior palpebral conjunctiva. In addition, focal white infiltrates (Horner-Trantas dots) and sectoral conjunctival and episcleral hyperemia can be seen at the superior limbus. Corneal shield ulcers are also a possible, yet uncommon, clinical manifestation. They are sterile in nature and result from mechanical rubbing of cobblestone papillae on the cornea.
First-line treatment includes topical antihistamines and mast cell stabilizers. Topical steroids can also be considered, especially in more severe cases. In addition, calcineurin inhibitors, NSAIDs and oral antihistamines can be employed. Avoid triggers such as eye rubbing, wind, heat and sunlight. Artificial tears and cool compresses can provide some supportive relief. In the case of corneal shield ulcers, recommend a broad-spectrum antibiotic QID until the epithelium heals.34

Kids often outgrow VKC with time, but it can take years to run its course. Vision loss is possible secondary to corneal neovascularization and subsequent scarring, but this outcome is rare. Peripheral ulcerative keratitis (PUK). This is a broad diagnostic term used to describe peripheral corneal thinning caused by a variety of collagen vascular conditions. These autoimmune conditions include rheumatoid arthritis, polyarteritis nodosa, Wegener’s granulomatosis, inflammatory bowel disease and systemic lupus erythematosus, to name a few. Rheumatoid arthritis is by far the most common of these etiologies to cause PUK. PUK is a unilateral condition consisting of an epithelial defect, crescent-shaped stromal inflammation with thinning and accompanying episcleritis or necrotising scleritis.35 In advanced disease, perforation can ensue, which is why prompt diagnosis and treatment is critical. PUK is the initial symptom of collagen vascular disease in 50% of cases.36

If PUK is observed in a patient with no known collagen vascular disease, thorough personal and family history and lab testing is recommended. A typical lab workup includes complete blood count with differentials, erythrocyte sedimentation rate, rheumatoid factor, antinuclear antibody, antineutrophil cytoplasmic antibodies, chest X-ray examination and liver enzymes. If associated collagen vascular disease is detected, direct therapy at managing the systemic condition with the help of a rheumatologist. Topical steroids can be used in early disease, but often systemic immunosuppressive agents are required. First-line therapy involves systemic corticosteroids and often a cytotoxic agent.35 Common immunosuppressants include cyclophosphamide, methotrexate, azathioprine and oral cyclosporine.37 Biologics can also be considered. In addition, frequent lubrication with preservative-free tears and possibly a bandage CL can improve the corneal microenvironment, as there is a high rate of concurrent dry eye disease in these patients. In more severe disease, surgical intervention may be required. Surgical techniques include tissue adhesive, lamellar graft, tectonic corneal grafting and amniotic membrane transplantation.35

Neoplastic/Deposition Processes Accumulations of tissue or other material form yet another threat to limbal health and function.

Pterygia. These are marked by fibrovascular tissue extending onto the cornea, caused primarily by UV exposure. The fibers are thought to develop from damaged fibroblasts. Pterygia destroy Bowman’s layer, and an iron line can often be observed at the leading edge, which consists of a flat gray zone.22 Pterygia are most common nasally. Symptoms include foreign body sensation, irritation and photophobia. These growths can induce irregular astigmatism, leading to a decrease in best-corrected visual acuity, especially as they encroach on the visual axis. Frequent lubrication is recommended for symptomatic pterygia, and topical NSAIDs or steroids can be used in cases of active inflammation. Perform surgical excision if the line of sight becomes threatened. Band keratopathy, caused by deposition of calcium in Bowman’s, is often stimulated by chronic uveitis or hypercalcemic conditions such as chronic kidney failure. Band keratopathy beginsipherally on the nasal or temporal side and is marked by a gray opacity that can become white and chalky. The edge of the opacity is often separated from the limbus by a lucent zone. This is thought to be caused by an absent Bowman’s layer or inability of limbal vessels to prevent calcium deposition.22
Band keratopathy can break through the epithelium in later stages of disease. Also in advanced presentations, calcific deposition can extend horizontally across the cornea from limbus to limbus. Additionally, hyaline material and fibrous neovascularization can surround the calcified lesion. Thankfully, these lesions progress slowly.

If the etiology is unknown, lab testing should include serum calcium, phosphorus, uric acid and renal function. Hyperparathyroid and sarcoid testing with PTH and ACE, respectively, should also be considered. Also, ask patients about vitamin and calcium supplement intake.

Band keratopathy is asymptomatic in its early stages, but patients can experience decreased acuity, foreign body sensation, tearing and light sensitivity in more advanced disease. Early stages only require management of the underlying etiology. Surgical intervention is indicated if the deposit is encroaching on the line of sight or if the deposits are causing surface discomfort. First-line surgical therapy is epithelial debridement followed by the administration of EDTA, a chelating agent. Phototherapeutic keratotomy is also effective. Amniotic membranes can be used following deposit removal to speed up the rate of healing.22

Degenerative Processes
Breakdown of corneal structures typically manifests slowly and subtly. Let’s look at two affecting the limbus.

Terrien’s marginal degeneration.
This rare, asymptomatic and bilateral peripheral degeneration is idiopathic in nature and progresses slowly. It occurs most commonly in 20- to 40-year-old men and presents with a largely white and quiet eye. The degeneration begins superonasally with superficial neovascularization, punctate opacities and a gutter between the opacities and the limbus.22 The epithelium remains intact but Bowman’s and Descemet’s are disrupted. Over the course of years, the stroma continues to thin. As it does, aqueous pockets and possibly a yellow-white lipid zone can be observed.

There are two forms of this degeneration: a quiet one seen in older patients, which yields little to no symptoms, and an inflammatory form in younger patients.22 The latter is often accompanied by episodes of episcleritis or scleritis, which can be treated with steroids.22 Many cases don’t require treatment, but in the rare event of perforation, consider lamellar or eccentric grafts.22

Pellucid marginal degeneration. This painless condition with no observable inflammation is marked by bilateral inferior corneal thinning. It often causes high amounts of irregular or against-the-rule astigmatism. It tends to progress slowly and can be diagnosed with corneal topography, which commonly reveals a “kissing birds” or “crab claw” pattern. Specialty CLs such as scleral may be indicated for optimal visual acuity.

Takeaways
The corneoscleral limbus is crucial for barrier protection, prompt corneal healing and corneal transparency maintenance. The limbus aids in fighting off infection, injury and inflammation. Maintain integrity by preserving limbal stem cells, keeping conjunctival tissue at bay and ensuring a hydrated, stable corneal surface. Restoring healthy limbal function includes adequately lubricating the ocular surface, minimizing inflammatory responses and avoiding mechanical damage. In cases of severe limbal compromise, consider amniotic membranes, grafts or LSCT to restore corneal structure and function.

It is unusual for a treatment algorithm to be dramatically re-written for ophthalmic pathology. This occurred in the early 2000s with anti-VEGF medications taking over as the treatment of choice for wet macular degeneration and subsequently most retinal vascular issues. At the same time, posterior lamellar transplants began replacing penetrating keratoplasty (PK) as the surgical treatment of choice for all forms of endothelial decompensation. We may currently be in the initial stages of another such shift in the clinical management of corneal endothelial disease with the use of rho kinase (ROCK) inhibitors.

A relatively new ophthalmic class, ROCK inhibitors have been around for a few years in the form of Rhopressa (netarsudil, Aerie) and Rocklatan (netarsudil/latanoprost, Aerie) in the field of glaucoma. The revelation that a glaucoma med might be repurposed for an altogether different condition involving an unrelated ocular structure could, if validated by large-scale studies, emerge as one of this decade’s biggest success stories.

**Grow Your Own**

The mechanism of ROCK inhibitors for IOP control has to do with the molecules’ influence on cytoskeleton and intracellular adhesions. These cytostructural changes result in a decrease in resistance to aqueous outflow at the point of the trabecular meshwork. Beyond the specific influence of ROCK inhibition, Rhopressa also targets norepinephrine transport, which secondarily reduces production of aqueous and reduced IOP.1,2

The two netarsudil-containing medications offer a nice, once-daily adjunct to the medical management of glaucoma and, depending on out-of-pocket patient expense, are near the front of the therapeutic options here. However, evidence is continuing to mount that the role of ROCK inhibitors in the treatment of endothelial-based corneal edema could be even more profound.

Given that the corneal endothelium is arrested in the cell cycle and therefore not mitotic, any process that damages these cells can only be treated via transplanting new ones into the eye. Currently, the standard practice with any form of endothelial decompensation resulting in corneal edema—regardless of specific etiology—is essentially a period of “handholding” until the patient is so bothered by reduced vision that they require surgical intervention.

Though the threshold to consider transplant surgery for endothelial decompensation has dropped significantly since the advent and widespread adoption of lamellar procedures like Descemet’s stripping automated endothelial keratoplasty (DSAEK) and Descemet’s membrane endothelial keratoplasty (DMEK), these techniques still present significant hardships for patients. These may include:

- potential long travel to a surgery center for those who do not live near a surgeon
- the need for the patient to remain in a supine position the first few days postoperatively
- the possibility of graft detachment and need for a subsequent re-bubble
ongoing risk of transplant rejection
• the potential sequelae of topical corticosteroid use needed to reduce risk of rejection
• failure of the graft over time and the need to re-transplant, as these grafts have a finite life expectancy

The ability to avoid or at least postpone these transplant-based issues led to the development of the Descemet’s stripping-only (DSO) procedure, also known as Descemetorhexis without endothelial keratoplasty (DWEK). In DSO, patients with substantial central and localized Fuchs’ dystrophy have the central zone of endothelium and Descemet’s membrane removed and then are left to allow their normal peripheral endothelial cells to migrate in and fill this zone without the light-scattering effects of guttata.

While DSO avoids many of the issues that develop with transplants, it is a niche surgery and only reasonable for a select population of patients with endothelial disease. In reality, it is likely more of a temporizing measure, as removing diseased endothelium does nothing to avoid long-term decompensation. Further, issues such as inducement of irregular astigmatism, deep scarring and non-clearing corneal edema, may also develop.

Enter ROCK inhibitors. The mechanism of ROCK inhibition on cellular and intracellular adhesions is not limited to the trabecular meshwork. Within the cornea, these molecules seem to promote migration and spread of corneal endothelium, which perhaps enhances the normal spread of endothelium following injury and localized cellular loss. It also prevents cellular apoptosis, a laudable effect that is critical for preserving existing endothelium. This can be initiated in Fuchs’ dystrophy secondary to the physical strain placed on the cell walls of endothelium adjacent to guttata.

Most exciting of all, there is some evidence that ROCK inhibitors may promote endothelial proliferation, which given the non-mitotic status of these cells, has enormous clinical potential. If ROCK inhibition truly does promote controlled proliferation of endothelium, the algorithm for the management of endothelium-mediated corneal edema may be re-written. These agents can also serve as important components of effective cultivation methods to propagate endothelial cells for use in cell-based therapies.

To date, the use of ROCK inhibition in corneal disease has been more complementary than revolutionary. Its first broad use in corneal disease was as adjunctive therapy to speed and facilitate recovery within DSO patients. More recently, as clinicians have become more comfortable with the use of these agents for corneal disease, case reports of ROCK inhibition prior to corneal surgery have been published. One small series in particular demonstrated the effect of ROCK inhibition in corneal edema secondary to a broad range of pathologies. The author of this series was quick to point out that not all patients with corneal edema will respond positively to ROCK inhibition, and even includes a case of treatment failure to illustrate the point—it can be impossible to predict at this point who will and won’t respond. Notably, this case series only had a treatment duration of a month, and its effect, when successful, was maintained even after cessation of netarsudil.

All that said, the use of ROCK inhibitors for corneal endothelial disease is a subfield in its infancy. It is possible further research will show less efficacy than hoped for and its use may remain a purely secondary therapy—promoting both graft survival or recovery from DSO. However, it’s also possible that as we become better at predicting...
the effectiveness of the agents or we develop more effective and targeted agents, ROCK inhibition may periodically or even widely replace corneal transplantation. Regardless, we are standing on the precipice in the management of corneal endothelial disease. Time will tell where we go from here, but for now, it’s worth keeping a close eye on the research.

A New Clinical Entity

Regardless of how far the application of ROCK inhibitors goes in corneal endothelial disease, we are left with one lasting effect of the drug: the potential of these meds to produce a totally new clinical manifestation called honeycomb corneal edema, alternately known as both macrocystic epithelial edema and reticular bullous epithelial edema.

Honeycomb edema is characterized by profoundly diffused but clearly delineated “cells” of epithelial edema that makes it wildly different in appearance from typical microcystic epithelial edema. This fascinating entity is related entirely to the use of ROCK inhibition in eyes with stromal edema or compromised endothelial health and is extremely common among that narrow population, but should not occur in eyes with healthy corneas using the medication for glaucoma.

Its mechanism likely has nothing to do with the endothelial effects of the drug—as our own experience with the medication suggests—where we have seen eyes develop honeycomb edema despite significant reduction in corneal thickness. Rather, honeycomb edema is probably a manifestation of the ROCK inhibitor–induced changes in how the epithelium responds to the strain of corneal edema through alterations in its intraacellular adhesions.

It has been well documented that ROCK inhibitors increase the permeability of tight junctions. Tight junctions in the corneal epithelium promote maintenance of transparency by impeding passage of sodium ions and subsequent fluid into epithelial cells. Perhaps the honeycomb-shaped epithelial edema phenomenon is a result of ROCK inhibitors weakening corneal epithelial tight junctions, leading to an increased passage of fluid into these squamous, polygonal-shaped basal and surface epithelial cells in the setting of stromal edema—resulting in a clinical appearance resembling a honeycomb.

However, this is all still a theory and speculative, as cellular studies need to be conducted. According to the review cited earlier, all patients with stromal edema on ROCK-inhibitor therapy will develop honeycomb edema, and importantly, its development does not portend long-term treatment success or failure. In eyes that respond positively to the drug, the honeycomb edema will fade over time. In those that fail to respond to ROCK inhibition, edema will persist until the medication is discontinued at which point it will clear.2 8

Watch this Space

Though much more robust research is required prior to declaring ROCK inhibitors a front-line therapy in the clinical management of corneal edema, their use does hold promise even though this field is in its infancy. It’s also not every day a brand new clinical entity manifests, which is what ROCK inhibitors have created with honeycomb epithelial edema in patients with pre-existing stromal edema. While much can change with further research, its apparent we should all keep a close eye on the research with ROCK inhibition in corneal edema and make ourselves familiar with the complication, however benign, of its use in this population—honeycomb epithelial edema.

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The many layers of cornea comanagement

The practice of medicine is a collaborative effort and optometry is no exception. As we discussed in the first installment of this series, comanagement plays a critical role in healthcare delivery and, as primary eyecare providers, ODs should be at the helm of the referral relationship.

While comanaging cornea cases is not as commonplace as with cataract patients, it remains an important relationship that every optometrist should have in their network. Understanding how to leverage collaborative efforts is beneficial for both patients and practice.

First and foremost is the convenience for patients. In many cases, the best cornea specialist is not always the closest. Comanagement ensures patients receive specialized care while also having the support of their primary optometrist, eliminating the need for unnecessary travel.

“Comanagement allows patients to obtain the most specialized care while maintaining a meaningful relationship with their local optometrist, who has fostered their care for years,” notes Katelyn Lucas, OD, of Price Vision Group, a large cornea specialty practice in Indianapolis. “It also allows the optometrist to see the cornea in the early phase so that they can better determine if issues arise such as rejection, wound dehiscence, increased inflammation or other problems that can occur over time.

“With cornea surgeries, especially transplants, lack of follow-up can lead to loss of graft and even permanent vision loss due to steroid induced glaucoma, immunologic rejections or infections,” she continues. “Getting in for a timely exam when problems arise is critical in taking care of corneal transplants, and having a doctor convenient to their location allows for prompt medical attention.”

This second installment of our comanagement series will delve into cornea management and ways to help ODs enhance their referral relationships. We will take a closer look at the role of optometry in closing the practice gaps that often exist in this category. Comanagement is not only a way to provide comprehensive patient care; when done successfully, it also can elevate individual practices and the profession as a whole.

Optimizing Cornea Management

Cornea care encompasses a host of conditions and therapeutic interventions; therefore, comanagement can vary greatly depending on the patient’s individual needs. However, no matter the procedure, optimal patient outcomes depend on effective collaboration.

“Surface procedures, such as phototherapeutic keratectomy (PTK), superficial keratectomy and corneal crosslinking (CXL), lend themselves well to comanagement between optometry and ophthalmology,” notes Mitch Ibach, OD, of Vance Thompson Vision, a large anterior segment surgical practice in Sioux Falls, SD. “Patients are still going to need some type of vision rehabilitation afterwards and optometrists are in the position to take the lead following surgery.

Intraocular corneal surgeries (or, more broadly, transplantations) do not follow the path of traditional comanagement, he explains. “The majority of early post-op management—the first three months or more—occurs at the surgical center due to the delicate nature of the follow-up care. The
primary referring OD often takes over post-op care in most cases later than in other, less invasive procedures.”

When a corneal transplant patient returns to their optometrist can vary and usually depends on how the surgical center handles postoperative care. However, the optometrist’s work begins well before surgery. Preparing the ocular surface for corneal surgery is an important preoperative step in maximizing healing, notes Dr. Ibach.

“Careful documentation of preoperative acuities is essential as well as a detailed assessment of the corneal condition,” says Joseph Shovlin, OD, also at a large multi-specialty practice with emphasis on anterior segment care. “Optometrists should report any other comorbidities or potential problems.

“A lot of these patients will have secondary issues, such as glaucoma,” he explains. “Some patients have lid disease that may be the driver for the corneal problem; therefore, careful documentation of the cornea and adnexa is key.”

One of the most important responsibilities of the OD is managing patient expectations. “You don’t want someone going to a cornea specialist expecting a 20/20 outcome when that might not be possible,” notes Dr. Shovlin. “Given their longstanding relationship, patients trust their optometrists to prepare them for any procedure, but this is especially important for an intimidating surgery like corneal transplant.”

While the surgical center will be more involved in postoperative care for corneal transplants and other invasive procedures, optometrists have an important role to play and should be included in all aspects of care.

“All corneal transplants need to be followed on a regular basis to monitor for rejection and steroid response which can be done with the comanaging optometrist,” says Dr. Lucas. “This helps ensure the success of optometry practices and improves the doctor-patient bond.

“Early post-op visits done with an optometrist also allows for a ‘second objective opinion,’” she notes. “A good co-managing surgeon values this as they can be unconsciously biased from seeing a complication in their work.”

Infection is always a concern postoperatively, notes Dr. Shovlin. “Depending on when you see the patient, you always have to make sure that the cornea is compact, and be sure to look for signs of early infection and rejection,” he says. “As time goes on, graft failure is a concern and it’s very important to monitor intraocular pressures as well as monitor for lens changes with needed prolonged steroid use.”

Dr. Lucas recommends reviewing the ophthalmology practice’s comanagement packet, which typically contains instructions for post-op visit frequency, necessary testing and medication changes. Familiarizing yourself with medication schedules and tapers from the surgical center helps improve consistency and outcomes, emphasizes Dr. Ibach.

No matter the type of procedure, communication between providers is essential. “Effective comanagement requires regular communication between the referring OD and the surgeon, especially if there are any questions,” Dr. Lucas reiterates. “There’s no such thing as a stupid
question. Optometrists need to feel comfortable enough to be able to ask questions about the care of their shared patients or how to handle a particular group of patients.”

Open lines of communication also help ensure the necessary follow-up occurs. “Failure to return for follow up visits is a risk; however, regular communication between physicians as well as improved understanding of one another’s scheduling preferences can help combat this issue,” Dr. Lucas suggests.

**Taking the Lead**

As the ranks of optometrists grow, ODs are in the position to take even more of a leadership role across the continuum of eye care. Beyond surgical procedures, there are other areas of cornea care that can often be handled solely by the optometrist.

There are a variety of ways ODs can take the lead, but two in particular are dry eye and corneal erosions, suggests Dr. Ibach. “Dry eye is definitely a space where optometrists want to do absolutely as much medical treatment as they can for those patients,” he says. “More specific to cornea, recurrent erosions are another.”

If a patient has a corneal erosion, it could be trauma- or dry eye–induced and a one-time event, he explains. “An optometrist can treat that patient in the office with topical antibiotics, lubrication and anti-inflammatories; often, a bandage contact lens or amniotic membrane will help for healing, too.” If it begins to become recurrent, then a referral may be necessary, but “if it’s just the first corneal abrasion or erosion, you don’t always have to send that off for a superficial keratectomy or PTK, especially if the optometrist is comfortable with these conditions.”

A number of clinical entities, such as microbial keratitis, require medical intervention rather than surgery and are well within an optometrist’s scope of practice. Why might an OD opt to send these cases to a specialist? For many, it comes down to what they are comfortable with and their experience with these cases.

“Comanagement looks different depending on your practice. Some generalists are comfortable managing an array of issues—ranging from corneal debridement for patients who have recurring erosion to treating corneal infections—and will only refer when they need a corneal transplant or lamellar surgery,” says Dr. Shovlin. “On the other end of the spectrum are optometrists who prefer to refer any cornea-related issues for confirmation of a diagnosis and appropriate treatment.”

“Confidence comes with experience,” he notes. “It’s a matter of seeing enough patients with a particular condition to feel comfortable managing their care. Spend time observing and learning so you can take on cases that are within your scope of practice. I would welcome anyone to come to our practice and spend a day or two with me and our corneal specialists.” Dr. Shovlin practices in Scranton, PA.

**Continuing Education**

Whether you’re an OD who wants to start expanding your cornea care or a seasoned clinician looking to take your expertise to the next level, there are number of ways to enhance your practice and become more comfortable caring for these patients.

“Optometrists should actively seek continuing education on current corneal practices, specifically with corneal specialists within their geographic area,” suggests Dr. Lucas. “These events are a good way to hear about new developments in their practice regarding patient care or new technology being used.”

In the time of COVID-19, there is no shortage of online offerings, notes Dr. Ibach. “I always recommend our national meetings, such as the American Academy of Optometry or American Optometric Association. There are also myriad regional meetings and focused optometric groups,” he says. “Many groups have a virtual library of accredited courses that can help you improve your practice.”

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**HOW TO WRITE A REFERRAL LETTER**

Providing detailed patient information lays the foundation for effective comanagement. Here is an example of a referral letter that will set you, your patient and the ophthalmologist up for success.

April 5, 2021

**RE: patient Randolph Duke**

Dear Dr. Winthorpe,

Please allow this letter to introduce my above-named patient, a 17yo white male, who has recently shown signs of progressive keratoconus on both his slit lamp exam and corneal topography. His best corrected acuity is OD 20/30+ and OS 20/25- with a moderate amount of myopia and oblique astigmatism that has increased significantly the past four months.

He shows mild apical thinning, posterior corneal stress lines and what appears to be faint iron lines inferiorly in both eyes. His topographic findings include inferior steepening in both eyes. He shows many of the discouraging signs of progressive keratoconus. The remainder of his ocular exam is normal, including a dilated fundus exam and intraocular pressures.

I’d like for you to evaluate the patient’s suitability for corneal crosslinking at this time.

Be assured that by way of introduction, I have provided sufficient information about corneal crosslinking, including expectations, potential risks and alternatives to this treatment option.

I am glad to provide any additional information you might find helpful. Thanking you in advance for seeing this pleasant young man.

Sincerely,

William R. Valentine, OD
A strong relationship with the surgical practice you refer to can also be beneficial to your professional growth. “Oftentimes, just jumping on the phone or even going in and spending a couple hours with the surgeon can be an excellent way to learn,” Dr. Ibach explains.

“Given the broad range of potential cornea conditions and procedures, everyone is not going to be comfortable comanaging every single one,” Dr. Shovlin states. “It is important to know your comfort level and not overextend yourself because that’s not good for you or the patient.”

That being said, “don’t be afraid to expand your knowledge and skills,” he continues. “Find a colleague—this can be a specialist or a local optometrist who has a special interest in the cornea—who can help guide you clinically so next time you see that particular condition you are more comfortable managing the patient.”

Addressing Gaps in Care
While advances in cornea management continue, challenges remain, and optometrists are uniquely positioned to tackle these difficulties and fill any gaps in care.

One area where optometrists can have significant impact is keratoconus. With the advent of corneal crosslinking, these patients now have an available intervention that could change the course of their disease. However, many patients are not receiving this treatment as soon as they could.

“With corneal crosslinking being an emerging area in the field, focusing our attention on diagnosing
keratoconus as early as possible and providing treatment will ensure the best possible outcomes for patients,” says Aaron Bronner, OD, of Pacific Cataract and Laser Institute, a large OD-MD multidisciplinary practice in the Northwest. “This requires a shift in professional screening for this disease. We have usually just let them develop organically because there hasn’t been a way to stop progression.

“When keratoconus gets bad enough it becomes apparent and the diagnosis is easy, but what we want to do at this point is identify the condition before it becomes really apparent,” he continues, noting that this requires the appropriate screening tools. “The Pentacam (Oculus) is a very good device that can diagnose keratoconus at stages way before the patient has symptoms.”

If an optometrist has a patient with findings consistent with the condition, such as dramatically increasing astigmatism or not correcting to 20/20 with glasses, they can use this device to confirm their suspicions, suggests Dr. Bronner, who acknowledges this technology may not be available in every optometry practice due to cost constraints.

“If you don’t have the device at your clinic, you can send them to the surgery center that comanages with you for testing only,” he notes. “It doesn’t have to be a full referral and is a cost-effective approach to diagnosing a problem.”

Another potentially challenging but important aspect of care is knowing when to make a referral, particularly when it comes to a procedure like corneal transplant. “Although there have been improvements thanks to DSEK/DMEK, corneal transplants are still challenging procedures with a greater risk of complication than routine procedures like cataract surgery,” says Dr. Bronner. “Therefore, you wouldn’t recommend this surgery at the first signs of endothelial disease. Instead, waiting for the condition to progress somewhat is important.”

“Keep in mind, the risk of DSAEK and DMEK are lower than traditional PK, but higher than say cataract surgery. Because of that, you’ll want to see a bit more VA reduction prior to a surgical referral than you would for a routine cataract operation,” he explains. “Of course, any corneal comanagement center will be happy to see your patient as early as possible in the disease process as you like, but one of the most critical aspects of comanagement is the patient hearing a consistent message from both the referring doctor and the surgery center. Therefore, early referrals should be accompanied by more guarded recommendations for surgery.”

Depending on how you handle comanagement, it can have a positive or negative impact on your patient relationships. “An important part of the patient relationship is the long-term trust that’s been built over time,” notes Dr. Ibach. “That can be put in jeopardy if you don’t thoroughly educate a patient on your findings prior to referring to a specialist.”

For instance, if a patient is “diagnosed” with basement membrane dystrophy or keratoconus at the surgical center, but they were referred for a different condition, it can cause them to doubt their primary eye care provider and be hesitant to return to their practice for postoperative care, according to Dr. Ibach. “Oftentimes, the OD did see and diagnose this problem, but didn’t discuss it with the patient prior to referral,” he explains. “As a result, there is potential for trust to breakdown between the patient and maybe even the co-managing ophthalmologist.”

An ongoing commitment to growing as a healthcare provider, both individually and in collaboration with ophthalmologists, is invaluable to not only for patients, but the profession at large.

“Our ophthalmology colleagues are staying pretty even in number while the patients who need care only continue to grow,” notes Dr. Ibach. “The more that we as optometrists can help facilitate pre- and postoperative management, the more opportunity we’re going to have for our surgical partners to take care of patients from a surgical standpoint.

“Comanagement is also a practice builder. Providing exceptional care can lead to referrals not just from patients, but also the ophthalmologists you work with,” he concludes. “Collaboration, when done well, elevates both patients and their providers.”
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Imagine encountering a progressive, vision-threatening ocular disease like glaucoma and recommending a treatment plan focused on monitoring, while temporarily improving acuity with glasses and contact lenses, and reserving treatment for only the most end-stage cases. Up until five years ago, this was the standard management approach for patients with keratoconus.

As eye care providers, how do we stop keratoconus, a disease that causes irreversible vision loss, before it gets to the final surgical option? This article presents a new paradigm in keratoconus management that focuses on early diagnosis and earlier intervention, to first catch and then halt this ocular disease.

The standard of care for keratoconus management should shift away from a “monitoring” approach and instead embrace an “interventional” one. The new mantra should prioritize new technology to facilitate earlier diagnosis united with early treatment to stop disease progression. The current staple in halting disease progression by strengthening the corneal biomechanics is corneal collagen crosslinking (CXL).

Once the cornea is stabilized, this opens the practitioner’s refractive toolbox of glasses, contact lenses, intrastromal corneal ring segments (ICRS) and topography-guided photorefractive keratectomy. Optometrists play a key role in all three phases of the keratoconus patient life cycle, starting...
with diagnosis, because many keratoconus patients initially present to optometric practices. After stabilizing treatment (second phase), optometrists are the key providers for optical rehabilitation, the final phase.

**Prompt Diagnosis**

Advancements in diagnostic technologies have simplified diagnosis for practitioners, especially in the early or pre-clinical keratoconus patient. Even if your practice doesn’t have these new tools, you can still be a master diagnostician. For example, honing in on a “scissoring” reflex with retinoscopy can approach tomography in sensitivity and specificity for diagnosis. In keratoconus patients, the retinoscope reflex bending or bowing is a sign of corneal irregularity. Critically analyzing refractive increases in myopic spherical equivalent and or cylinder increases can raise red flags for early ectasia. An adolescent patient who has yearly continued increases in myopic spherical equivalent greater than 1.00D deserves further testing.

Diagnostic technology can be subdivided into tools for definitive diagnosis where clinical keratoconus is present and diagnostic tools for pre-clinical disease. Assessing corneal curvature with topography and tomography is a mainstay in keratoconus management. A non-symmetric elevation pattern or bowtie, most commonly displaying inferior steepening on topography, is pathognomonic for corneal ectasia (Figure 1). Corneal topography is an analysis of the anterior corneal curvature which reports two simulated keratometry values (steep and flat K), corneal astigmatism and the corneal shape/symmetry pattern. Topographers most commonly apply placido disc imaging techniques, but scanning slit technique (Orbscan, Bausch + Lomb) does allow posterior corneal measurements.

**Fig. 2. Pentacam Belin-Ambrosio Enhanced Ectasia Display showing early keratoconus.** Note how the corneal percentage thickness increase (lower right graph) is outside normal limits. Also, the color-coded “front” and “back” differences, comparing this patient to a best-fit sphere, are a cause for concern.

Advanced technology is giving practitioners more sensitive data that can allow for earlier interventions. While these new tools are valuable additions to the optometrist’s arsenal, clinicians can still diagnose keratoconus even if they do not have access to these technologies. Corneal tomographers employ Scheimpflug imaging which uses a rotating camera to analyze slit beams at different angles. This technique is best suited for a non-planar surface like the cornea.
Corneal tomography summarizes anterior corneal shape, posterior corneal shape and corneal pachymetry mapping, and can enroll advanced analyses specifically designed for corneal ectasias like the Belin-Ambrosio Enhanced Ectasia Display (Figure 2). Tomography has become the diagnostic gold standard for management of the irregular cornea in today’s research and clinical practice. Anterior segment optical coherence tomography (AS-OCT) is another tool that fits more likely as an adjunctive diagnostic for definitive keratoconus diagnosis. AS-OCT provides a high-definition cross-section of the corneal shape.

In the pursuit of earlier diagnosis, evolving tools for pre-topographic detection of keratoconus are gaining traction. Epithelial mapping uses specialized OCT, which is able to measure and map the most anterior corneal layer. Specifically comparing keratoconic eyes vs. normal, a keratoconus patient’s exhibit thinner central epithelium, thinner minimum epithelial thickness, thinner inferior temporal epithelium and a greater difference in superior to inferior epithelial mapping. Summarizing these findings, the epithelium drifts toward thinning directly over the area of posterior corneal bulging, which corresponds to the apex of the cone, and can precede topographic findings.

Although familial history shows a scattered genetic pattern, patients with a positive keratoconus familial history are at a higher risk of developing the disease. Having a first-degree relative with keratoconus is an established risk factor for the disease. Thus, genetic testing is a blossoming method for determining a patient’s risk. AvaGen (Avellino Labs) is an in-office cheek swab that examines 75 genes with over 1,000 variants to quantify a patient’s relative risk for keratoconus. In genetically high-risk patients, this data may spur practitioners and patients to tighten their follow-up schedule, seek genetic counseling or consider interventional treatment at an earlier stage.

**What is Keratoconus?**

The condition is a non-inflammatory, bilateral, often asymmetric corneal degeneration that leads to corneal thinning and steepening (ectasia). In the early stages, patients will lose uncorrected visual acuity, followed by increases in myopic spherical equivalent and regular astigmatism, and finally lose best-corrected visual acuity (BCVA) due to irregular corneal astigmatism.

The prevalence of keratoconus is debatable and inarguably increasing in published literature and among several demographic groups. A now outdated but heavily referenced study for US rates of keratoconus revealed that about one in 1,835 people were affected by the disease. More recent global estimates of keratoconus reported a worldwide prevalence of about one in 750. Most recently, the Raine Study looked at a cohort of 1,259 patients from Western Australia and found that the prevalence of keratoconus to be 1.2%, or one out of every 84 people. This rise in reported prevalence isn’t because keratoconus has suddenly become transmissible or contagious, but rather is likely due to improved diagnostic technologies and increased patient awareness driven by the availability of new treatment options.

A keratoconus patient’s risk profile can vary based on family history and environmental factors. Overall, the disease shows weak genetic linkages in both autosomal dominant and autosomal recessive genes. First, systemic diseases showing increased risk of keratoconus include Down syndrome, Ehlers-Danlos syndrome, osteogenesis imperfecta and mitral valve prolapse among others to a lesser extent. Studies have shown that patients with Down syndrome have anatomically thinner and steeper corneas, an increased likelihood of aggressive eye rubbing and a keratoconus prevalence at least 10 times higher than non-Down syndrome patients. Mechanical eye-rubbing is a modifiable risk factor for the development and progression of keratoconus. Ocular atopies (including allergic/venal conjunctivitis and atopic dermatitis), floppy eyelid syndrome, dry eye disease, blepharitis, digital eyestrain and poorly fitting contact lenses can all lead to the stimulus to rub. Treating these comorbid ocular conditions is an important adjunctive approach for keratoconus. The painless nature of keratoconus, combined with the relative ease of maintaining adequate visual acuity with stronger optical devices, may cause delays in the detection of ectasia.

**Early Intervention**

After diagnosis, the next step in keratoconus management is to prevent further progression. Currently, the only treatment aimed at halting corneal ectasia progression with long-term efficacy is CXL. Optometrists play a key role in pre-op education and referral. They are also instrumental in collaborative care including vision rehabilitation with optical devices.

A timely referral for intervention is critical because vision loss due to corneal warpage is often irreversible. In many cases as an early keratoconus patient progresses, changing the prescription in glasses or contact lenses can improve acuity but mask worsening disease. Since patients aren’t born with the disease, all keratoconus patients had progression at one point, but younger patients and patients who are aggressive eye rubbers have been shown to have a greater risk of keratometry steepening/worsening. The right patient for CXL is any patient showing progression to this ectatic disease who can safely receive treatment.

Concerning the question, “Which comes first: crosslinking or specialty contact lenses?” I prefer to crosslink these corneas first. After the cornea has been stabilized, specialty contact lenses should provide years of
improved visual acuity. However, if a patient has reduced visual function at work or is unable to drive due to vision loss, a specialty contact lens may bridge the gap and greatly improve their quality of life. It’s imperative to remember despite improved visual acuity, these patients still have a biomechanically unstable cornea.

The main contraindication to CXL is pregnancy. This patient subset was not studied in the FDA clinical trials.9 Relative contraindications include individuals under the age of 14 or older than 65 years.9 More commonly patients under age 14, but in both groups, CXL has been safely performed and can be of tremendous benefit. Patients with active infectious keratitis should be avoided as this is currently off-label, but studies suggest potential benefit and are ongoing.

Epithelium-off CXL was approved by the FDA in 2016 for the treatment of progressive keratoconus and post-refractive surgery ectasia. CXL is a medical procedure that combines riboflavin (vitamin B2) photosensitizer with ultraviolet light (365nm to 370nm) to stiffen the cornea. The coupling of riboflavin and UVA light forms free radicals and singlet oxygen molecules creating covalent bonds or “crosslinks” in the corneal lamellae.10,11 This chemical reaction leads to a shortening, thickening and stiffening of the corneal tissue.10,11 The currently approved Dresden protocol starts with epithelium removal followed by a 30-minute riboflavin soak before UVA irradiation for another 30 minutes (Figure 3).12

The pivotal trial for epithelium-off CXL in the US was a prospective, randomized, controlled clinical trial that examined 205 patients with progressive keratoconus.9 Patients were randomized into a treatment group, which underwent CXL with the Dresden protocol, and a sham control group, which received the photosensitizer but no epithelial debridement or UV exposure.12 The primary outcome was the mean change in maximum keratometry (Kmax) value at 12 months post treatment.9 At 12 months, the sham group steepened 1.0D on Kmax while the CXL group showed Kmax flattening of 1.6D.9

A crucial step in the CXL referral process is patient education and setting realistic expectations. Epithelium-off crosslinking is FDA approved and, when progression is well documented, commercial insurance coverage for patients is now quite good (Figure 4). Compared with 2017, when only three insurance carriers covered CXL, today 96% of commercially insured patients have coverage for CXL.13

Epithelium-off CXL’s primary goal is to freeze the cornea in place, and large improvements in corneal flattening or visual acuity can’t be overpromised.

Optometrists referring patients for CXL can help prepare patients by telling them two things: initially vision will be worse for about a week before it moves back to baseline, and the surgical eye will have a mild to moderate amount of pain, discomfort and photophobia for three to four days.

Although pre-op CXL education should focus on the goal of stability, a clinical trial showed on average patients achieved Kmax flattening plus a bonus of mild improvement to BCVA.9 The most frequent adverse event in the treatment group was corneal haze, but only three eyes had retained haze/scarring at one year, and only two of those showed a decrease in BCVA.9 Long-term, CXL has shown continued corneal stability out to seven and 13 years in two separate studies.14,15 One study had a Kmax progression in 0% of cases, while the other had a 7% failure rate at 13 years.14,15

The future is also promising for accelerated epithelium-on CXL (ACXL) in the US. ACXL speeds up the procedure and healing for patients and may allow more patients to be treated. Two hurdles in the epithelium-on quest are the barrier function of the epithelium and the CXL reaction requirement of surplus oxygen.16 Epithelium-on CXL is not currently approved, but FDA trials are underway.

**Fig. 3. Procedural view during CXL.**

**Fig. 4. Example of progression parameters that can be helpful for insurance reimbursement.**
Optimizing post-op care for CXL requires a collaborative approach between the referring practitioner and the surgical practice. CXL has no global period, so follow-up care providers should bill office visits and ancillary testing accordingly. Similar to photorefractive keratectomy (PRK), an important early healing step is achieving corneal epithelialization and deciding when to remove the bandage contact lens.

Immediately following the procedure, we can educate patients that their vision will be worse, slowly improving to functional vision around one week and back to baseline at post-op month one to three. If a topography/tomography is performed at one- or three-months post-op, it is not uncommon to see steepening/worsening secondary to epithelial remodeling, and the most informative keratometry stability measurements are nine to 12 months postoperatively. Similarly, corneal pachymetry often measures thinner post-op. Stromal corneal haze is an expected finding at one- and three-months post-op and predictably will fade over time (Figure 5). This haze is visually insignificant and resolves without further intervention.

Since CXL is not a refractive procedure, patients may be anxious for new glasses or contact lenses. Depending on patient function, the Rx can be predictably updated at three months post-op. For patients with exceptional needs, prescription or re-fitting may occur sooner than three months with the expectation that significant adjustments may be necessary in the near-term as the cornea stabilizes.

**Vision Rehabilitation**

The proposed third step in our new mantra for keratoconus management is vision rehabilitation, an area where optometrists should be front and center. Once the cornea is biomechanically, and largely refractively stabilized, a myriad of options opens up in the refractive toolbox. In reviewing these options, all have their individual “pros” and “cons,” and they are commonly employed together.

No matter the severity of the keratoconus, it is important that patients have an updated glasses prescription. At minimum glasses provide a “contact lens break” in patients who are heavily reliant on these lenses. If a patient’s keratoconus is mild and glasses correction satisfies their visual needs, this is a great option because patient convenience and safety is unmatched. Unfortunately, in moderate to severe ectatic disease where more corneal warpage has occurred, higher-order aberrations like glare, starbursts and ghosting aren’t corrected by spectacle lenses. If a keratoconus patient has developed ectasia-induced scarring—for example, a patient with a history of corneal hydrops (Figure 6)—glasses will be of little visual benefit.

Contact lenses for keratoconus can be divided into “soft disposable lenses” and “specialty hard lenses.”

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**Fig. 5. Patient one month post-CXL with anterior corneal haze. Note the demarcation/hyperreflective line on OCT.**
In large part, disposable soft contact lenses provide similar vision quality in ectatic corneas compared to glasses because the soft disposable lenses mold to the irregular anterior corneal shape. Specialty soft lenses, which are dispensed in more mild disease, are designed for the irregular cornea and feature an increased lens center thickness to help retain their shape over an irregular cornea thereby providing more optimal vision correction.

Gas permeable (GP) contact lens designs are more frequently used for patients spanning all levels of keratoconic disease because the tear prism under the lens masks the irregular anterior refractive surface. By minimizing higher order aberrations (glare, ghosting, starbursts) in ectatic corneas, the vision improvement with GP lenses can be life-changing for patients. There are a variety of gas permeable lens options available from smaller corneal and limbal designs to larger diameter scleral lenses. If the optics of a gas permeable lens are desirable, hybrid lenses (GP center and soft lens skirt) are also an option for the irregular cornea patient.

Specialty contact lenses not only improve patient visual function, but by masking corneal irregularities, they also likely delay or prevent corneal transplantation surgeries for patients. One of the newest studies compared the rates of keratoplasty in patients who wore either no lenses, soft lenses, corneal gas permeable lenses or scleral lenses for vision rehabilitation in keratoconus. This study concluded that GP corneal lens or scleral lens wear significantly lowered the risk of undergoing keratoplasty.

In specialty lenses, specifically scleral lenses, innovations in lens Dk (oxygen permeability), solutions and coatings have improved patient comfort, wear time and negative visual symptoms. Increasing a lenses Dk in simple terms means increasing the oxygen transmissibility from the environment to the cornea. Studies have suggested increasing Dk to a value of 150 or higher, but physicians must balance higher Dk lenses with the potential for less wettability and increased lens debris deposition.

Hydra-PEG (Tangible Science) is a novel biocompatible polymer that creates a more consistent and durable lens coating. Hydra-PEG surface treatment has been shown to increase lens comfort, decrease lens deposits and decrease lens fogging. A third expanding area is scleral lens insertion solutions. Buffered vs. non-buffered products, single-use vials and the addition of electrolytes among others have all been additions to practitioner options.

A final addition in scleral lenses is impression-fit lenses. Analogous to a dentist using impression molds to make dental implants with a precision fit, these impression-based scleral lenses can be customized to a patient’s cornea and sclera. Impression-based lenses can serve all types of patients, but commonly they are reserved for highly irregular corneas with more advanced pathology.

In the surgical space for keratoconus vision rehab, there are both addition procedures and subtraction procedures. First, addition procedures revolve around using either biocompatible PMMA intrastromal ring segments (Figure 7) or allogenic ring segments inserted into the deep stroma.

Intrastromal ring segments aim to flatten and reshape the ectatic cornea while concurrently mechanically supporting the cornea. By mechanically centering the steep irregular cone, ICRS have the ability to decrease myopia and astigmatism, leading to better vision in glasses and disposable contact lenses. In our practice, our usage of Intacs most commonly is sequential to corneal crosslinking. When a patient’s best-corrected glasses vision is less than 20/40, or a patient is unable or unwilling to wear specialty contact lenses, these are the most frequent
Optometry’s role in the management of keratoconus is preserving patients’ visual potential and rehabilitating visual acuity to maintain a higher quality of life. Innovations in treatment allow practitioners to deviate from “monitoring the bend until it breaks,” to a more interventional mindset.

Researchers studied quality of life in keratoconus patients and, not surprisingly, BCVA in the better-seeing eye was the number one factor in quality of life. Early diagnosis and earlier intervention align beautifully with the goal of saving BCVA. CXL also contributes to higher quality of life scores in both early and late stages of keratoconus.

Adopting a management approach where early diagnosis leads to intervention with CXL allows the practitioner to follow-up with a number of options for vision rehabilitation. This shifts patients away from a cycle of “watch and wait” and toward the new paradigm of “intervene and treat”—a more proactive approach that any eye care practitioner can support.

- A key in setting expectations for TG-PRK in keratoconus patients is to change the definition of “success” from total glasses and contact lens independence to alleviating the dependence on specialty lenses, therefore gaining good visual quality in glasses and contact lenses.

Fig. 8. Topography-guided PRK ablation profile for a keratoconic patient 12 months post-CXL. Note the inferior increased amount of planned laser energy.

Acknowledgments

We thank the Optometric Study Center for providing the topography-guided PRK data presented in Figures 7 and 8. We also acknowledge the contributions of the authors listed below.

References


## OPTOMETRIC STUDY CENTER QUIZ

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1. The prevalence of keratoconus is on the rise due to increased detection. A recent Australian study showed the highest rate at approximately what level?
   a. 1 in 1,000.
   b. >1 in 100.
   c. 1 in 500.
   d. <1 in 1,000.

2. Which of the following systemic diseases is associated with an increased risk of keratoconus?
   a. Parkinson’s disease.
   b. Lyme disease.
   c. Ehlers-Danlos syndrome.
   d. Legionnaires’ disease.

3. Which of the following conditions can lead to increased eye rubbing?
   a. Eye strain.
   b. Allergic conjunctivitis.
   c. Atopic dermatitis.
   d. All of the above.

4. When counseling patients on the effects of corneal crosslinking, the portrayed goal should be?
   a. To help patients immediately see better after CXL.
   b. To halt corneal progression, flatten the keratometry values and improve contact lens fits.
   c. To halt keratometric progression.
   d. To set patients up for the ability to have topography-guided laser vision correction.

5. When comparing normal eyes to early keratoconic eyes, the epithelial OCT maps of keratoconus patients show which change?
   a. Thinner minimum epithelial thickness.
   b. Increased superior epithelial thickness.
   c. Thinning in the midperipheral cornea.
   d. No difference.

6. Genetic testing for keratoconus traits can now examine approximately how many genes?
   a. 70.
   b. 85.
   c. 99.
   d. 125.

7. Which function do specialty contact lenses for keratoconus best serve?
   a. Altering corneal biomechanics.
   b. Rehabilitating visual function.
   c. Strengthening corneal lamellae.
   d. Reducing eye rubbing.

8. Which of the following is true regarding the FDA clinical trials for epithelium-off corneal crosslinking?
   a. The control group received riboflavin and epithelial debridement.
   b. The treatment group was treated following the Dresden protocol.
   c. The control group received riboflavin, but nothing else.
   d. Answers B and C.

9. What was the primary outcome in the FDA clinical trial for corneal crosslinking?
   a. Change in steep keratometry/Kmax at 12 months.
   b. Improvement in best-corrected visual acuity.
   c. Prevention of corneal transplantation.
   d. Improvement in uncorrected visual acuity.

10. What was the most common adverse event in the FDA clinical trial for corneal crosslinking?
    a. Infectious keratitis.
    b. Corneal haze.
    c. Permanent reduction in BCVA.
    d. Corneal staining.

11. For coding requirements, how long is the global period for epithelium-off corneal crosslinking procedures?
    a. 10 days.
    b. 30 days.
    c. 90 days.
    d. There is no global period.

12. After CXL, it is common for the topography/tomography to look for the first few months, and the for the pachymetry to become.
    a. improved, thicker.
    b. improved, thinner.
    c. worse, thinner.
    d. worse, thicker.

13. In more advanced keratoconus, glasses are less effective for patients due to:
    a. High amounts of myopia.
    b. Lower-order aberrations.
    c. Higher-order aberrations.
    d. Regular astigmatism.

14. Increasing the Dk of a contact lens material serves what purpose?
    a. Increase the oxygen transmissibility.
    b. Decrease the deposition formation.
    c. Steepen the base curve.
    d. Decrease the wear time.

15. Which of the following specialty contact lens options represents the highest level of customization for a keratoconus patient?
    a. Corneal gas permeable lenses.
    b. Scleral lenses.
    c. Impression-molded lenses.
    d. Specialty soft lenses.

16. What is the goal of additive surgical procedures for keratoconus?
    a. Reshape the ectasia.
    b. Stop progression of the ectasia.
    c. Making specialty contact lens fits easier.
    d. Give patients freedom from glasses and contacts.

17. Intrastromal corneal ring segments are inserted into which corneal layer?
    a. Anterior stroma.
    c. Descemet’s membrane.
    d. Deep stroma.

18. Topography-guided laser vision correction creates the ablation profile from which source?
    a. Corneal topography maps.
    b. Higher-order aberration profiles.
    c. Manifest refraction data.
    d. The base curve of a corneal lens.

19. In a recent study on keratoconus patient quality of life, which factor correlated most closely with improvement?
    a. Contact lens wear.
    b. BCVA in the better-seeing eye.
    c. Avoiding corneal transplantation.
    d. Rubbing their eyes.

20. An updated paradigm in the management of keratoconus consists of which of the following sequence of steps?
    a. Diagnosis, monitoring, optical rehabilitation, referral for corneal crosslinking.
    b. Diagnosis, optical rehabilitation, monitoring.
    c. Diagnosis, referral for crosslinking, optical rehabilitation, monitoring.
    d. Diagnosis, referral for keratoplasty, optical rehabilitation.
Examination Answer Sheet

A New Consensus on Keratoconus
Valid for credit through April 15, 2024

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

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

Answers to CE exam: Post-activity evaluation questions:

1. A B C D Rate how well the activity supported your achievement of these learning objectives. 1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent
3. A B C D 22. Discuss the current standard of care for keratoconus.
5. A B C D 24. Determine when CXL is an appropriate treatment option.
6. A B C D 25. Based upon your participation in this activity, do you intend to change your practice behavior? (Choose only one of the following options.)
7. A B C D (A) I do plan to implement changes in my practice based on the information presented.
8. A B C D (B) My current practice has been reinforced by the information presented.
9. A B C D (C) I need more information before I will change my practice.
10. A B C D (D) I do not intend to change my practice.
11. A B C D
26. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ______
12. A B C D 27. If you plan to change your practice behavior, what type of changes do you plan to implement? (Check all that apply.)
13. A B C D 28. How confident are you that you will be able to make your intended changes?
14. A B C D 29. Which of the following do you anticipate will be the primary barrier to implementing these changes?
15. A B C D 30. Additional comments on this course:
16. A B C D (A) System constraints (B) Lack of interprofessional team support
17. A B C D (C) Time constraints (D) Insurance/financial issues
18. A B C D (D) Formulary restrictions (E) Patient adherence/compliance
19. A B C D (E) System constraints (F) Treatment related adverse events
20. A B C D (F) Other, please specify: ___________________

Post-activity evaluation questions:

21. Review the impact of advancing diagnostics. 1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

22. Discuss the current standard of care for keratoconus.
23. Recognize the role of corneal crosslinking.
24. Determine when CXL is an appropriate treatment option.
25. Based upon your participation in this activity, do you intend to change your practice behavior? (Choose only one of the following options.)
26. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ______
27. If you plan to change your practice behavior, what type of changes do you plan to implement? (Check all that apply.)
28. How confident are you that you will be able to make your intended changes?
29. Which of the following do you anticipate will be the primary barrier to implementing these changes?
30. Additional comments on this course:

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

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Even though contact lens designs and materials have improved, the rate of corneal infection in wearers hasn’t changed in over two decades. What are the most common causes of infection? How can we limit them? Are there any barriers?

“The ocular surface is resilient to infectious events,” according to optometrists Mile Brujic and David Kading, who practice in Ohio and Washington, respectively. However, it is not immune to infection. Contact lens abuse can challenge the ocular surface and increase a patient’s chance of developing microbial keratitis.

Interestingly, there is a difference in pathogens between corneal infections that are secondary to contact lens wear vs. those that are not. A recent study demonstrated that the causative pathogen in cultured contact lens-related ulcers was predominately *Pseudomonas* species (44%), followed by gram-positive (33%) organisms, fungi (13%), other gram-negative (6%) bacteria and *Acanthamoeba* (5%). Cultured non-contact lens-related ulcers were primarily caused by gram-positive (64%) and gram-negative (26%) organisms, as well as fungi (11%).

Among the many risk factors, overnight wear of contact lenses and failure to wash and dry hands prior to lens insertion are known to increase the chance of infection. Poor storage hygiene also increases the risk of infectious keratitis. The lens modality is another important factor and can influence corneal infection, with the lowest risk of severe disease occurring in daily disposable and rigid gas permeable lens wearers.

### Infection Origins

Understanding there are risk factors that are modifiable and conveying them to patients is critical to ensure contact lens success and avoid corneal infection, Drs. Brujic and Kading note. Continuous lens wear through the day and overnight creates the greatest risk of corneal infection. Educating patients on the destructive nature of this habit and helping them steer clear of it makes a world of difference in ocular health.

Upon determining an appropriate wear schedule, patients must learn how to properly wash and dry their hands prior to lens handling to avoid introducing any harmful substances into the ocular environment. For the same reason, appropriately cleaning and replacing the case and storing the lenses is equally as important. This last risk factor can be mitigated by prescribing options that eliminate the need for storage cases, such as daily disposable lenses, as long as they are available in the patient’s prescription and able to be prescribed. This modality also takes overnight wear out of the equation.

### Clinical Takeaways

Corneal infection is destructive, but also avoidable in most cases of contact lens wear. Educating ourselves on how to appropriately manage any relevant risk factors and then passing on our knowledge to our patients is key. By doing our part, we’ve given our patients the tools they need to do it on their own.

---

**Operation Risk Reduction**

*It’s up to us to help mitigate the factors that can contribute to corneal infection.*

**Q** Even though contact lens designs and materials have improved, the rate of corneal infection in wearers hasn’t changed in over two decades. What are the most common causes of infection? How can we limit them? Are there any barriers?

**A** “The ocular surface is resilient to infectious events,” according to optometrists Mile Brujic and David Kading, who practice in Ohio and Washington, respectively. However, it is not immune to infection. Contact lens abuse can challenge the ocular surface and increase a patient’s chance of developing microbial keratitis.

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A 76-year-old Caucasian female with moderately advanced open-angle glaucoma, OD>OS, was initially seen in 2007, at which point she was undiagnosed. At that visit, her optic nerve appearances warranted further evaluation, which confirmed she was a glaucoma suspect without frank structural and functional glaucomatous damage.

In 2012, I noted structural changes to both optic nerves, OD>OS, that showed circumpapillary retinal nerve fiber layer (RNFL) thinning OU and affected the neuroretinal rims.

After a trial period of glaucoma medications, we settled on a prostaglandin analog dosed at one drop OU HS. The patient remained on that regimen until 2017, at which time she was switched to Zioptan (tafluprost, Merck) OU HS for toxicity-related ocular surface issues.

The patient presented most recently in February 2021. She reported good compliance with the Zioptan and no resultant irritation. Her other medications included amlodipine, ibersartan, simvastatin and over-the-counter supplements. She is allergic to sulfa medications.

The patient’s entering visual acuities were 20/25- OD and 20/30- OS. Her last refraction was approximately nine months earlier, which yielded similar acuities. Her pupils were equally round and reactive to light and accommodation, with no afferent pupillary defect. Her extraocular muscles were full OU.

Slit lamp examination of the anterior segments was essentially normal. There were bilateral (mild) Salzmann’s nodules off the visual axis OU. Her angles were open OU. Her crystalline lenses were characterized by mild/moderate nuclear sclerosis and cortical spoking to a degree consistent with her visual acuities.

To obtain quality HRT-3 (Heidelberg) and OCT images, the patient was dilated in the usual fashion. Her cup-to-disc ratios were 0.75/0.80 OD and 0.65/0.70 OS. The nerve appearances were consistent with her previous visits over the past few years. The neuroretinal rim OD was thinnest in the superior temporal sector; whereas, the rim OS was thinnest in the inferior temporal sector.

Both macular evaluations were consistent with normal age-related changes centrally and characterized by mild retinal pigment epithelium granulation. There was a small epiretinal membrane along the superior arcade OS, not involving the foveal avascular zone. Her peripheral retinal evaluations at previous visits were normal.

The patient’s average intraocular pressures on Zioptan were 15 mm Hg OD and OS. Central corneal thicknesses were 553 µm OD and 565 µm OS. Applanation tensions were 13 mm Hg OD and 15 mm Hg OS at 10:30 in the morning.

The 3.5mm circumpapillary RNFL scan shows loss of tissue at the 10:30 position in the superior temporal sector compared with baseline, indicating progression. In this case, the damage (progression) first occurs and is more visible in the RNFL.
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Discussion

Periodic follow-ups for glaucoma patients are necessary to identify a variety of problems, with the most important being progression. Stability, or lack thereof, is determined by different testing methodologies, primarily by evaluating structural and functional indices. As we see glaucoma patients over the course of several years, it becomes somewhat easier to determine stability, as we now have multiple points of comparison. Aberrations or declines in structural or functional stability become readily evident with the accumulation of data points. Also with time, the protocols we follow for glaucoma management become almost second nature in stable patients.

Therein lies a caution: just because a glaucoma patient has remained stable for many years doesn’t mean they will always be stable. While gross changes or deterioration are easily detectable, subtle changes can be overlooked if we become complacent. On the other hand is the difficulty in determining progression vs. inter-testing variability. This is especially true in evaluating OCT findings that are measured in microns. While OCT technology has revolutionized glaucoma care, it can be challenging to detect early, subtle progression.

OCT instruments offer a plethora of information, much of which cannot fit in a simple 8.5x11 inch printout. It is helpful for me to have access to all the information obtained when a patient is scanned. Namely, it allows me to evaluate all areas where there may be progression. I have access to a progression analysis tool for OCT scans that not only plots progression in the RNFL and the neuroretinal rim, but also in each sector of both areas.

It is incumbent on us to know the nuances and limits of our imaging technologies, both what they tell us and what they don’t. Understanding each particular technology will help you decipher results from instrument to instrument and from test to test.

In this particular patient, previous visual field studies remained stable, with bilateral arcuate defects and nasal step formation. Her most recent visit aimed to determine structural stability. Along these lines, we need to keep a couple of points in mind. First, we must identify exactly where the disease has manifested. In early conversion or early disease, the damage may be isolated to one of three areas: the macular ganglion cell layer, circum-papillary RNFL or neuroretinal rim; whereas in advanced disease, damage can be seen in all three locations. The inferior temporal and superior temporal sectors of the neuroretinal rim and RNFL are typically affected first.

Next, we must remember how each patient first converted from a glaucoma suspect to a frank glaucoma patient. You may not know this information, especially if you inherited a long-standing glaucoma patient from a different provider. But a good rule of thumb to keep in mind is that wherever or however the initial signs of damage manifested, progression is likely to be seen in the same location or manner.

Which brings us back to this particular case. This patient initially converted to glaucoma as evidenced by changes in her neuroretinal rim (as imaged by the HRT-3) and the peripiotic RNFL (as imaged by OCT). In her right eye, the initial changes were seen in the superior temporal sectors of each. I pay particularly close attention to the areas that were first compromised, despite the fact that progression can occur anywhere.

So, is the glaucoma worsening in this case? It certainly appears so. If we glossed over the case findings, the patient’s subtle progression may have easily been missed. After all, what’s 17µm? Well, it’s 17µm that are no longer there. It’s not going to get better, only worse.

Now we must decide what to do with this information. Is that enough of a change to warrant an adjustment in therapy? Or should we reassess again in a few months? That is where clinical decision-making comes into play. Our technology may help guide us, but the decision is ultimately up to you. Missing the details and doing nothing, however, is not an option.

Progression analysis of the superior temporal sector of the neuroretinal rim over four scans spanning four years. The progression follows the expected gradual decline over time as noted by the green line. This indicates a normal situation and not significant glaucoma progression.

Progression analysis of the superior temporal sector of the RNFL using a 3.5mm scan, showing a subtle decline over a four-year period. This is suggestive of subtle progression.
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Build Back Better
When the corneal epithelium is compromised, sometimes the best approach is to let the eye start over with a clean slate.

BY ETHAN ZIMMERMAN, OD
AUSTIN, TX

We try our best to protect the ocular surface from the onslaught of forces that can disrupt it, but sometimes the superficial cornea just isn’t amenable to repair. If so, superficial keratectomy (SK) can greatly improve visual quality. This minimally invasive procedure removes the epithelium down to Bowman’s membrane, then uses a corneal hoe or beaver blade to remove deposits, opacities or diseased tissue. A diamond Burr polishes Bowman’s, ensuring a smooth surface for new corneal epithelial cells to migrate over and proliferate.

The overall goal is to create a more regular corneal surface by allowing new epithelial growth to proceed uniformly.

Since many cases can be comanaged by optometrists, it’s important for ODs to be knowledgeable about SK’s indications and well-versed in the procedure to tackle any post-op complications. Diseases treated with SK include:

- Salzmann’s nodular degeneration, in which superficial, elevated, blue-white lesions appear in Bowman’s immediately behind the epithelial basement membrane. The pathogenesis is unclear, but these typically occur after chronic inflammation to the ocular surface, which can be brought on by longstanding dry eye disease.
- Anterior basement membrane dystrophy, an inherited disorder and the most common corneal dystrophy, occurs when areas of the basement membrane thicken and protrude, causing raised areas and irregularities. It’s typically diagnosed from significant negative fluorescein staining on the epithelium. Severe cases can induce irregular astigmatism, decreasing quality of vision.
- Recurrent erosion, which occurs from poor hemidesmosomal connection between epithelial cells and their basement membrane. This causes epithelium to slough off in specific areas, which can be quite painful when located close to corneal nerves.
- Pterygium, or abnormal scleral tissue growth that extends over the cornea. This is a protective response usually seen from increased UV light exposure. When significant, it can cause extensive irregular astigmatism.
- Band keratopathy, due to calcium salt deposits in Bowman’s, is usually not visually significant but can be removed if cosmetically unappealing.

Since mild forms of these diseases are managed with various dry eye treatments and topical steroids, SK is typically reserved for more moderate-to-severe cases. However, it may be considered for milder ones that cause only minor irregularities if there is a need for detailed refractive measurements (e.g., prior to cataract extraction surgery).

Postoperative Care
Immediately following the procedure, a bandage contact lens is placed on the eye, sometimes with an amniotic membrane under the lens to aid re-epithelialization. The patient is often prescribed a topical antibiotic, steroid and artificial tear. Discomfort the night after the procedure, as well as the next day, is common. If needed, an oral pain medication can also be prescribed. Vision is also expected the first week as the cornea re-epithelializes and slowly improves over the next few weeks.

Follow-up schedule includes a one-day post-op visit to evaluate comfort, look for signs of inflammation and confirm the bandage lens has remained centered. Next, the patient usually returns one week later to ensure the cornea has re-epithelialized. If so, the lens is removed and the antibiotic is discontinued. Lastly, the patient returns for a one-month post-op visit to ensure vision has improved and stabilized.

Keep in mind that a patient may need to undergo multiple SKs in their lifetime due to the recurrent nature of things like Salzmann’s nodules. The conditions listed can worsen with chronic dry eye, which is more prevalent with age. This procedure is not meant to be a “cure,” but rather a long-term maintenance of the patient’s condition. It’s important to articulate this to your patients to ensure realistic expectations.

About the Author
Dr. Zimmerman is an ocular disease resident at Dell Laser Consultants in Austin, TX. He has no financial disclosures.

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Dr. Cunningham is the director of optometry at Dell Laser Consultants in Austin, TX. He has no financial interests to disclose. Dr. Whitley is the director of professional relations and residency program supervisor at Virginia Eye Consultants in Norfolk, VA. He is a consultant for Alcon.
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Scratching the Surface
Doing a thorough job with corneal abrasions helps greatly.

A recent stint on call for emergencies brought home the importance of properly handling a common condition. The first patient was a 34-year-old man who had been playing with his daughter and cat. His daughter spooked the cat, which then accidentally scratched the man’s left eye. The second was a 47-year-old woman who had a mishap involving a child’s fingernail scratch to her left eye. They presented within an hour of each other. Both had the classic appearance of a person with a corneal abrasion: they were driven in by a family member, held a washcloth across the injured eye and were in such discomfort with tearing and photophobia that they had difficulty engaging throughout the exam. The main difference I found was that the man had been injured an hour earlier, while the woman had been the previous day.

She sought care at a local emergency room, where she got temporary relief with anesthetic. The remainder of the exam involved the physician using fluorescein dye and a cobalt blue filter to correctly diagnose a corneal abrasion, after which she was prescribed a topical antibiotic and discharged. However, the antibiotic did little to relieve her discomfort once the anesthetic wore off; hence, her presentation to seek treatment and relief after not being able to sleep the night before. Adding insult to injury (literally), she incurred a very substantial bill for her ER visit.

Conjunctival abrasions are one of the most common ocular emergencies, encountered and treated by optometrists, ophthalmologists, ER physicians, physician assistants and nurse practitioners.1,2 However, it is the eye care professional who is poised to offer the best therapy.

Breaking the Cornea Down
Patients typically present, to varying degrees, with acute pain, photophobia, pain upon blinking and eye movement, lacrimation, foreign body sensation and blurry vision following direct ocular trauma. Biomicroscopy of the injured eye often reveals diffuse corneal edema and epithelial disruption. In severe cases, folds in Descemet’s membrane may be visible. Cobalt blue light following instillation of sodium fluorescein dye will illuminate the damaged, denuded epithelium with bright green dye accumulation within the abrasion.3 The initial trauma potentially creates a mild anterior chamber reaction (iritis or iridocyclitis).3

The corneal epithelium is comprised of the stratified surface epithelium (whose microvilli increase surface area and permit adherence of the tear film by interacting with its mucus layer), the wing cell layer (containing the corneal nerves) and the mitotically active basement membrane.

Bowman’s membrane prevents penetrating injuries.4 Superficial abrasions do not involve Bowman’s, while deep abrasions penetrate this structure but do not rupture Descemet’s membrane. Abrasions result from numerous causes, including foreign bodies, chemicals, fingernails, hairbrushes and tree branches, to name a few.

The cornea has remarkable healing properties.5 The healthy epithelium adjacent to the abrasion expands and fills in the defect, typically within 24 to 48 hours.4 Lesions that are purely epithelial in nature often heal quickly and completely, without intervention or subsequent scarring. Lesions that extend below Bowman’s membrane leave scars. Stromal opacification after corneal trauma is often due to myofibroblasts and chemokines forming a disorganized extracellular matrix that these cells and their chemokines produce.7

Treatment
Management begins with visual acuity and then proceeds from adnexal to fundus examination, if necessary. If blepharospasm precludes an acuity measurement, administer one drop of topical anesthetic and measure the acuity immediately thereafter (pinhole, if necessary). Depending on the nature of the injury, evert the eyelids and examine and flush the fornices to rule out foreign material. Instill fluorescein dye (without anesthetic) to identify the extent of the corneal defects.

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to observe for aqueous leakage) if the injury was high speed with any possibility of full-thickness damage. Clean the abrasion and scrutinize it for foreign matter.

If there are loose or ragged edges of epithelium, these devitalized tissues will impede wound healing and should be removed with a cotton-tipped applicator or forceps following instillation of topical anesthesia. Observe the anterior chamber for any evidence of inflammation. A dilated examination may be completed to rule out any posterior effects from the trauma, if indicated.

Prescribe topical antibiotics to prevent infection in cases of corneal abrasion. An inexpensive topical fluoroquinolone or equivalent may be prescribed QID until the cornea has healed. Keep in mind that antibiotic in abrasions is merely prophylactic and will do little to provide healing or comfort. Unfortunately, this is where most non-eye care practitioners end management—with correct diagnosis and prophylaxis but falling short of thorough management. Use topical cyclopedia to put the uvea at rest. This will greatly ameliorate a patient’s pain and photophobia.

Unfortunately, the number of available cycloplegics has been greatly reduced over the years. One drop of 1% atropine or three to five drops of 1% Cyclogyl (cyclopentolate-hydrochloride ophthalmic solution, Alcon) in-office is usually sufficient and typically does not need to be prescribed. For patients who are in a great deal of pain, a topical non-steroidal anti-inflammatory drug (NSAID) can be given for acute corneal pain. Generic ketorolac or diclofenac can be prescribed BID to QID for a short period of time. Avoid topical anesthetics for these patients due to the potential for misuse, with subsequent neurotrophism. Cold compresses, artificial tears and over-the-counter analgesics such as ibuprofen and acetaminophen can be used to relieve acute pain. Pressure patching, while no longer commonly used, is still useful for larger abrasions. Patching has been commonly replaced by the use of bandage contact lenses, and these have been shown to greatly increase patient comfort, especially when employed with the previously mentioned pharmaceuticals. In cases of corneal abrasion occupying more than 15% of the corneal area, prescribe a thin, low water content, low-powered, high oxygen, extended-wear soft bandage contact lens. In cases where there is significant anterior chamber inflammation, a topical steroid or steroid-antibiotic combination agent can be employed; however, the risks of secondary superinfection must be weighed against the benefits.

Topical steroids increase the efficiency of corneal wound healing by suppressing inflammatory enzymes. Such use of steroids merits close follow-up. Reevaluate patients every 24 to 48 hours until the abrasion has significantly re-epithelialized. If a bandage lens is employed, the doctor should remove it at the first follow-up using topical anesthesia and thorough irrigation to float the lens off, to not disturb the healing epithelium. It is often prudent to do this at the biomicroscope with forceps rather than digitally to not damage the cornea further. If necessary, a new bandage contact lens can be reapplied if the abrasion is not adequately filled in at the first follow-up.

**To Sum Up**

Both patients presented here had a similar appearance with corneal abrasions and no evidence of infection.

Following examination, they were cycloplegic in-office with 1% Cyclogel and prescribed a generic fluoroquinolone antibiotic QID. A low-powered, soft extended wear soft contact lens was inserted for each patient, and both were reappointed to be seen by the next day. Since the woman had been injured for a longer period and had more discomfort and inflammation, a topical NSAID was added to her regimen QID as well. Upon presentation the following day, both patients felt much better, their bandage lenses were removed and each had nearly 95% resolution of their abrasions.

The woman did inquire about the reasons the emergency room personnel didn’t do as much to make her feel better. It was explained that there are procedures best left to eye care professionals and that, while her treatment was not incorrect and she didn’t get a corneal infection, it didn’t adequately address her pain and discomfort.

**References**


This woman received her corneal abrasion as a result of being scratched by her child’s fingernail.
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Epithelial basement membrane dystrophy (EBMD) is the most common of the anterior corneal dystrophies and one of the most likely etiologies of recurrent corneal erosion syndrome (RCE). More than 2% of the population is affected. The condition typically develops between ages 20 and 50. Although considered an age-dependent corneal degeneration, it also seems to have an autosomal dominant method of inheritance. EBMD stems from an inherent dysfunction in basal epithelial cells that results in secretion of abnormal basement membrane extending into the epithelium, as well as accumulation of fibrillogranular material between Bowman’s and the basement membrane and also within the epithelium. Over the years our understanding of EBMD has matured, as has our ability to diagnose and manage it. Here, we review causes, symptoms and treatments.

**Diagnosing EBMD**

EBMD is characterized by an abnormal basement membrane that protrudes into the epithelium and the presence of intraepithelial microcysts. Diagnosis begins with a careful history and review of symptoms. EBMD is usually asymptomatic, but patients may present with dryness, fluctuating vision, grittiness or photophobia. In fact, EBMD patients are often misdiagnosed with dry eye. Approximately 10% of patients develop painful, recurrent epithelial erosions. On slit lamp exam, EBMD is characterized by bilateral (and frequently asymmetric) subepithelial fingerprint lines, geographic map-like lines and epithelial microcysts.

**Treating EBMD**

First-line therapies for mild asymptomatic cases include artificial tears, ointments, punctal plugs and bandage contact lenses. More advanced options such as autologous serum eye drops can be useful in more severe cases. In refractive surgery candidates, it is important to consider the presence of EBMD, as it can affect the accuracy of keratometry and biometry measurements, which are essential for correct IOL power selection.

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**EBMD in Cataract Patients**

Intervention is important in all EBMD patients, but it’s of particular concern in cataract surgery candidates since accurate keratometry and biometry depend on a clear cornea and smooth surface. Any corneal lesion, including EBMD, can have an adverse effect on the reliability of such measurements. This, in turn, can impact IOL selection and postoperative visual outcomes. In a small retrospective review of EBMD, cataract patients were preemptively treated with superficial keratectomy or phototherapeutic keratectomy (PTK) to evaluate the impact of treatment on biometry, IOL power prediction and suggested IOL toricity. Researchers found that spherical IOL power decreased for 18 eyes and increased for three. The decrease in power was more than 1.0D for four eyes, 1.0D for seven eyes and 0.5D for seven eyes. There were two instances of power increase by 0.5D and one by 3.0D. Approximately 67% of patients eligible for toric IOLs had adjustments to the recommended IOL cylinder power after intervention, with a mean change of 1.2D.

These findings speak to how essential corneal surface optimization is and the difference it can make. For every degree of misaligned toric power, there is a 3.3% loss of correcting effect of the cylindrical power. Considering that this study found a mean absolute change of more than 39 degrees of the axis of astigmatism, toric IOL alignment inaccuracies could be quite significant.

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**About Dr. Karpecki**

Dr. Karpecki is medical director for Keplr Vision and the Dry Eye Institutes of Kentucky and Indiana. He is the Chief Clinical Editor for Review of Optometry and chair of the New Technologies & Treatments conferences. A fixture in optometric clinical education, he consults for a wide array of ophthalmic clients, including ones discussed in this article. Dr. Karpecki’s full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.
drops, corticosteroids and cryopreserved amniotic membrane also may be used. Medically refractory cases are recommended for surgical treatments, including epithelial debridement before applying amniotic membrane. Both debridement with diamond-dusted burr polishing and PTK ablation to the level of Bowman’s have proven effective.

We have long been aware of the risks EBMD can pose in LASIK patients: severe corneal epithelial sloughing, epithelial ingrowth (73%), diffuse lamellar keratitis (55%), flap microfolds (18%) and flap melting (36%). Knowing this, take great care to detect subclinical EBMD prior to elective refractive procedures. Although we also endeavor to diagnose all forms of RCE prior to cataract surgery, sometimes the necessity of the procedure will lower our treatment threshold. But as co-managing optometrists, we must be on the lookout for EBMD in all anterior segment surgical patients. By treating them to the best of our ability prior to referral, we help overcome significant risks, and at the very least, we reduce the likelihood that eager surgical candidates will experience disappointment when their procedure is delayed.

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**DIAGNOSTIC EQUIPMENT**

**New Non-Myd Camera Aims for Ease**

Practices looking for a new retinal camera without a steep learning curve might want to consider the Ezer EFC-2600, says manufacturer US Ophthalmic. Autofocus allows anyone to perform photography successfully, and the intuitive interface and large touchscreen enable straightforward operation, according to the company.

The EFC-2600 captures 12-megapixel retinal images at a field of view up to 100°, without the need for pupil dilation. It works for anterior segment photography, too. For mosaic imaging, the operator can choose up to 10 fixation targets and the device will combine several results to produce a widefield of view of the retina. The touchscreen can also double as a display capable of side-by-side image view for serial analysis of change between visits.

DICOM compatibility allows images and reports to be easily transferred or integrated with other patient software, US Ophthalmic says. All electronic patient files and reports can be stored on the device and shared with specialists via cloud or email.

**New Language Options Enhance AMD Screenings**

If you’ve recently added the AdaptDx Pro for dark adaptation screening to your practice, or are considering it, bear in mind that the device’s on-board intelligent assistant, Theia, now speaks multiple languages, Maculogix announced. Theia guides patients through the test with instructions and cues to maintain attention. Your tech can operate the device in one language while selecting a different one for the patient to experience, Maculogix says.

Language options now include English, North American Spanish, European Spanish, French, Italian, German and Canadian French. Theia is also available with a British and Australian accent. Tailoring patient instructions to their native language improves communication, understanding and trust, Maculogix says.

**REFRACTION SYSTEMS**

**Digital Refractor Empowers Staff, Speeds Exams**

You can give your manual phoropter a rest and speed up practice efficiency with the newest version of the Visionix Eye Refract system, which comes with an array of upgrades based on user feedback, manufacturer Luneau says. The device combines a digital phoropter, wavefront-based auto-refractometry and keratometry, a digital acuity short chart and integrated lens analyzer in a footprint of just eight square feet, according to a company press release.

Among the new features in the second-gen product, clinicians and techs can now use K readings to fit soft contact lenses without having to run the patient through another instrument, and the device can quickly identify the patient’s refractive “comfort zone.” Additional improvements were made to the algorithm and wavefront laser, resulting in faster and more accurate results on a wider diversity of challenging patient demographics, the press release states.

Luneau staff train new users to follow a scripted, tablet-driven process that generates recommended final prescriptions in about four minutes, the company says. Visionix’s algorithm-guided interface allows even novice users to obtain consistently accurate recommended final prescriptions and K-readings in the pre-test area, the company says, so ODs can focus their patient consult time on more robust medical consultations or other growth opportunities to transform their practice. The final review and approval process remains with the doctor, but they can focus on more challenging patient cases that require use of a manual phoropter to complete the subjective portion, Luneau suggests.

**Fast Phoropter Offers Three-Minute Refractions**

If you’re in the market for a manual phoropter but want the benefits of speed and digital assistance that conventional models lack, the new Vision-R 700 from Essilor Instruments may fit the bill, according to the company.

The Vision-R 700 uses a unique optical module with variable focus and a new cylinder-search method, a company press release explains. These allow simultaneous and continuous variations of sphere power, cylinder axis and cylinder power by automatically compensating for the effect that any change in sphere, cylinder and axis has on the other dimensions. Essilor calls this “digital infinite refraction” and believes it offers great potential for advancements in
refraction methods. The company says its new lens module and use of refraction algorithms cuts time from the refraction process—potentially down to three minutes—without sacrificing accuracy.

Essilor also says the Vision-R 700 offers a wider field of view, which eliminates the need for superposition of lenses as in traditional phoropters, giving the patient a more comfortable viewing experience without the effect of tunnel vision. Another nice feature: the ability to incorporate a patient response of “I don’t know” when presented with Rx choices.

**Vision Tester Fine-Tunes Eye Misalignment**

To help binocular vision patients who need prism, as well as others who find near vision work taxing to their eyes and bodies, this summer Neurolens will be introducing a new version of its testing equipment called the Neurolens Measurement Device Gen 2 (NMD2).

Neurolens says the NMD2 is an objective and thus more accurate way to measure binocular vision because it doesn’t rely on the patient’s subjective feedback, which also allows it to be operated by a tech at any experience level.

The new version’s eye tracking system can identify misalignment as small as 0.1 prism diopters, according to the manufacturer. The diagnostic data from the NMD2 is used to prescribe custom lenses that vary the amount of prism from top to bottom to account for differing visual needs as focal length changes. Neurolens calls this concept “contoured prism” and says it offers ergonomic benefits to patients by allowing better posture during near vision tasks like computer use.

The NMD2 is 80% smaller than the original device, making it easier to incorporate into any practice space, the company says. It’s also 35% faster, which the company says can improve pre-test efficiency and overall patient flow. Finally, the new device also supports faster eye tracking and increased fields of vision, allowing for additional testing capabilities.

The NMD2 will be commercially available on July 1, but the company will take orders immediately as part of a pre-sale offer.

**CONTACT LENSES**

**Two-Week Multifocal Allows Pupil-Independent Fits**

If you have successful Acuvue Oasys patients who are reaching presbyopic age, now you can offer them a correction in the same lens family, as Johnson & Johnson Vision recently introduced Acuvue Oasys Multifocal in the same material (senofilcon) and also for two-week replacement. The new product uses the pupil optimized design of the 1-Day Acuvue Moist Multifocal, which J&J says allows the lens to be fit independent of pupil size.

The design uses a hybrid back curve: an aspheric center allows the lens to drape across the cornea without creating distortion while a spherical skirt holds the lens in place, J&J explains. Like other Acuvue Oasys lenses, it embeds polyvinylpyrrolidone in the matrix, which mimics the mucins found in tear film to help support a stable tear film, according to the company. J&J calls this wetting agent “Hydraclear Plus.”

The Acuvue Oasys Multifocal is available in sphere powers from -9.00D to +6.00D and three add power ranges: low (+0.75D to +1.25D), mid (+1.50D to +1.75D) and high (+2.00D to +2.50D).

**Small Sclerals, Bigger Benefits**

The BostonSight Scleral is now available in 16mm, 16.5mm and 17mm diameters, in addition to the original 18mm, 18.5mm and 19mm diameters introduced in 2017. Each series is part of a diagnostic set that includes three diameter options, right and left anatomical designs to address unique scleral shape profiles, and a fitting guide, the company says.

Unique design features the company points to include:

- oval optic zones for better lens centration and more predictable fitting endpoints
- independence among parameters like base curve, vault and optic zone to allow for easier changes
- ventilating channels that allow fitting lenses over anatomical obstacles, promote tear exchange and minimize suction
- built-in front surface eccentricity and custom HOA correction for optimal vision

BostonSight says that 90% of fits achieve optimal vision, 75% are achieved with the go-to standard lens from the from the diagnostic set and 65% of fits don’t require haptic design change.
A 62-year-old female presented to the office with a chief complaint of floating spots in the right eye of four weeks’ duration. Her systemic and ocular histories were unremarkable for treatable disease. She denied taking medications and reported no allergies of any kind.

**Diagnostic Data**
Her best-corrected visual acuity was 20/20 at distance and near OU. Her confrontation fields were full-to-finger-counting. Pupils were equal and normally reactive with no afferent defect. Refraction uncovered hyperopia with presbyopia OU. Biomicroscopy demonstrated normal anterior segment structures with intraocular pressures measuring 16mm Hg OD and 17mm Hg OS. The photographs illustrate the pertinent posterior segment finding.

Additional studies included photodocumentation and three-mirror evaluation with and without scleral depression.

Ophthalmic ultrasonography may also be helpful in confirming the diagnosis. If uncertain, the clinician may opt for a referral to retinology, where further tests may include deep field optical coherence tomography, additional ultrasound testing, color doppler flow imaging or retinal angiographic testing.

**Your Diagnosis**
What would be your diagnosis in this case? What is the patient’s likely prognosis? To find out, please read the online version of this article at www.reviewofoptometry.com.

Dr. Gurwood thanks Nick Karbach, OD, for contributing this case.
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