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REVIEW® OF OPTOMETRY

August 15, 2019

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43rd ANNUAL CONTACT LENS REPORT

ATTRACT—AND RETAIN—CONTACT LENS PATIENTS

Exceptional care and custom fits will keep patients from straying.

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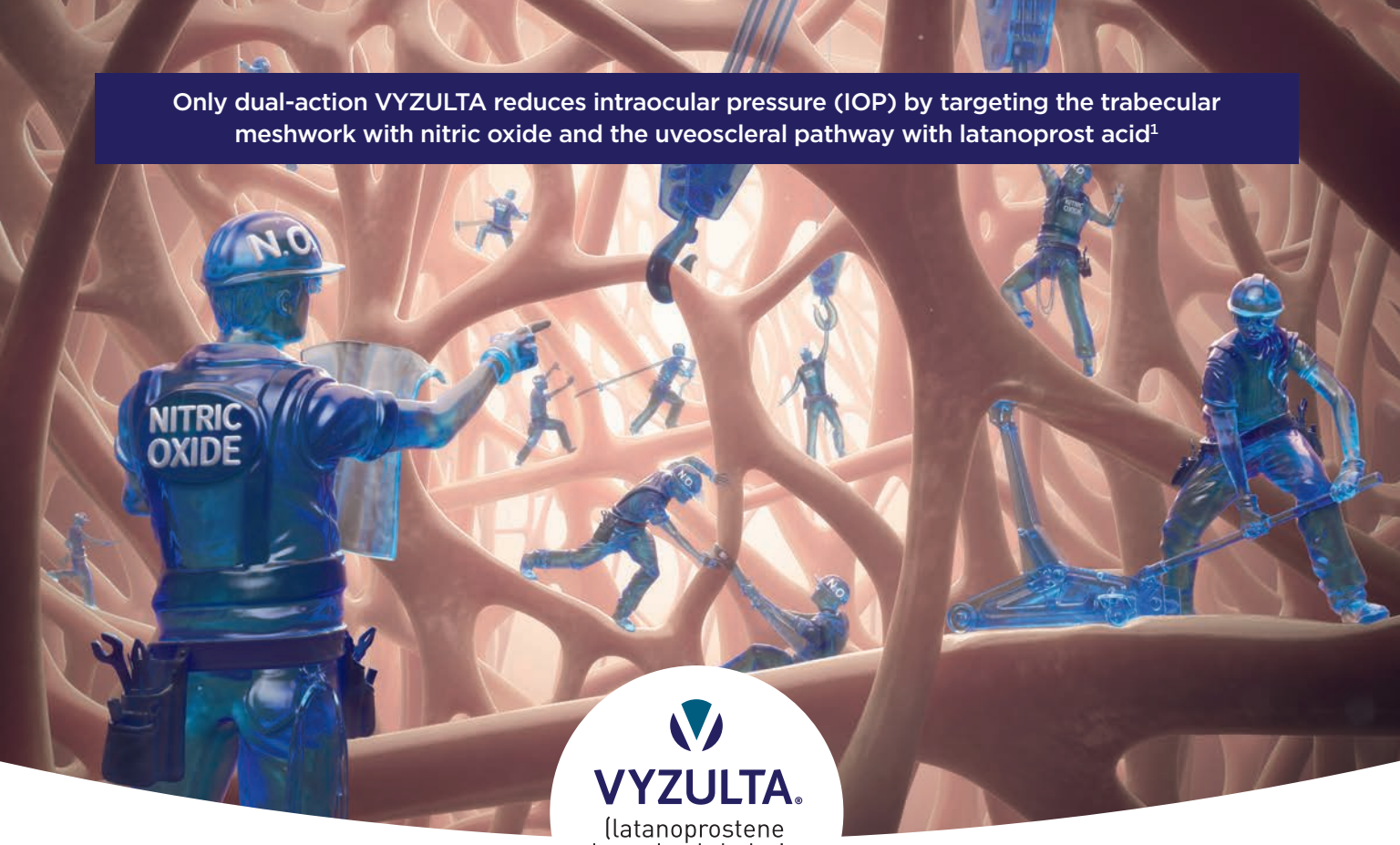

Only dual-action VYZULTA reduces intraocular pressure (IOP) by targeting the trabecular meshwork with nitric oxide and the uveoscleral pathway with latanoprost acid[1]

VYZULTA®
(latanoprostene bunod ophthalmic solution), 0.024%

EXPAND THE TRABECULAR MESHWORK WITH THE POWER OF NITRIC OXIDE[2-6]

VYZULTA achieved significant and sustained long-term IOP reductions vs Timolol 0.5% in pivotal trials[7]

P<0.001 vs baseline at all pre-specified visits over 12 months in a pooled analysis of APOLLO and LUNAR clinical trials (N=831)

VYZULTA demonstrated safety profile in clinical trials

Only 6 out of 811 patients discontinued due to ocular adverse events in APOLLO and LUNAR clinical trials[1,8,9]

Visit VYZULTANOW.com to see our efficacy results

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

IMPORTANT SAFETY INFORMATION cont'd

- 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 Prescribing Information on next page.

References: 1. VYZULTA Prescribing Information. Bausch & Lomb Incorporated. **2.** Cavet ME. *J Ocul Pharmacol Ther*. 2018;34(1):52-60. DOI:10.1089/jop.2016.0188. **3.** Wareham LK. *Nitric Oxide*. 2018;77:75-87. DOI:10.1016/j.niox.2018.04.010. **4.** Stamer DW. *Curr Opin Ophthalmol*. 2012;23:135-143. DOI:10.1097/ICU.0b013e32834ff23e. **5.** Cavet ME. *Invest Ophthalmol Vis Sci*. 2015;56(6):4108-4116. **6.** Kaufman PL. *Exp Eye Research*. 2008;861:3-17. DOI:10.1016/j.exer.2007.10.007. **7.** Weinreb RN. *J Glaucoma*. 2018;27:7-15. **8.** Weinreb RN. *Ophthalmology*. 2016;123(5):965-973. **9.** Medeiros FA. *Am J Ophthalmol*. 2016;168:250-259.

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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.

4 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 periorbital 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 periorbital 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, sternebral and vertebral skeletal anomalies, limb hyperextension 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 hyperextension 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 hyperextension 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 toxicology study administered topical ocular doses of latanoprostene bunod to one eye of cynomolgus 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 not observed at the 0.024% dose.

U.S. Patent Numbers: 7,273,946; 7,629,345; 7,910,767; 8,058,467.

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VOL. 156 NO. 8 ■ AUGUST 15, 2019

IN THE NEWS

Heavy metals, toxic elements and oxidative stress may all play a role in the development of early- and late-stage age-related macular degeneration (AMD), researchers from India suggest. Their study found significantly increased levels of lead, cadmium, chromium, nickel and arsenic in the choroid-retinal pigment epithelium and retinas of donor patients' eyes with early and late AMD.

Aberami S, Nikhalashree S, Bharathselvi M, et al. Elemental concentrations in choroid-RPE and retina of human eyes with age-related macular degeneration. Exp Eye Res. July 1, 2019. [Epub ahead of print].

Another study from India reviewed 229 patients who had penetrating keratoplasty (PK) for microbial keratitis and identified **several significant risk factors for recurring infection, including a fungal etiology, retro-iris exudates, coexisting endophthalmitis and grafts 10mm² or larger**. Despite these risks, the researchers still advocate for PK as an effective treatment for severe cases of unresponsive microbial keratitis.

Chatterjee S, Agrawal D. Recurrence of infection in corneal grafts after therapeutic penetrating keratoplasty for microbial keratitis. Cornea. June 26, 2019. [Epub ahead of print].

Certain systemic drugs—**ACE and alpha-glucosidase inhibitors, fibrates and insulin—may increase the risk of developing cortical cataracts**, a study reports. The investigation also found the association was independent of hypertension, hyperlipidemia and diabetes. "Consistently, the four medications were also associated with a greater severity level of cortical cataract," the researchers wrote.

Dai W, Tham YC, Chee ML, et al. Systemic medications and cortical cataract: the Singapore Epidemiology of Eye Diseases Study. Br J Ophthalmol. July 4, 2019. [Epub ahead of print].

Trifocal IOL Beats EDOF in Near Work

Both still scored high marks for distance and intermediate visual acuity.

By Catherine Manthorp, Associate Editor

When considering which of the newer-generation intraocular lenses (IOLs) may be best for your cataract patients, a study in *Eye & Contact Lens* reports both a diffractive trifocal lens and an extended depth-of-focus (EDOF) lens performed well in distance and intermediate vision, but the trifocal did better for near vision.

In the prospective, six-month study, researchers compared the visual results of both lenses. The investigation enrolled 160 eyes of 80 patients who had bilateral cataract surgery and divided them into two groups. The patients were then implanted with either the trifocal or the EDOF lens in both eyes. In addition to visual acuity measurements, subjects also filled out a spectacle dependence questionnaire.

The study found no statistically significant difference between the groups in monocular and binocular uncorrected distance visual acuity and corrected distance visual acuity. Researchers noted monocular and binocular uncorrected intermediate visual acuity and monocular distance-corrected intermediate visual acuity were also comparable.

However, the trifocal had better results at all near visual acuity measurements. Still, the study reported no differences in visual quality and symptoms between the groups.

While the trifocal had a clear advantage over the EDOF in near visual acuity, both showed excellent performance in distance and intermediate visual acuity, investigators noted. Both IOLs provided high percentage of spectacle independence and patient satisfaction with minimal level of disturbing photic phenomena.

Investigators noted several limitations of their study, including the inclusion of just one trifocal lens design. Significantly, patients completed the questionnaire six months after surgery—any photic phenomena may have decreased by then, and patients may also have adjusted to new routines over the time period. Also, the IOLs were targeted for emmetropia, which may confound near and intermediate vision results, and the follow-up period of six months was relatively short to assess the occurrence of posterior capsule opacification, the researchers said.

Singh B, Sharma S, Dadia S, et al. Comparative evaluation of visual outcomes after bilateral implantation of a diffractive trifocal intraocular lens and an extended depth of focus intraocular lens. Eye Contact Lens. July 5, 2019. [Epub ahead of print].

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2019 Income in *Review*

Like any other year, 2018 was full of highs and lows for optometry, income-wise. Has this year been any better? ***We want to hear how you've been doing financially in 2019.***

While earnings decreased among those with the most experience and the gap widened between self-employed and employed ODs last year, average income increased, the mid-career plateau disappeared and the gender gap narrowed.

Will 2019 continue the positive trend? Last year, 56% of our survey respondents thought so, reporting that they expected an increase in income, while 36% didn't expect a change and only 8% were speculating a decrease. Here's our chance to find out if you were right!

If you're a practicing OD, please take a few minutes to respond to our annual income survey and share your financial experience over the last year with us. The results will be published anonymously in the December issue. All personal and financial information is confidential and used for no other purpose than this survey.

Just in case you need a little extra push, here's an incentive: upon completing all of the questions in the survey, you'll be entered to win a ***$100 American Express Gift Card***. It just takes a few minutes, as there are only a handful of questions. Thank you for your participation—we wouldn't know where the field stood financially without you!

Take the Survey

To participate in the survey, visit www.surveymonkey.com/r/2019incomesurvey or scan the QR code.

Uveitic Glaucoma: a Combo for Concern

Glaucoma patients with uveitis have a much higher age-corrected rate of rapid visual field loss than people with primary open-angle glaucoma (POAG) alone, a large UK-based study reports.

The investigation, using real-world data from five glaucoma clinics in England, also reported that eyes with glaucoma and uveitis had nearly double (1.9x) the risk of disease progression compared with those with POAG, yet the average frequency of visual field monitoring was the same for both diseases—about 10 months.

The study included 205 donor eyes with uveitis and glaucoma and 4,600 POAG-only eyes. Eyes with uveitis presented with worse median mean deviation than those with POAG (–3.8dB vs. –3.1dB), leading researchers to speculate that early visual field loss may be under-detected in uveitic glaucoma. Researchers also noted 11% of eyes with glaucoma and uveitis progressed ≥ 1.5dB, while only 7% of POAG eyes progressed.

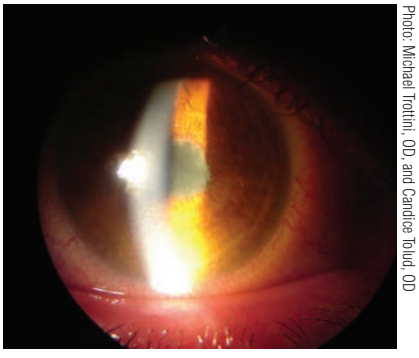

Photo: Michael Trottini, OD, and Candice Tolud, OD

Be on the lookout for glaucoma damage in uveitis patients.

Secondary analysis of intraocular pressure (IOP) parameters showed no difference in the mean IOP between the two groups. However, the researchers noticed the IOP range was wider in the fast-progressing eyes of both groups, with the widest range (21mm Hg) in the progressing uveitic glaucoma group. Further analysis indicated the uveitis group has a higher proportion of significantly progressing eyes (21.2%) compared to the POAG group (18.5%).

By identifying rapid progressors early, clinicians can target interventions to preserve vision.

"While patients with a combination of uveitis and glaucoma lose visual function more rapidly than POAG, on average, they are monitored with visual fields at the same intensity," the researchers wrote.

As such, clinicians managing patients with uveitis should remain vigilant for glaucoma damage in these high-risk patients, the study concludes.

Liu X, Kelly SR, Montesano G, et al. Evaluating the impact of uveitis on visual field progression using large scale real-world data. Am J Ophthalmol. June 25, 2019. [Epub ahead of print].

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Maryland Implements Screen Safety Guidelines

Critic calls the effort to limit digital device use in schools "profoundly mediocre."

With the potential hazards of screen use gaining more widespread awareness, Maryland's Department of Education released a state-sanctioned best practices guide for digital device use in the classroom this month, in accordance with a bill aimed at protecting students from the dangers of excessive screen time. The resulting guidelines offer health and safety pointers, some of which take digital eyestrain into account.[1]

With respect to eye care, the one-page guideline calls for educators in the state to limit students' time on devices to 10 to 20 minutes, with reminders to take eye and stretch breaks. Students should use devices at least 20 inches from their eyes while seated at a desk or table, and educators should keep the lighting conditions even throughout the room to minimize contrast between the environment and the screen.

The state is also offering free resources to help educators ensure proper posture, lighting and appropriate distance from the screen. These documents are available online, along with video guides.[2,3]

In 2018, a Maryland bill requested the state's schools come up with guidelines on how long students can be exposed to screens and how laptops, tablet computers and digital readers can impact a child's ocular health. The state's board of education, in partnership with its department of health, was tasked with working alongside physician groups to develop preventative measures for digital device-associated eye diseases as well as other screen-related health issues.[4]

Cindy Eckard, an activist who worked on the initial bill, called the state's guidelines "profoundly mediocre," and complained that they don't mention digital screens' association with myopia or the impact of blue light on the retina, although it does provide information about blue light's effects on sleep as well as computer vision syndrome and its relationship to dry eye, eye strain and fatigue and headaches. However, it's a start, she notes. "Parents now have a framework to reference when working to protect their children at school," she says.

1. Spector C. Maryland mother pushes for screen time regulations in schools. Star Democrat. www.stardem.com/news/local_news/maryland-mother-pushes-for-screen-time-regulations-in-schools/article_29f329ca-7748-50e4-bfcb-1c298a70bf77.html. July 1, 2019. Accessed July 16, 2019.
2. Maryland Department of Education. Health and safety best practices digital devices in the classroom. Maryland Public Schools. marylandpublicschools.org/programs/Pages/ITSLM/HealthSafetyBestPractices.aspx. July 1, 2019. Accessed July 16, 2019.
3. Maryland Department of Health. Health and safety best practice guidelines: digital devices. Maryland Public Schools. marylandpublicschools.org/programs/Documents/ITSLM/Health_and_Safety_Best_Practice_Guidelines_Digital_Devices.pdf. July 1, 2019.
4. Arentz D. Public schools – health and safety best practices—digital devices. Maryland General Assembly. mgaleg.maryland.gov/webmga/frmMain.aspx?id=hb1110&stab=01&pid=billpage&tab=subject3&ys=2018RS. Accessed July 16, 2019.

Fasting Yields Better FA Results

Researchers recently found that fasting oral fluorescein angiography (FA) yielded images of significantly better quality at a faster, more optimal rate when compared with non-fasting oral FA.

This observational, case-crossover study evaluated 160 eyes of 80 patients undergoing routine oral FA for retinal disease and compared fasting and non-fasting images of the same patient for different image quality parameters.

The researchers found the images taken when patients were fasting achieved better angiography quality scores. They noted that non-fasting patients with higher body mass indexes had the worst scores.

The identification of other clinical parameters, such as drusen staining, disciform scar staining and central and peripapillary atrophy, were also significantly better during the pre-fasting exam. As for test speed, the researchers obtained quality images approximately 22% faster (time to fluorescein dye appearance) when patients had fasted compared with non-fasting (18.7±6.9 minutes vs. 25.14±8.1 minutes).

"Oral FA could be a useful adjunctive examination to optical coherence tomography (OCT) and OCT angiography in patients who require FA studies but who have difficult access or refuse an invasive procedure," the study authors concluded. ■

Amador-Patarroyo MJ, Lin T, Meshi A, et al. Identifying the factors for improving quality of oral fluorescein angiography. Br J Ophthalmol. July 4, 2019. [Epub ahead of print].

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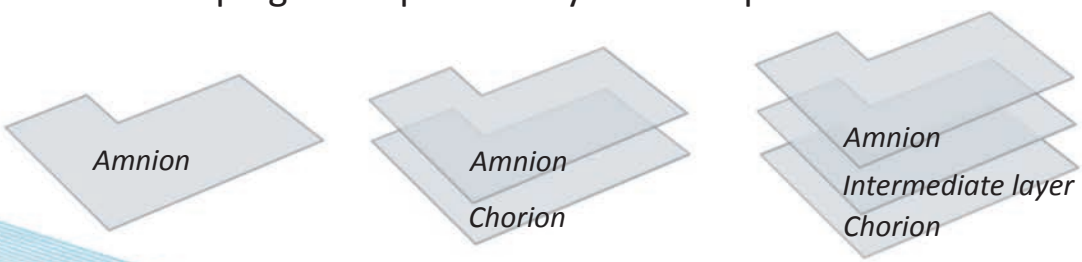

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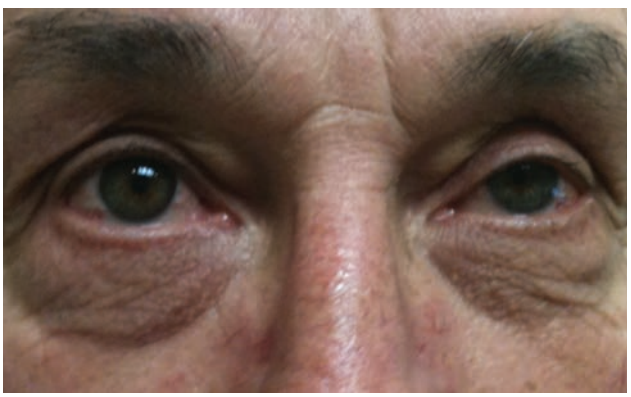

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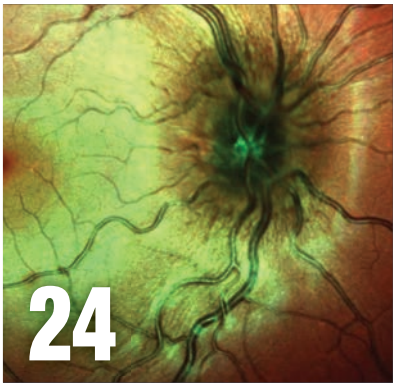

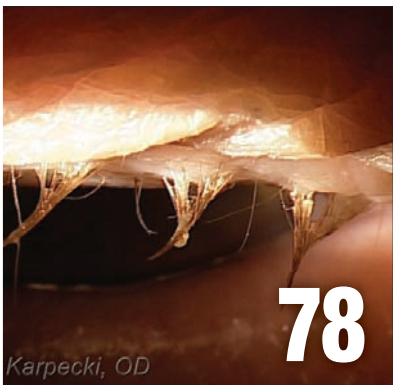

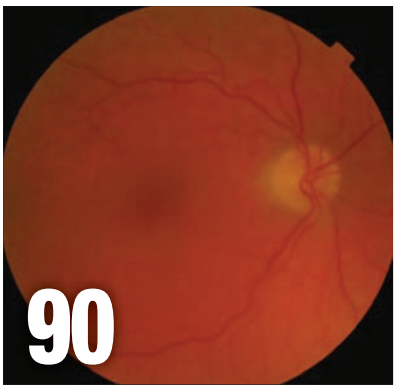

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1. Xiidra [Prescribing Information]. Lexington, MA: Shire US. **2.** TFOS DEWS II Research Subcommittee. Report of the Research Subcommittee of the Tear Film & Ocular Surface Society Dry Eye WorkShop II (2017). *Ocul Surf.* 2017;15(3):269-649. **3.** FDA approves new medication for dry eye disease. FDA News Release. July 2016. http://www.fda.gov/newsevents/newsroom/pressannouncements/ucm510720.htm. Accessed July 12, 2016. **4.** Food and Drug Administration. Electronic Orange Book. http://www.fda.gov/downloads/Drugs/DevelopmentApprovalProcess/UCM071436.pdf. Accessed June 26, 2018.

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Xiidra is contraindicated in patients with known hypersensitivity to lifitegrast or to any of the other ingredients in the formulation.

ADVERSE REACTIONS
Clinical Trials Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in clinical studies 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 five clinical studies of dry eye disease conducted with lifitegrast ophthalmic solution, 1401 patients received at least 1 dose of lifitegrast (1287 of which received lifitegrast 5%). The majority of patients (84%) had ≤3 months of treatment exposure. 170 patients were exposed to lifitegrast for approximately 12 months. The majority of the treated patients were female (77%). The most common adverse reactions reported in 5-25 % of patients were instillation site irritation, dysgeusia and reduced visual acuity. Other adverse reactions reported in 1% to 5% of the patients were blurred vision, conjunctival hyperemia, eye irritation, headache, increased lacrimation, eye discharge, eye discomfort, eye pruritus and sinusitis.

Postmarketing Experience
The following adverse reactions have been identified during postapproval use of Xiidra. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure.

Rare cases of hypersensitivity, including anaphylactic reaction, bronchospasm, respiratory distress, pharyngeal edema, swollen tongue, and urticaria have been reported. Eye swelling and rash have been reported.

USE IN SPECIFIC POPULATIONS
Pregnancy
There are no available data on Xiidra use in pregnant women to inform any drug associated risks. Intravenous (IV) administration of lifitegrast to pregnant rats, from pre-mating through gestation day 17, did not produce teratogenicity at clinically relevant systemic exposures. Intravenous administration of lifitegrast to pregnant rabbits during organogenesis produced an increased incidence of omphalocele at the lowest dose tested, 3 mg/kg/day (400-fold the human plasma exposure at the recommended human ophthalmic dose [RHOD], based on the area under the curve [AUC] level). Since human systemic exposure to lifitegrast following ocular administration of Xiidra at the RHOD is low, the applicability of animal findings to the risk of Xiidra use in humans during pregnancy is unclear.

Animal Data
Lifitegrast administered daily by intravenous (IV) injection to rats, from pre-mating through gestation day 17, caused an increase in mean preimplantation loss and an increased incidence of several minor skeletal anomalies at 30 mg /kg / day, representing 5,400-fold the human plasma exposure at the RHOD of Xiidra, based on AUC. No teratogenicity was observed in the rat at 10 mg /kg /day (460-fold the human plasma exposure at the RHOD, based on AUC). In the rabbit, an increased incidence of omphalocele was observed at the lowest dose tested, 3 mg /kg /day (400-fold the human plasma exposure at the RHOD, based on AUC), when administered by IV injection daily from gestation days 7 through 19. A fetal No Observed Adverse Effect Level (NOAEL) was not identified in the rabbit.

Lactation
There are no data on the presence of lifitegrast in human milk, the effects on the breastfed infant, or the effects on milk production. However, systemic exposure to lifitegrast from ocular administration is low. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for Xiidra and any potential adverse effects on the breastfed child from Xiidra.

Pediatric Use
Safety and efficacy in pediatric patients below the age of 17 years have not been established.

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Carcinogenesis: Animal studies have not been conducted to determine the carcinogenic potential of lifitegrast.
Mutagenesis: Lifitegrast was not mutagenic in the *in vitro* Ames assay. Lifitegrast was not clastogenic in the *in vivo* mouse micronucleus assay. In an *in vitro* chromosomal aberration assay using mammalian cells (Chinese hamster ovary cells), lifitegrast was positive at the highest concentration tested, without metabolic activation.
Impairment of fertility: Lifitegrast administered at intravenous (IV) doses of up to 30 mg/kg/day (5400-fold the human plasma exposure at the recommended human ophthalmic dose (RHOD) of lifitegrast ophthalmic solution, 5%) had no effect on fertility and reproductive performance in male and female treated rats.

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Outlook

By Jack Persico, Editor-in-Chief

Breaking Down Barriers

There's nothing special about multifocal and toric contact lenses. And that's a good thing.

When Henry Ford launched the Model T in 1908, he joked that it comes in any color you want, "as long as you want black." The Model T was the first mass-produced automobile, and the only way Ford could build it at high volume was to limit consumers' choices. Contact lenses had arrived 20 years prior, but wouldn't be mass-produced until the early 1970s. For more than 80 years, they were a highly customized product—what today might be called artisanal or bespoke. They were also highly fragile and fraught with problems. Practitioners had to be specialists to get it right.

Standardization made contact lenses accessible to millions. It also made this clinical service available to thousands of ODs who otherwise might not have had the background and motivation to offer it.

Fast forward to today. Contact lens fitting is under siege by online sellers who interrupt the doctor-patient relationship so they can swoop in with cheap (perhaps knock-off) products. Experts say it's not that hard to push back if you play up your skills as a contact lens specialist and offer specialty contact lenses that can't be easily substituted online.

This brings up two questions: what is a contact lens specialist, and what is a specialty contact lens? It's commonly accepted that a contact lens specialist is someone who can fit GPs, and lately sclerals, plus custom soft lenses—in addition to the mass-market soft lenses.

More problematic is the definition of a specialty lens. It feels like the terminology didn't keep pace with the technology. Standardization and decades of refinements by manufacturers have made toric and multifocal contact lenses closer to single vision lenses in fitting ease. Granted, they do have limitations that require compromise by the patient—just as Ford's Model T did—but that enables more people to benefit from them. Yet they continue to get the 'specialty' tag. Doing so perpetuates the belief that these are niche applications reserved for those doctors so enamored of contact lens practice as to call themselves specialists.

Enough already. There's no good reason why these shouldn't be mass-market lenses. There are millions of astigmats, presbyopes and even astigmatic presbyopes out there. To mentally cordon these people off as 'specialty lens patients', as so many still do, sets yourself up for failure and missed opportunities. The barrier to success here is an artificial one, more mental than practical. You wouldn't call a PAL or a single vision eyeglass lens with cyl 'specialty ophthalmic lenses', would you?

Those who receive our publication *Review of Cornea & Contact Lenses* will see that this month's issue is our annual compendium of every contact lens product on the market. We've given it a clean and colorful new look this year. We also grouped the lenses a little differently, breaking the soft lens listings into two main categories: *general use* and *special use*. We intentionally put into the general-use category a few lens modalities that some might be surprised to see there: you guessed it, torics and multifocals. Just go with it—Henry Ford would. ■

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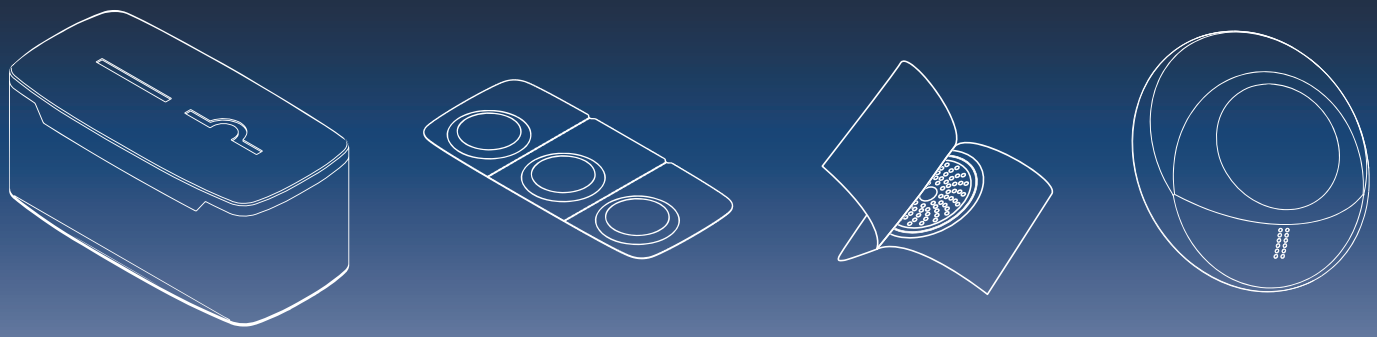

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Stay in the Fast Lane

Optometrists are in the driver's seat. Take some new opportunities for a spin.

By Paul M. Karpecki, OD, Chief Clinical Editor

Optometrists don't see themselves in a power position, but any profession that sees 88% of all comprehensive exams, in this case eye exams, wields considerable power. And with an election that will focus on healthcare, 2020 will be a critical year for optometry. It's time to embrace this position and use it to serve our patients—and our practices—better. A significant part of doing that is recognizing disruptors in the field and adapting to stay ahead of the inevitable changes.

Test Drive the New Model

Today, 120 million people are in their 40s, 50s and 60s, and more than 38% of them have significant astigmatism. Those 32 million patients need a multifocal contact lens with astigmatic correction, yet most of them believe they are not candidates for contact lenses because of presbyopia, astigmatism or both.

Bausch + Lomb's new Ultra Multifocal for Astigmatism contact lens now provides an easy in-office option. This lens provides a high DK/t (163), high water content (46%) and a high modulus (70). It has parameters from -6.00D to +4.00D, around the clock in 10° steps, three cylinder powers and two add powers. In clinical trials, 92% of patients said they could shift naturally from near to far throughout the day.

Upgrade Your Tech Package

Another valuable addition for your patients—and your bottom line—is in-office, patient-pay procedures. Until insurance covers these, doctors can set pricing to appeal to patients while also maintaining a healthy margin. One essential in-office treatment is BlephEx, because removing the biofilm from the lashes and lid margins for patients with meibomian gland dysfunction (MGD)/blepharitis can significantly improvement signs and symptoms. Many patients state that their eyes have not felt this good in decades.

Another important in-office procedure is thermal pulsation with LipiFlow (Johnson & Johnson Vision), iLux (Tear Film Innovations), TearCare (Sight Sciences) or, soon, Ocusoft's eyelid warming device. Intense pulsed light (IPL), with either the Eye-Light IPL (Lombart) or Lumenis's IPL, is another in-office option for most ODs. I recently purchased an Eye-Light IPL system and treated 50 patients with it in the first six weeks. We've been very impressed with its ease of use, the patient experience and the results. Most patients require only two treatments and I can see a significant improvement in telangiectatic vessels and overall inflammation in the eye. Patients have noticeable improvements in symptoms.

Many patients who have delayed treatment of their MGD or dry eye will require a combination of all three in-office procedures. When combined with dry eye therapies and at-home care, these procedures may solve the complex puzzle of dry eye for many patients.

Be a Co-pilot

Patients trust the provider they have seen for the last two, or 20, years more than someone they meet for 15 minutes before a procedure. That's why ODs should be integral to the IOL or MIGS selection process.

By the end of the year, RxSight will introduce the light-adjustable lens. I've seen the adjustment process first hand, and most patients with an adjustable light lens had uncorrected distance vision of 20/12 or better. We will be the doctors determining the optimal refraction post-surgery, whether that's with monovision, full distance correction in both eyes, astigmatism modification or, one day, presbyopic correction designs. Our involvement is key to alleviating the demand on surgeons, considering that the need for cataract surgery will exceed the supply of surgeons within the next seven years. That means we need to handle more of the care, including comanagement and, when we can, laser procedures. ODs can also look into working with surgeons with in-office surgical suites to offer more advanced procedures and help meet the growing needs of patients.

When you control the majority of all comprehensive eye exams and are more than 40,000 strong, you can be disruptive for the betterment of patients. There are many opportunities to choose from—make sure you aren't the one being disrupted. ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

More Lenses, More Problems

If only we could upgrade our patients the way we upgrade our contact lenses.

By Montgomery Vickers, OD

As the technologies in chemistry and plastics explode, contact lenses are becoming more comfortable, safer and more efficacious than ever before. Of course, it's just our luck that our patients have simultaneously devolved. In the older, more primitive lenses they were thrilled to see 20/40 and had no problem removing them after six hours to alleviate dryness and pain. Now, in the amazing new lens designs, they are apoplectic because the lenses don't make them 25 years old again and they missed one letter on the 20/15 line in their left eye.

Obviously, all of this is your fault, and they aren't afraid to tell you so.

Never Good Enough

Thus, we still see contact lens dropouts every day. I get it… it's really frustrating to have to put contact lenses in every day, even if they do let you do cool things like see. It's also way too much work to remove and discard them every day. That takes time away from what's important like Googling, "How tall is LeBron James?"

Plus, better technologies have increased the (perceived) cost to our patients. When I started wearing contact lenses in high school, my mom paid $300 for two lenses, and I was grounded for a month after I accidentally dropped them down the sink drain the first night. Now, they are ridiculously expensive, something like $2.00 for two lenses. To absorb such an absurd cost increase, I think the kids should pay for their own contact lenses by skipping one can of soda per day (I know, I know, that's really hard for the poor little souls, and no one should have to sacrifice like that).

For our older patients trying to read the game score on the phone hidden in their lap while pretending to care about their boss's quarterly performance presentation, bifocal contact lenses have created a new era of psychological pathologies. But they seem to work really well for patients who are motivated and understand they still beat wearing glasses at work. If only we could get them to stop driving to Louisiana at midnight in a hurricane while wearing them.

As I am now 66 years old, I can truly relate to the challenges of trying to see distance and near without glasses. I have been known to change my multifocal contact lenses two or three times in one day at the office in search of the Holy Grail of lens wear. Of course, hiding my age spots and dermatochalasis behind a cool pair of glasses may actually make a lot more sense for me.

A Taste of the Future

Meanwhile, contact lens manufacturers diligently march on. Lenses continue to improve with higher oxygen permeability, better optics and even UV protection. We even have lenses that darken in the sun, making us all look like cats after too much catnip. What's next, they taste like bubble gum and you just eat them when you're done to be environmentally friendly?

While you're at it, why not design a lens that knows the answers to a kid's SATs so we can keep their movie star parents out of jail? I don't want to pay more taxes so they can have caviar in Riker's.

Somewhere, deep in the bowels of a laboratory, some mad scientist is working on the perfect contact lens. You know… the one that will lead to world peace. I can't wait to see what it tastes like. ■

Red All Over

If certain glaucoma medications cause adverse effects, consider these options.

Edited by Paul C. Ajamian, OD

Q I have a patient who was put on Rhopressa (netarsudil, Aerie Pharmaceuticals) after other medications did not work. Her pressure lowered significantly, but six weeks later her eyes turned very red, and she's now mortified to go out in public. Lumify (brimonidine tartrate ophthalmic solution 0.025%, Bausch + Lomb) didn't help quiet the eyes. Is this a common problem?

A "I've personally had a fair amount of clinical success prescribing Rhopressa to treat glaucoma; however, ocular hyperemia does occur at some level in roughly 50% of patients who begin netarsudil 0.02% therapy," says Chris Wroten, OD, of Bond-Wroten Eye Clinic in Louisiana. As many as one in five patients experience conjunctival hemorrhage, and 5% to 10% manifest eyelid and/or instillation site erythema, so red eyes are not uncommon.[1]

"Although a number of patients do experience side effects such as these, most are mild and tolerable, so netarsudil is certainly a welcome addition to our glaucoma treatment arsenal," Dr. Wroten notes.

Two other side effects of note are corneal verticillata, which can occur in up to 20% of patients and is usually after four or more weeks of therapy, and blurred vision, which is reported in 5% to 10% of those taking netarsudil.[1]

Dr. Wroten has had three patients whose intraocular pressure (IOP) responded well to the medication but were forced to discontinue therapy due to significantly blurred vision.

While netarsudil therapy may be effective, patients can experience conjunctival hyperemia.

However, he has also had two patients with uncontrolled IOP on maximum topical glaucoma therapy who experienced a nearly 40% reduction in pressure when netarsudil was added—atypical cases.

"It will be interesting to see if tachyphylaxis develops because netarsudil was found to be most effective when IOP was below 26mm Hg at initiation of therapy," Dr. Wroten says.[2]

A New Hope

Given the side effects, Dr. Wroten had no choice but to stop the patient's medication. A week later, her eyes were dramatically quieter, but it will take time for the corneal changes to resolve. Because the patient also presented with cataracts, he determined that she would be a great candidate for minimally-invasive glaucoma surgery (MIGS) to manage her IOP in conjunction with cataract surgery.

MIGS devices and procedures that shunt and/or reduce aqueous production are becoming increasingly attractive options for patients. "MIGS should be considered as adjunctive therapy, especially when topical therapies have failed or are poorly tolerated," Dr. Wroten says.

In the hands of an experienced surgeon, they are quick and effective, with device instillation occurring immediately after the intraocular lens is inserted in the capsular bag.

If this patient was not a candidate for cataract surgery, then selective laser trabeculoplasty would be another attractive option. This procedure is well-tolerated and effective for about 80% of patients, lowering IOP by 20% to 30% for about 24 months on average.[2] It is also repeatable once the effect wears off, with similar therapeutic effect expected. More invasive filtering and valve surgeries were probably not warranted given her mild stage of glaucoma.

With our patient, Dr. Wroten had tried just about every medication, so the laser and surgical options were all that were left. He controlled her IOP at 12mm Hg after the iStent Inject (Glaukos) was put in, and she is only on a beta blocker now.

"Fortunately, with today's diagnostic technologies and treatment options, patients with glaucoma have a far better prognosis than ever before," Dr. Wroten says. ■

1. Rhopressa. Prescribing Information. rhopressa.com/assets/pdf/RHOPRESSA-Prescribing-Information.pdf. Accessed July 3, 2019.
2. Khouri AS, Lari HB, Berezina TL, et al. Long term efficacy of repeat selective laser trabeculoplasty. J Ophthalmic Vis Res. 2014;9(4):444-8.

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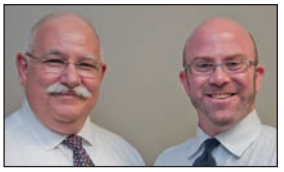


How Much is Too Much?

Many patients are over-plussed at near. With these simple strategies, you can learn how to avoid this. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

A question that often arises is how to determine bifocal power for patients. We all learned about the tables and charts that depict the loss of accommodation with age in optometry school. Presbyopia is in full force by the time most patients reach 40, and by the age of 70, everyone needs a +2.50 add at near, right? Not so fast. Here, we will discuss how our method of using ranges and showing them to our patients in varying real-life conditions helps us tailor lenses to their specific needs.

This patient is completing range testing with an iPad as his target.

The Problem With Too Much Plus

It turns out that, based on age alone, many patients get too much plus at near. Over time, these patients show an increase in hyperopia at distance well beyond the +0.50 or +0.75 they measured for many years. The period between being prescribed full plus at near based on age and developing increasing hyperopia at distance varies from three to five years, and many patients go through it.

When looking at progressive myopia, many believe the problem begins at near and spreads to distance. Sustained close work causes a type of form deprivation in the periphery, which, when combined with peripheral defocus, seems to be the driving force behind axial length changes in the eye. The blur at distance is just a side effect of the close work. Without the close work causing peripheral defocus and form deprivation, the eye wouldn't change the way it does or experience myopia that progresses faster than normal.

Too much plus at near also sets up a situation where patients are not rewarded for supplying their own accommodation. If they have too much plus at near and do not like looking or having to look closely at things to see them, they learn to inhibit accommodation or, put more blatantly, start to get lazy. The "zero-position," or the dark focus point of the whole system, shifts outward, which, over time, can manifest as an increase in the hyperopia at distance. When left unchecked for long enough, this can cause patients to become dependent on some plus for clarity and relaxation at distance.

How to Avoid Too Much Plus

The development of many different near retinoscopy techniques—including book, bell, MEM, stress-point and just-look retinoscopy—made it possible to steer clear of giving too much plus at near. The pioneers of our profession looked at different aspects of the retinoscopic reflex in relation to the demands patients experience and the targets they look at. They discovered that there is an optimum amount of plus and that too little or too much can cause short- and long-term problems for patients.

Another test we all learned in optometry school is the fused cross-cylinder (FCC) test. It's problematic in that about 25% of patients don't see a difference between the vertical and horizontal lines when the test is done in the traditional way. Some phoropters, however, can flip the Jackson cross cylinders used to administer the test to an alternate orientation, making it possible to obtain

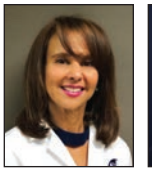
a clear endpoint for nearly all patients. But, modifying how we arrive at these endpoints still does not give us a foolproof way to come up with add powers that aren't too high. FCC test results and negative relative accommodation and positive relative accommodation findings only give us a framework within which we can come up with add powers for our patients.

Stress-point retinoscopy was then developed to come up with the optimum plus lens for near. The technique is very difficult for many to master and requires the use of a spot retinoscope. Though a new spot retinoscope is now on the market, the technique still remains difficult to grasp. When used correctly, it yields an optimum amount of plus that the patient can use successfully immediately without risk of rejection. Often, the plus power a patient receives for near from the stress point retinoscopy method is less than the FCC test.

If you don't know how to do stress-point retinoscopy or you don't have a spot retinoscope, trial framing the plus to see if it is clear at a patient's habitual or desired reading distance and to measure ranges for the lenses over which the patient can see clearly is another option. Give the patient a target, such as a computer or phone screen, that resembles something they normally look at. Have them bring it closer to them until it begins to blur, and then have them push it further from them until it begins to blur. This establishes the range over which the lenses will work and sets the stage for a discussion about the reduced depth of field of focus and the loss of accommodation amplitude that occur with age. Our patients want one lens to do it all and everything to be perfectly clear all time, regardless of where the demand is in their visual field. It's up to us to help them achieve that, with whichever method we decide to use.

We have found that many people prefer sacrificing a bit of the very close range for big extensions in the back end of the range, which is less plus for near, and self-select this option when given the chance to see and understand their choices.

It may be beneficial to determine the add power you would normally give with your current method of prescribing and then obtain the range measurements with the technique we suggested to give your patients two options and see what they prefer. You may find that less is best and stabilizes your patient's distance refractions over time. ■




Caution: Congestion Ahead

Use your diagnostic toolkit to navigate the crucial distinction between true and pseudopapilledema. **By Jay M. Haynie OD, Diana Shechtman OD, and Rashid Taher, MD**

When a patient has an elevated optic nerve head (ONH) with blurred disc margins, alarm bells should go off. These could be harbingers of true papilledema caused by an underlying systemic process or mass lesion in need of urgent treatment.

But these signs could also be pseudopapilledema. Optic nerve head drusen (ONHD), the most common etiology of pseudopapilledema, are found in approximately 1% of the population with bilateral distribution in 75% to 85%.[1-3] Although patients with benign ONHD are often asymptomatic, the drusen can cause elevation, congestion and blurred margins of the optic nerve, simulating papilledema.

Patients with ONHD are often asymptomatic, leading to a high rate of incidental discovery. Visual inspection of an elevated ONH is often insufficient to confirm pseudopapilledema and rule out a more serious diagnosis. Thus, a multitude of tests—including radiologic neuroimaging and a spinal tap—are warranted to rule out an intracranial mass or lesion and to assess for increased intracranial hypertension. Many other useful diagnostic tests are available in the optometric setting to assess ONHD, including spectral domain OCT (SD-OCT), fundus autofluoresence (FAF), intravenous fluorescein angiography (IVFA), OCT angiography (OCT-A) and B-scan ultrasonography.

SD-OCT can be quite helpful in differentiating pseudopapilledema from true papilledema, as the latter will show nerve fiber layer edema on SD-OCT imaging, which may cast a shadow on the structures below (*Figure 1*). ONHD, on the other hand, will be hyper-reflective on SD-OCT.

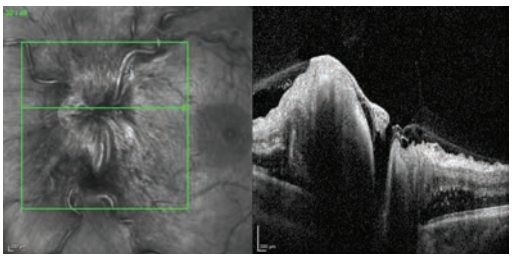


Fig. 1. SD-OCT of a patient with papilledema shows the shadow or darkening of the deeper structures.

Blurry Signs

Cases by Dr. Haynie

Case 1. An 11-year-old Caucasian female with an elevated ONH was referred to rule out papilledema. She had no visual complaints and denied headaches. Her visual acuities measured 20/20 OU, intraocular pressures (IOPs) measured 15mm Hg OD and 14mm Hg OS, pupil assessment was normal without afferent pupillary defect (APD) and her body mass index (BMI) was 21.0. Her anterior segment examination was unremarkable, but her dilated examination revealed a congested ONH in each eye with blurred disc margins (*Figure 2*). We then performed SD-OCT, FAF and B-scan ultrasonography (*Figure 3*), which helped us diagnose her with buried ONHD and advise annual follow-up.

ONHD are generally seen well with FAF; however, in younger patients, as in this case, the drusen deposits are more posterior and may not been seen until later in life.

Case 2. A 37-year-old Hispanic woman was referred by her neurologist for an evaluation of optic nerve edema. She was diagnosed with papilledema by a local ophthalmologist

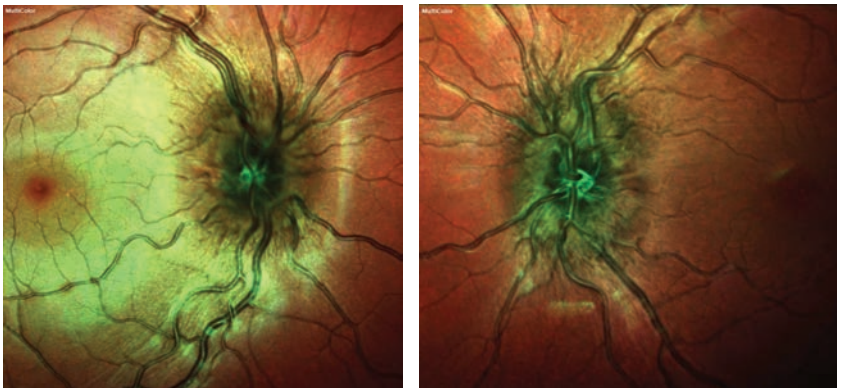


Fig. 2. Color fundus images reveal crowded elevation of the ONH with indistinct margins in both eyes.

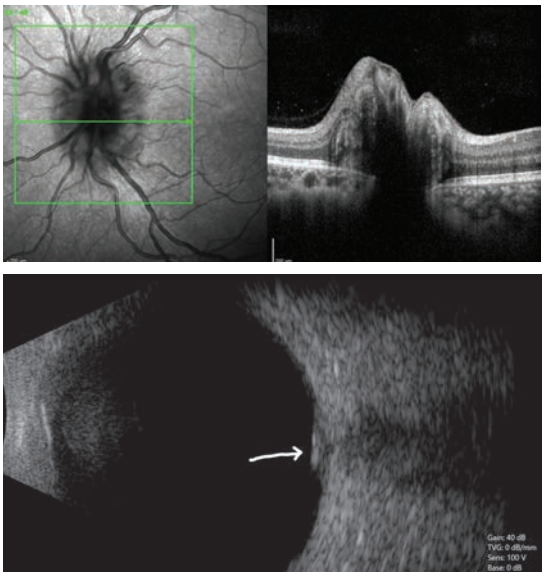


Fig. 3. SD-OCT, top, and ultrasonography, bottom, show hyper-reflective lesions deep in the ONH consistent with drusen. Findings were similar in both eyes.

who then referred her to neurology. However, her radiologic neuroimaging and lumbar puncture were both normal, and the neurologist referred her for a retinal consultation. Her chief complaint was headaches, which were later attributed to probable migraine. Her past medical history was negative. Her visual acuity measured 20/20 OU, IOPs measured 20mm Hg OD and 21mm Hg OS, pupil assessment was normal without APD and her BMI was 27.0. Her anterior segment examination was unremarkable, but dilation revealed congested ONHs with blurred disc margins. Her IVFA was normal without late leakage as we would expect with papilledema. Her SD-OCT and FAF images confirmed the diagnosis of ONHD (*Figures 4 and 5*).

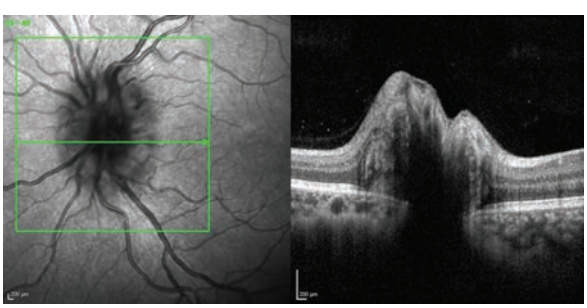


Fig. 4. SD-OCT of the left eye shows hyper-reflective lesions deep in the ONH consistent with drusen. The right eye shows similar findings.

A Diagnostic Roadmap

Commentary by Dr. Shechtman and Dr. Taher

Although no set protocols exist for the assessment of pseudopapilledema vs. true papilledema, a comprehensive eye examination with a dilated fundus evaluation and the use of various diagnostic modalities can be quite valuable when distinguishing between the two (*Table 1*).

We have found that IVFA is particularly useful in distinguishing papilledema from pseudopapilledema by revealing ONH leakage. On ultrasonography, another helpful diagnostic tool, drusen appear as hyper-reflective calcified bodies in the optic nerve and will continue to show increasing brightness even at a low gain. Other ancillary testing such as OCT, visual fields and FAF can also be helpful. SD-OCT of ONHD can reveal an elevated disc with a characteristic "lumpy-bumpy" appearance. A nerve with true papilledema, however, may reveal a smooth internal contour of the ONH with a characteristic hyporeflective "V" pattern in the subretinal


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Table 1. ONHD vs. True Papilledema

Features	ONHD	True Papilledema
Visual symptoms	Transient vision loss and visual field defects can occur	+/- transient vision loss, double vision and visual field defects (typically an enlarged blind spot)
Headaches	Not associated with ONHD	If present, often described as worse upon awakening and/or with postural changes
Neurological symptoms	None	Tinnitus, vertigo, nausea/vomiting, peripheral neuralgias
Optic nerve appearance	Elevation confined to disc	Elevated swollen nerve, hyperemia, peripapillary vessel obscuration; +/- flame shaped hemorrhages, cotton wool spots, +/- Paton's lines; SVP absent
Vasculature	Anomalous branching pattern	Microvascular dilation

space adjacent to the ONH. Although these are distinct findings, SD-OCT alone should not be used to distinguish between the two conditions. Furthermore, because the calcific properties of drusen have inherent autofluorescent ability, ONHD will show hyperfluorescence on FAF. Drusen autofluorescence is inversely proportional to its depth, meaning deeply buried drusen may be difficult to assess. Additionally, if the cause of pseudopapilledema is not ONHD, FAF would show unremarkable findings.

Although ONHD are often found in isolation, they may be associated with other ocular findings, such as angioid streaks, that correlate with underlying conditions. Additionally, lowering IOP in patients with ONHD can help prevent progressive optic neuropathy and slow visual field loss, but ONHD are dynamic and can cause shifts of the refractile bodies at any time.

In any case where the diagnosis of pseudopapilledema is not confirmed, referral to a neuro-ophthalmologist is warranted. True papilledema requires an immediate referral for radiological imaging, such as MRI or CT MRA/MRV, followed by lumbar puncture. ■

1. Auw-Haedrich C, Staubach F, Witschel H. Optic disk drusen. Surv Ophthalmol. 2002:47(6):515-32.
2. Erkkila H. Clinical appearance of optic disc drusen in childhood. Albrecht Von Graefes Arch Klin Exp Ophthalmol. 1975;193(1):1-18.
3. Mehrpour M, Torshizi F, Esmaeeli S, et al. Optic nerve sonography in the diagnostic evaluation of pseudopapilledema and raised intracranial pressure: A cross-sectional study. Neurology Research International. 2015:Article ID 146059.

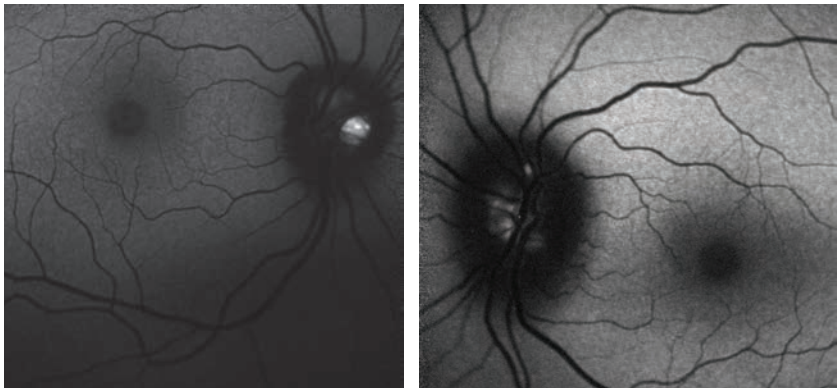

Fig. 5. FAF confirmed focal hyperautofluoresence of the ONH consistent with lipofuscin (ONHD) in both eyes.

Not Covered? No Problem

Financial waivers of liability are the key to getting paid in today's world.

By John Rumpakis, OD, MBA, Clinical Coding Editor

Today's healthcare system is a fickle beast, and the challenges of navigating the payor system can be overwhelming. But you can always get paid for what you do. If you ever write a charge off to a zero balance, you are not taking full advantage of the rules that exist to get paid. Consult with your billing department and spot check your EOBs to make sure they aren't writing off patient balances. This is where understanding the rules that govern the financial waivers of liability is crucial.

Paperwork

A financial waiver of liability is an informed consent document you use when you expect a patient's insurance will not cover a procedure or durable medical equipment (DME) such as eyeglass frames and lenses. The document informs the patient prior to a procedure being performed or materials ordered that they may be financially liable for the costs should the carrier deny the claim. The patient must provide consent by signature and accept financial responsibility for you to proceed. The carrier can then legally and properly transfer the financial liability to the patient, so you don't have to write off a balance that you had a right to collect.

The form you use is incredibly important, as failure to use the right one prevents you from collecting from the patient should a claim be denied. Medicare Part B requires an advanced beneficiary notice (ABN) form, and you must use Medicare's specific form, found at www.cms.gov/medicare/medicare-general-information/bni/abn.html. Medicare Advantage Plans (Medicare Part C) have their own forms with distinct and separate rules. For commercial payors, the Medicare Part B ABN form works if you remove the word *Medicare* and substitute "your medical insurance carrier." This allows you to properly submit claims to any commercial medical carrier and preserve your payment rights.

Modifiers

Once the patient completes the ABN, add it to the patient record. Notify the carrier on a claim-by-claim basis that you have a completed ABN form by using modifiers on the specific procedure or DME in question. Four common modifiers can be appended to the CPT codes—either as a requirement by Medicare or voluntarily:

Modifier GA: *ABN issued as required by payer policy, individual case.* This is used to report that a required ABN was issued and filed for a service. CMS will assign financial liability to the beneficiary should the services be denied.

Modifier GX: *Notice of liability issued, voluntary under payer policy.* This reports that a voluntary ABN was issued for a service that is statutorily excluded from Medicare reimbursement. Medicare will reject non-covered services appended with GX and assign liability to the beneficiary. Since this is a voluntary ABN, the patient always has financial responsibility for the procedure or test being conducted.

Modifier GZ: *Item or service expected to be denied as not reasonable and necessary.* This reports that an ABN was not issued. CMS will automatically deny these services and indicate the beneficiary is not responsible for payment. Without an ABN prior to performing the service, you cannot bill the patient.

Modifier GY: *Item or service statutorily excluded or does not meet the definition of any Medicare benefit.* This reports when a service is specifically excluded by Medicare and an ABN was not issued. CMS will deny these claims and the beneficiary will be totally responsible for all financial liability.

Modifiers GA and GZ are often used if a procedure doesn't meet medical necessity as determined by a Medicare local or national coverage determination. Modifiers GX and GY are for items or services statutorily excluded from the Medicare program. Here, an ABN is optional, but provides proof the beneficiary understands he will be liable for payment. When using either modifier, the provider should bill the patient for the services provided.

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The Optometrist and Obstructive Sleep Apnea

Learn to keep your snoring patients' increased risk of disease from keeping you up at night. **By Susan Kovacich, OD**

Although several types of sleep-disordered breathing exist, obstructive sleep apnea (OSA) is certainly the most common and the most publicized.[1] The condition is caused by a complete or partial anatomical upper airway collapse that temporally restricts or obstructs breathing, often in a cyclical pattern. The reduction of breathing is hypopnea, the cessation of breathing is apnea. This respiratory disruption reduces blood oxyhemoglobin saturation and impacts blood pressure, heart rate, sympathetic activity, metabolic activity and sleep. It elevates an individual's risk for hypertension, coronary artery disease, myocardial infarction, congestive heart failure and stroke.

OSA patients may experience cognitive dysfunction, depression and the sleep disturbances may even trigger metabolic syndrome and lead to diabetes.[2,3] Additionally, the risk for gout is increased by two times in older patients and OSA is a risk factor for dislipidemas.[3,4] As you can see, OSA and the disordered sleep it causes can have a deleterious impact on literally every part of the human body, and the eyes are no exception. In fact, OSA is associated with several ocular conditions, from the anterior to posterior segment and impacting a wide range of structures from the tear film to the optic nerve.

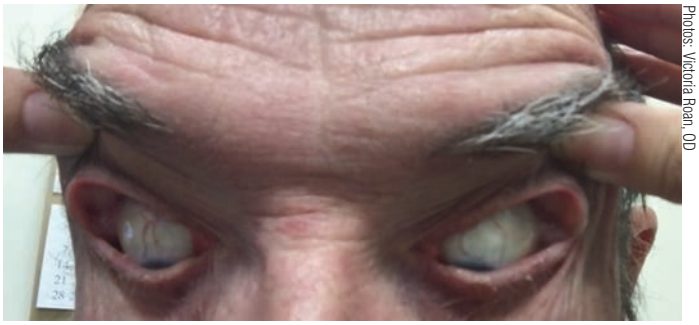
Vogt's striae, as seen here, can indicate keratoconus, which is more common in patients with OSA than those without.

Here, we review the risk factors for OSA, how to recognize and diagnose an OSA patient and how the optometrist can comanage these patients, with special attention to its associated ocular diseases.

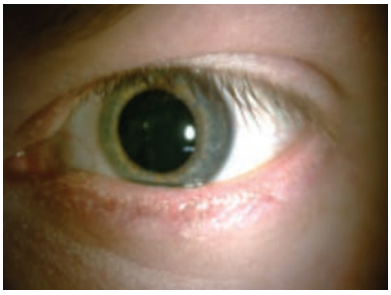
Diagnosis

The main risk factor for OSA is obesity (which can double the risk), specifically a neck circumference greater than 17 inches.[2,5,6] It is more prevalent in males, with females catching up following menopause. Aging, craniofacial deformities, smoking and alcohol use before bed are also associated with OSA.[2] Most patients with OSA have no problems breathing while awake, but during sleep, muscle tone relaxes and soft tissue in the pharynx collapses to obstruct the airway. It's the obesity that increases the soft tissue (adipose) around the pharynx combined with a decrease in muscle tone with age—OSA is two to three times more prevalent in patients older than 65.[2] While a complex neurochemical feedback mechanism exists to promote breathing when blood CO_2 rises, the stimuli is insufficient to override the soft tissue obstruction when the airway is compromised.

The impaired feedback mechanism will result in imprecise ventilation undershoots and overshoots with sudden neurological arousals to promote breathing which disrupt the sleep cycle.[7] While questionnaires can be helpful for screening, OSA is formally diagnosed with polysomnography, or a continuous overnight sleep study, where the patient's breathing, heart rate, brain waves, blood oxygen levels and other parameters are monitored.[2,6]



Photos: Victoria Roan, OD



Patients with OSA may experience a variety of lid and lash conditions such as lash ptosis, at left, and floppy eyelid syndrome, which is characterized by superior lids that are easily everted with minimal lateral force, as shown above.

Sleeping with OSA is characterized by loud snoring, periods of not breathing, followed by gasps when breathing resumes.[2] People who sleep alone may be unaware of their breathing disorder, and may become habituated to the daytime sleepiness and fatigue that results from fragmented sleep. People in relationships are more likely to be diagnosed with OSA as they are more likely to be informed of their sleep breathing disorder (SBD).[2] Even so, approximately 78% to 80% of the OSA population who could benefit from treatment go undiagnosed.[2] Furthermore, as the American population becomes more obese, the rate of sleep disordered breathing in the American population continues to rise. One study using data from 2007 to 2010 estimates that the rate of SBD in American adults aged 30 to 70 is 14% in men and 5% in women with symptoms that meet the Medicare criteria for OSA, which is a significant increase compared with data collected from 1988 to 1994.[6]

Ocular Associations

OSA is associated with several eye conditions. In 2005, investigators reported an association with floppy eyelid syndrome (FES), primary open angle glaucoma (POAG), normal-tension glaucoma (NTG), optic neuropathy, nonarteritic anterior ischemic optic neuropathy (NAION) and papilledema with raised intracranial pressure (ICP).[8] Additional research shows an association with keratoconus and an increase in diabetic retinopathy (DR), especially proliferative diabetic retinopathy (PDR) as well as central serous retinopathy.[9-11] Research shows OSA's impact on an array of structures within the eye, including the lids, the cornea, the optic nerves, and the retina.

Anterior segment and lids. In a population of patients with keratoconus, 18% had previously been diagnosed with OSA with sleep testing and an additional 47% of patients at high risk for developing OSA, according to a 2012 study.[9] Of note, the average BMI of the patients in this study was 31.2.[9]

Another study focused on FES, a condition characterized by an elastic-like upper eyelid that is easily pliant and everted during sleep or manually with minimal lateral traction. The study found 16 out of 17 patients with OSA had FES.[8] This high correlation suggests that every patient with OSA should have their lids everted to check for FES related papillary conjunctivitis.

Any patient diagnosed with FES who has not been diagnosed with OSA should be referred to their primary care physician for a workup, or a cardiologist, neurologist, or pulmonologist if the patient is currently under their care. The rubbery tarsal plate in FES fails to support the eyelid platform properly, with the eyelashes pointing downwards over the visual axis, making the presence of lash ptosis a red flag.[12] Differential diagnosis includes other types of chronic papillary conjunctivitis. FES patients tend to be obese males with OSA.

For any acute corneal or conjunctival insult, eye care providers may apply an ophthalmic ointment, such as erythromycin, and switch to a lubricating ointment when the lesions resolve. Protecting the eyes during sleep by taping the eyelids or using a tightly-fitting eye mask or shields may be indicated. For severe cases, surgical tightening of the eyelids is an option.[13]

Optic nerves and glaucoma. Researchers believe OSA can damage the vascular and mechanical

Ocular Conditions Associated with OSA
Keratoconus (KC)
Floppy eyelid syndrome (FES)
Nonarteritic anterior ischemic optic neuropathy (NAION)
Papilledema with raised intracranial pressure/ Idiopathic Intracranial Hypertension (IIH)
Primary open angle glaucoma (POAG)/Normal tension glaucoma (NTG)
Retinal vascular disease (retinal vacular occlusion (RVO), diabetic retinopathy (DR))
Central serous retinopathy (CSR)

structures within the second cranial nerve. Among other factors, the recurrent impaired nocturnal vascular perfusion caused by OSA may damage the optic nerve, while raised nocturnal intracranial pressure and elastic fiber depletion can cause mechanical damage.[14]

NTG patients frequently have OSA issues, according to one study showing that a large percentage of middle aged or older NTG patients test positive for OSA with polysomnography. Researchers hypothesize that impaired autoregulation of optic disc circulation results in nerve damage.[15] NTG is more highly associated with and OSA than with POAG.

NAION patients with OSA experience nocturnal hypoxia, which may result in episodic hypotension. Also, the nocturnal hypoxia may directly damage the optic nerve. Investigators point to an exceptionally high association with NAION and OSA, with 70% of NAION patients having OSA. When referring NAOIN patients, be sure to mention the association with OSA.[16]

Papilledemas can include pseudopapilledema, papillitis, ischemic optic neuropathy and hypertensive optic neuropathy among others. All involve optic disc swelling caused by raised intracranial pressure (ICP). Since papilledema can sometimes be caused by an intracranial

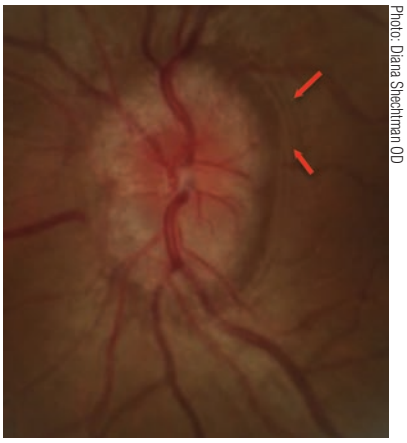


Photo: Diana Shechtman OD

This pediatric patient's papilledema is evident due to the microvascular dilation of the optic nerve, vessel obscuration and Paton's line (red arrows). Papilledema is associated with OSA.

mass, emergency cranial imaging is indicated.[17] The nocturnal hypoxia caused by OSA changes the cerebral vasculature which itself is associated with elevated ICP.[18]

Idiopathic intracranial hypertension (IIH), also called pseudotumor cerebri, is a condition that primarily impacts females with obesity. It is also associated with OSA and disordered sleep.[19] Signs include increased ICP, normal brain imaging and increased opening pressure on lumbar puncture with normal cerebral spinal fluid findings and an enlarged blind spot. Symptoms for patients with IIH and elevated ICP are similar to the symptoms of those with papilledema with the addition of pulsatile tinnitus. Acetazolamide is often prescribed if there is no sulfa allergy, whether the patient is symptomatic or not.[20] The *Journal of Clinical Sleep Medicine* reports on one patient treated with both acetazolamide and CPAP who showed resolution of papilledema.[21] Another study suggested that using the Berlin questionnaire would be a practical tool to direct IIH patients at high risk for OSA for polysomnography.[22]

Retina and vasculature. Retinal vascular occlusion (RVO) includes sudden, painless vision loss that is usually unilateral, with visual field defects. Retinal hemorrhages and dilated, tortuous veins are often seen.[23] Symptoms of RVO usually manifest upon waking similar to NAION, suggesting microvascular and hypercoagulable changes during nocturnal apnea events. Research shows that patients with RVO often have OSA, and OSA is now considered to be a risk factor for RVO events.[1]

Diabetic retinopathy (DR). The hyperglycemia caused by diabetes leads to oxidative stress and damage to the vascular endothelium, resulting in permeable blood vessels.[24] Early nonproliferative diabetic retinopathy (NPDR) is characterized by retinal hemorrhages and microaneurisms. The leaky blood vessels allow the deposition of hard exudates (lipids) and fluid in the macula resulting in diabetic macular edema (DME) and vision loss. Progressive capillary nonperfusion resulting in ischemia will promote the formation of PDR which is characterized by the proliferation of neovascularization on the surface of the retina or optic disc. PDR can result in vitreous hemorrhages, fibrosis and tractional detachments.[25] In a study of diabetic patients who already

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Systemic Conditions Associated with OSA

Comorbid condition	Prevalence of OSA
Congestive heart failure	11% to 37%[1]
Stroke and transient ischemic attack	43% to 72%[1]
Development of hypertension	42%[2]
Gout	50%[3]

1. Young T, Skartrud J, Peppard P. Risk factors for obstructive sleep apnea in adults. JAMA. 2004;291(16):2013-6.
2. Peppard P, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med. 2000;342(19):1378-84.
3. Zhang Y, Peloquin C, Dubreuil M, et al. Sleep apnea and the risk of incident gout. Arthritis and Rheumatology. A population-based, body mass index-matched cohort study. 2017;67(12):3298-302.

had DR, PDR was much more prevalent in patients with OSA.[10] Vascular endothelial growth factor (VEGF) is sensitive to hypoxia and responsible for new blood vessel production.[26] Nocturnal desaturation and reoxygenation caused by OSA was thought to be directly related to the increased development of PDR in these patients.[10] The management of diabetic retinopathy includes tight glycemic control, panretinal and focal laser photocoagulation, anti-VEGF therapy and vitrectomy.[25,27]

Central serous retinopathy (CSR) is an idiopathic serous retinopathy characterized by blurred vision, metamorphopisia and a central blind spot. The patient presents with a serous detachment of the neurosensory retina in the macula. The classic patient profile is a male between the ages of 25 and 50 who is stressed, which is why an association with cortisol is suspected.[28] The constant interruption of the sleep cycle in OSA affects the sympathetic system and promotes an increased production of circulating norepinephrine and epinephrine which is thought to increase vascular permeability leading to serous fluid leakage.[11]

Systemic Treatment

Before 1981, the only treatment for OSA was a tracheostomy, a surgically created hole with a tube leading through the front of the neck and into trachea, created by a tracheotomy.[2] This invasive procedure adversely affects the ability to speak and impacts eating. The patient is also burdened by the care of the "trach tube" to prevent clogging and infection.[29]

All of this changed when the continuous positive airway pressure (CPAP) ventilator was developed, which is now considered standard in OSA treatment.[30] A machine provides constant airflow, and the patient wears a mask over the nose or mouth or both to direct the flow through the airway during sleep. This continuous pushing of air through the trachea keeps the airway open and prevents the airway collapse experienced in OSA. Research shows that CPAP usage reduces airway obstructions and improves sleep in patients with OSA, reducing daytime sleepiness and other systemic impacts.[31]

CPAP usage is not without its own issues, as some patients cannot tolerate wearing a mask during sleep or develop a dry nose or other issues. CPAP usage can also have deleterious effects on the eyes, also, which will be discussed later. Other OSA treatments include mouth guards or splints which widen the airway by pushing the jaw forward and advancing the tongue, but these are not as effective as CPAP. Reconstructive surgery to the upper airway to improve airflow has also been performed, but the effectiveness of such procedures is not well studied.[31]

CPAP usage needs to be long-term which is impacted by poor adherence by users. The benefits of extended usage cannot be ignored—CPAP usage can reduce snoring and nocturnal awakenings which improves sleeping.[32] The associated reduction in daytime sleepiness has also been associated with a decrease in motor vehicle accidents attributed to OSA.[32] CPAP usage seems to have its largest impact on cardiovascular outcomes and hypertension. Metabolic syndrome and hyperlipidemia is also improved.[32] The sleep fragmentation in OSA seems to primarily affect attention/vigilance while hypoxia is linked to global cognitive function.[33] A meta-review of CPAP treatment on cognitive function showed medium to large improvements in five subcategories of executive function.[34]

Due to the strong association with obesity, lifestyle changes are encouraged for those patients with OSA who are obese—defined as any body mass index (BMI) of greater than 30. While diet and exercise leading to weight loss can help reduce the severity of OSA, it is thought that weight reduction alone may be insufficient to reverse the condition.[35] Cessation of smoking along with the reduction of nighttime alcohol use are also encouraged.[31]

CPAP Impact

If the ocular impacts of OSA weren't enough, CPAP usage also can have a deleterious effect on the eye. Air blown into the eyes from poor fitting CPAP masks has long

been known to cause ocular dryness. Conjunctivitis resulting from CPAP use was reported as early as 1984.[36] One paper identified anterior segment problems such as dryness and recurrent corneal infections observed in contact lens wearers being treated with CPAP.[37]

Improvements in CPAP mask fitting can reduce air leakage, and the importance of keeping the CPAP disinfected is better understood, but dryness can still result from air being pushed up through the nose into the nasal lacrimal canal, especially with those who have nasal lacrimal tubes.[38,39] If improvement in dryness cannot be attained with changes in the mask fit, punctal plugs may be used to help with air reflux.[40] In addition, besides the aforementioned association of OSA with glaucoma, CPAP use is also linked to elevated intracocular pressure, making the monitoring of OSA patients who are glaucomatous or glaucoma suspects who are treated with CPAP even more critical.[41]

OSA is a prevalent condition that is often underdiagnosed and that has serious health implications. Pay special attention to patients who have keratoconus, FES, lash ptosis, papilledema and vascular issues. Question those known to have OSA about their treatment protocol, especially if they're using a CPAP. Patients are not always forthcoming about this and may not be aware of its potential impact on their eyes. Ask them about any dry eye issues and consider testing them for ocular dryness. Unusual or unexplained recurrent bacterial conjunctivitis could also be a possible indicator of CPAP complications.

The optometrist is well positioned to be alert to both ocular and systemic conditions that may be related both to OSA and CPAP usage and be able to make a timely referral to the patient's primary care physician or appropriate managing specialist for diagnosis and treatment. ■

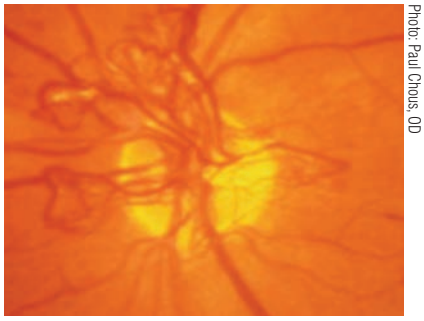

Photo: Paul Chous, OD

Patients with concurrent diabetes and OSA who already have diabetic retinopathy are more likely to progress to proliferative diabetic retinopathy, as seen here, than those without OSA.

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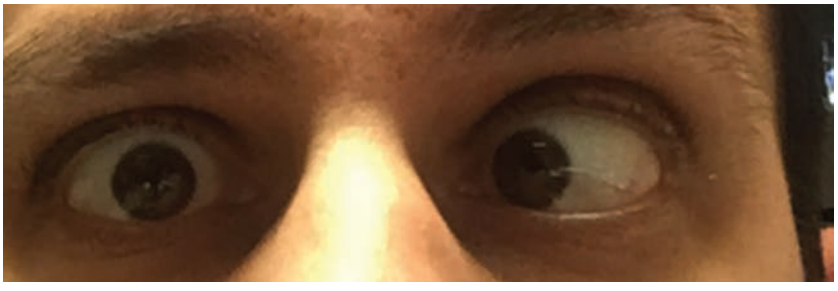
1. Santos M, Hofmann J. Ocular manifestations of obstructive sleep apnea. J Clin Sleep Med. 2017;13(11):1345-8.
2. Young T, Skartrud J, Peppard P. Risk factors for obstructive sleep apnea in adults. JAMA. 2004;291(16):2013-6.
3. Singh J, Cleveland J. Gout and the risk of incident obstructive sleep apnea in adults 65 years or older: an observational study. J Clin Sleep Med.2018;14(9):1521-7.
4. Karkinski D, Georgievski O, Dzekova-Vidimliski P, et al. Obstructive sleep apnea and lipid abnormalities. Open Access Maced J Med Sci. 2017;5(1):19-22.
5. Davies R. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apneoa syndrome. European Respiratory Journal. 1990;3(5):509-14.
6. Peppard P, Young T, Barnet, et al. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol. 2013;177(9):1006-14.
7. Dempsey JA, Veasey S, Morgan B, O'Donnell C. Pathophysiology of sleep apnea. Physiol Rev. 2010;90(1):47-112.
8. McNab A. The eye and sleep. Clin Exp Ophthalmol. 2005;33(2):117-25.
9. Gupta P, Stinnett S, Carlson A. Prevalence of sleep apnea in patients with keratoconus. Cornea. 2012;31(6):595-9.
10. Shiba T, Sato Y, Takahashi M. Relationship between diabetic retinopathy and sleep-disordered breathing. Am J Ophthalmol. 2009;147(6):1017-21.
11. Huon LK, Liu SY, Camacho M, Guilleminault C. The association between ophthalmologic diseases and obstructive sleep apnea: a systematic review and meta-analysis. Sleep Breath. 2016;20(4):1145-54.
12. Schlötzer-Schrehardt U, Stojkovic M, Hofmann-Rummelt C, et al. The pathogenesis of floppy eyelid syndrome: involvement of matrix metalloproteinases in elastic fiber degradation. Ophthalmol. 2005;112(4):694-704.
13. Floppy Eyelid Syndrome. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed, Bagheri N, ed. Philadelphia: Wolters Kluwer; 2017:136-7.
14. Perez-Rico C, Gutierrez-Diaz E, Mencia-Gutierrez E, et al. Obstructive sleep apnea-hypoopnea syndrome (OSAHS) and glaucomatous optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2014;252(9):1345-57.
15. Mojon D, Hess C, Goldblum D, et al. Normal-tension glaucoma is associated with sleep apnea syndrome. Ophthalmologica. 2002;216(3):180-4.
16. Mojon D, Hedges T, Ehrenberg B, et al. Association between sleep apnea syndrome and nonarteritic anterior ischemic optic neuropathy. Arch Ophthalmol. 2002;120(5):601-605.
17. Papilledema. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed. Bagheri N, ed. Philadelphia: Wolters Kluwer; 2017:259-60.
18. Jennum P, Borgensen SE. Intracranial pressure and obstructive sleep apnea. Chest. 1989;95(2):279-83.
19. Marcus D, Lynn J, Miller J, et al. Sleep disorders: a risk factor for pseudotumor cerebri? J Neuroophthalmol. 2001;21(2):121-3.
20. Idiopathic Intracranial Hypertension/Pseudotumor Cerebri. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed, Bagheri N, ed. Philadelphia: Wolters Kluwer;2017:261-2.
21. Javaheri S, Golnik K. Resolution of papilledema associated with OSA treatment. J Clin Sleep Med. 2011;7(4):399-400.
22. Thurtell MJ, Bruce B, Rye D, et al. The Berlin questionnaire screens for obstructive sleep apnea in idiopathic hypertension. J Neuroophthalmol. 2011;31(4):316-9.
23. Central retinal vein occlusion. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed, Bagheri N, ed. Philadelphia: Wolters Kluwer;2017:289-91.
24. Ceriello A. New insights on oxidative stress and diabetic complications may lead to a "causal" antioxidant therapy. Diabetic Care. 2003;26(5):1589-96.
25. Mohamed Q, Gillies M, Wong T. Management of diabetic retinopathy: A systematic review. JAMA. 2007;298(8):902–16.
26. Schulz R, Hummel C, Heinemann S, et al. Serum levels of vascular endothelial growth factor are elevated in patients with obstructive sleep apnea and severe nighttime hypoxia. Am J Respir Crit Care Med. 2002;165(1):67-70.
27. Diabetic retinopathy. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed, Bagheri N, ed. Philadelphia: Wolters Kluwer;2017:295-300.
28. Central Serous Chorioretinopathy. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 7th ed, Bagheri N, ed. Philadelphia: Wolters Kluwer; 2017:305-6.
29. Mitchell R, Hussey H, Setzen G, et al. Clinical consensus statement: tracheostomy care. Otolarynol Head Neck Surg. 2013;148 (1):6-20.
30. Weaver T, Grunstein R. Adherence to continuous positive airway pressure therapy. Proc Am Thorac Soc. 2008;5(2):173-8.
31. Giles T, Lasserson T, Smith B, et al. Continuous positive airways pressure for obstructive sleep apnoea in adults. Cochrane Database of Sys Rev. 2006;25(1):CD001106.
32. Donovan L, Boeder S, Malhotra A, Patel S. New developments in the use of positive airway pressure for obstructive sleep apnea. J Thorac Dis. 2015;7(8):1323-42.
33. Bucks R, Olaithe M, Eastwood P. Neurocognitive function in obstructive sleep apnoea: A meta-review. Respirology. 2013;18(1):61-70.
34. Olaithe M, Bucks R. Executive dysfunction in OSA before and after treatment. A meta-analysis. Sleep. 2013;36(9):1297-305.
35. Araghi MH et al. Effectiveness of lifestyle interventions on obstructive sleep apnea (OSA): systematic review and meta-analysis. Sleep. 2013;36(10):1553-62.
36. Stauffer J, Fayter N, MacLurg B. Conjunctivitis from nasal CPAP apparatus. Chest. 1984;86(5):802.
37. Harrison W, Pence N, Kovacich S. Anterior segment complications secondary to continuous positive airway pressure machine treatment in patients with obstructive sleep apnea. Optometry. 2007:78(7);352-55.
38. Singh N, Walker R, Cowan F, et al. Retrograde air escape via the nasolacrimal system: a previously unrecognized complication of continuous positive airway pressure in the management of obstructive sleep apnea. Ann Otol Rhinol Laryngol. 2014;123(5):321-4.
39. Cannon P, Madge S, Selva D. Air regurgitation in patients on continuous positive airway pressure (CPAP) therapy following dacrocystorhinostomy with or without Lester-Jones tube insertion. Br J Ophthalmol. 2010;94(7):891-3.
40. Goktas O, Haberman A, Thelen A, Schrom T. The punctum plug as an option for treating retrograde airflow from the lacrimal sac. Laryngorhinootologie. 2007;86(10):732-5.
41. Kiekens S, De Groot V, Coeckelbergh T, et al. Continuous positive airway pressure therapy is associated with an increase in intraocular pressure in obstructive sleep apnea. Invest Ophthalmol Vis Sci. 2008;49(3):934-40.

Top Causes of Double Vision

Getting to the root of the problem is the key to treating and referring properly. Here's a look at the common etiologies of diplopia and how to tell them apart.

By Christopher L. Suhr, OD, MPH, Luanne Chubb, OD, and Lisa Himmelein, OD

A patient presenting with diplopia—whether horizontal, vertical or diagonal—is often a clinical challenge.[1] Constant diplopia with acute onset will have different differentials than intermittent diplopia, for example.[2,3] While the cause can be benign, some cases, such as those accompanied by new headache, ocular pain, unilateral pupil dilation, muscle weakness, ptosis, trauma or papilledema, raise red flags for immediate referral.[4,5] Most etiologies will fall into one of five categories: (1) refractive, (2) binocular vision disorder, (3) orbital disease, (4) neuromuscular junction dysfunction, or (5) injury to the central nervous system/cranial nerves (CNs).[6] A systematic approach to the differentials is key to identifying and treating benign causes—and promptly referring patients when it is vision or life threatening.



CN VI palsy, seen here in the right eye, accounts for 50% of all isolated CN palsies.

Patient History

The first step on the path to proper identification is a thorough patient history. The clinician must determine if the diplopia is monocular or binocular, as binocular diplopia may have a life-threatening cause.[3,4]

Monocular Diplopia

Diplopia that persists when one eye is covered falls into the category of monocular diplopia, or polyopia (greater than two images). Clinicians should have the patient cover each eye separately when testing for monocular diplopia. This finding is rarely due to cortex lesion and is generally attributable to causes within the eye itself. Decreased vision due to uncorrected astigmatism, dry eye and tear film deficiencies, corneal pathology or scarring, iris abnormalities, lenticular changes, vitreal opacities and macular disease are all possible causes of monocular diplopia.[4,7,8] Medications (e.g., antidepressants, antihistamines, diuretics) may contribute to ocular surface dryness and induce a monocular diplopia.[9]

Binocular Diplopia

Unlike monocular diplopia, binocular diplopia, due to ocular misalignment, will disappear when either eye is covered. The type of diplopia the patient complains of—horizontal, vertical or diagonal; worse at distance or near; increased or decreased in a particular gaze position—helps to identify which extraocular muscle

is involved. A thorough systemic health history and step-by-step examination is key to localizing most underlying etiologies.[2,3,10] A systemic health history should include questions regarding trauma, diabetes, hypertension, thyroid disease, cancer, infection and immunosuppression—all of which could cause CN palsies and diplopia through vascular or restrictive mechanisms.[2,6,10]

Although less frequently, certain medications can cause binocular diplopia, such as anticonvulsants, selective serotonin reuptake inhibitor antidepressants, erectile dysfunction medications, migraine therapies and other medications with anticholinergic properties. Many antidepressants may aggravate the symptoms of a convergence insufficiency by affecting accommodation.[6,11]

Ocular motility and alignment testing may include the cover/uncover test, alternate cover test, Maddox rod and corneal light reflex. Ocular misalignment may be caused by a tropia, and an obvious eye turn is noted. A phoria occurs when the misalignment is not obvious, and diplopia occurs only when binocularity is disrupted. A key point in alignment testing is the evaluation for comitancy, in which the size of an ocular deviation remains the same in all directions of gaze. A comitant deviation, such as a decompensating heterophoria, presents with an intermittent or gradual onset, shows full range of ocular movement in all positions of gaze and may have a history of childhood strabismus.[12] In contrast, CN palsies and extraocular muscle restrictions cause non-comitant deviations with the greatest diplopia noted in the direction of action of the weakened muscle.[2,3,9,10] Clinicians must examine each eye separately (ductions) to catch a subtle restriction that could be missed when evaluating both eyes together.[3,5,13] To test versions, the patient fixates on a target that is slowly moving laterally while the clinician checks the medial rectus of the adducting eye and the lateral rectus of the abducting eye. The target is then moved superiorly to evaluate the superior/inferior rectus of the abducted eye and the inferior/superior oblique of the adducting eye. The test is repeated on the opposite side to test contralateral gaze.[3,5,13] Forced duction testing can identify muscle restriction such as in thyroid disease or muscle entrapment by a fracture following trauma.[3,5,13]

Horizontal diplopia, when the images are truly side-by-side, is suggestive of a medial or lateral rectus under action or restriction.[2,3,9,10] Horizontal diplopia present only at near, and especially noted with prolonged near work, is more likely attributable to a convergence insufficiency, which can occur in children and adults idiopathically. Convergence insufficiency can occur after trauma, in neurodegenerative diseases such as Parkinson's disease and with medications that have an anti-cholinergic effect on accommodation.[7] Differential diagnosis for horizontal diplopia at distance includes unilateral or bilateral CN VI palsy, internuclear ophthalmoplegia (INO), age-related decompensating esophoria or muscle restriction, most commonly from thyroid disease, a space occupying lesion or myasthenia gravis (MG).[2,9,10]

Vertical diplopia assessment involves the four remaining muscles: the superior and inferior recti and the superior and inferior oblique. CN III and IV palsies, skew deviations (with or without INO), muscle restrictions and decompensated phorias can all cause vertical diplopia.[9]

Some diseases may cause variable patterns of horizontal, vertical and oblique diplopia throughout the day. Diplopia that varies throughout the day, improves with rest and may have an associated ptosis is highly suggestive of MG as the underlying cause.[7] Other differentials to consider that present with a variable pattern of diplopia include thyroid eye disease, Guillain-Barré syndrome, Parinaud syndrome, Miller-Fisher syndrome, trauma, Parinaud (dorsal midbrain) syndrome and Wernicke's encephalopathy.[2,9,14]

Here some common underlying etiologies of binocular diplopia:

Refractive. Misalignment of the optical centers of prescription glasses or non-prescription reading glasses, poor fitting glasses and the edges of high prescription glasses may all cause diplopia or worsen an existing heterophoria. Aniseikonia from refractive error results in differences in image size and shape in the visual cortex, causing diplopia when wearing glasses. Contact lens use often resolves the image difference in most cases of aniseikonia.[1]

Binocular vision disorder. A patient with a history of childhood strabismus may develop diplopia later in life due to a decompensation of their misalignment.[10] Decompensating phorias and vergence problems are the most common cause of diplopia at near only. Asthenopia occurs with extended near activities, resulting in diplopia and headaches. Convergence insufficiency results in diplopia after prolonged near work and may be associated with uncorrected refractive error, dry eye and Parkinson's disease.[1]

Orbital disease. Thyroid eye disease (TED), idiopathic orbital inflammation and orbital tumors are the most common extraocular muscle and orbital diseases that cause diplopia.[2,3] Orbital inflammation is usually unilateral and may affect the orbital fat, extraocular muscles, lacrimal gland, sclera or optic nerve.

Onset may be sudden and painful, and the eye may appear proptotic. This has been associated with rheumatoid arthritis, sarcoidosis and, less frequently, giant cell arteritis (GCA). Testing includes rheumatoid factor, chest x-ray and ACE level for sarcoidosis and anti-nuclear antibody for systemic lupus erythematosus.

TED predominantly occurs in hyper-thyroid states, although approximately 10% of patients can present with hypo- and euthyroid states, which may not correlate with the thyroid status.[8] Lab testing includes thyroid function and thyroid antibody tests, and risk factors are higher in females, smokers and those with family history of disease.[3] Painless proptosis, muscle restriction, lid retraction and variable lymphocytic inflammatory infiltration are notable findings that occur in approximately 50% of patients with Graves' Disease.[3] The inferior and medial rectus are the most affected, causing a vertical diplopia due to restriction in elevation and an esotropia due to restricted abduction.[2,3,5,8,9] Most patients have a mild form of TED, but 3% to 7% will have vision-threatening concerns from corneal disease or optic nerve compression.[8] A CT scan of the orbit will assess the extraocular muscles and optic nerve and reveal muscle enlargement and risk of optic nerve impingement.[8]

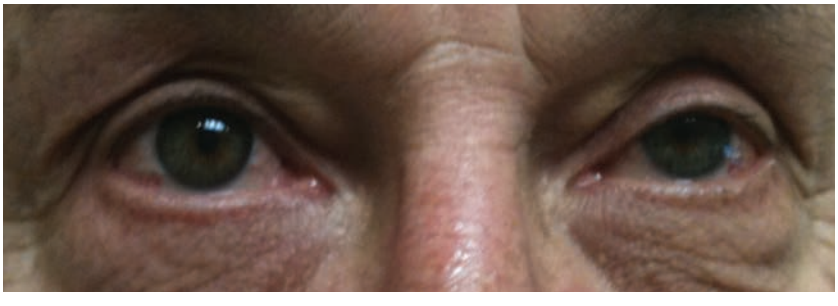
Other, less common, orbital causes of diplopia include trauma and neoplasms. A blow-out fracture is an emergent situation, as the sinus can cause a negative pressure that pulls on the inferior orbital wall, trapping the inferior rectus muscle, resulting in an inability to elevate the affected eye and vertical diplopia.[3,6]

The orbital floor in adolescents is flexible and can quickly open and close, trapping the inferior rectus, and may present with no other obvious signs of trauma. Patients with a history of orbital trauma and a white eye (or lack of subconjunctival hemorrhage) will need emergent imaging to determine if there is entrapment of the inferior rectus, especially in children. In these cases, decompression surgery is urgently needed within 24 to 48 hours to avoid ischemia of the muscle.[15] Conversely, if there is no muscle entrapment, due to orbital floor fracture, surgical intervention may be considered in two weeks.[16]

Neoplasms and sinus-related issues should be considered in the presence of a correlating health history. Secondary orbital tumors, lymphomas and metastatic cancers are the most common orbital neoplasms presenting with unilateral proptosis and resistance to retropulsion.[3] All patients with a new onset of diplopia and a history of cancer should have urgent imaging studies.[17] Rarely, a silent sinus syndrome will cause a downward displacement and enophthalmos of the eye. The obstruction of the ostium of the maxillary causes a negative pressure that pulls downward on the inferior orbital wall, resulting in a vertical diplopia.[3,6,18]

Giant cell arteritis. A patient with GCA can present with any CN palsy.[10] GCA should be ruled out in all patients who present with diplopia, especially those older than age 60. Urgent blood work should include complete blood count, c-reactive protein (CRP) and sedimentation rate (ESR). If the platelet count, CRP and ESR are elevated or GCA is suspected based on accompanying scalp tenderness, headache, fever and generalized malaise, clinicians should refer the patient for urgent treatment.[8,19] Studies indicate that GCA is the underlying cause of diplopia in anywhere from 3% to 15% of presenting cases of diplopia with biopsy proven GCA, but the risk of morbidity and mortality is too high to miss this disease.[8,19]

Neuromuscular junction dysfunction. MG is the classic neuromuscular junction disease that can become life threatening when it affects the muscles of respiration, causing respiratory failure. Approximately 50% to 60% of MG patients present with a ptosis and diplopia, and approximately 20% to 30% have localized ocular involvement.[5,8] The most common age of onset is in the third decade for women and the seventh decade for men.[5] Weakness of the medial rectus is fairly common, but diplopia can vary between horizontal, vertical and oblique. Patients report variable fatigue and ptosis of one or both eyelids that worsens with prolonged activity or toward the end of the day. However, MG can cause a fluctuating diplopia at any time of the day, even on waking.[9] A recent history of weakness and difficulty walking or swallowing are found in generalized MG but absent in the ocular form. As with TED, clinicians should remain suspicious of MG in all cases, as it can mimic CN IV, VI and partial CN III palsies in

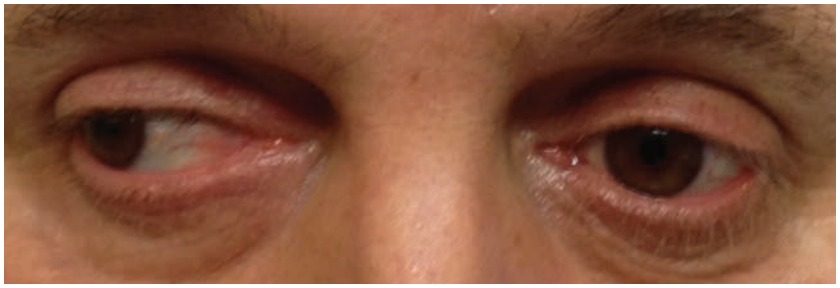


As many as 60% of MG patients, such as this one, present with ptosis and diplopia.

addition to INO, although the pupil is never involved.[2,3]

Several in-office tests are available to help support the diagnosis of MG. During the Cogan lid twitch test, the patient looks down for a few seconds and the clinician then watches the lid reaction when they return to primary gaze. A 1mm to 2mm drop of eyelid elevation immediately after returning to primary gaze is a positive response. Application of ice packs for one to two minutes or a resting state for 10 minutes is an another easy in-office test, and an improvement in the ptotic eyelid is a positive response in suspected cases of MG. Fatigue in prolonged upgaze for at least two minutes with a resulting ptosis, worsened ptosis or inability to maintain upgaze is considered a positive test.[2] Approximately 15% of MG patients will have thyroid changes and co-existing TED, while about 10% will have thymoma present and will be evaluated for surgery.[5]

Internuclear ophthalmoplegia. This is a lesion or injury of the medial longitudinal fasciculus. Clinically, the patient will not be able to adduct the affected eye (or look nasally) and the non-affected eye will show an abducting nystagmus (when looking temporally); convergence, if present, will be spared.[3] An INO may occur unilaterally or bilaterally, and a review of 410 inpatients in 33 years shows the underlying cause can be divided into three major categories: (1) stroke, (2) multiple sclerosis (MS) and (3) other causes such as trauma, injury, infection, tentorial herniation, tumor and GCA.[14] A lesion in the pontine or para-pontine area can cause a gaze palsy opposite the INO, resulting in a "one-and-a-half syndrome." An INO that presents bilaterally results in a large exotropia in both eyes causing a "wall-eyed effect."[9] In patients with MS, an INO is the most common



This patient has longstanding medial rectus palsy secondary to facial trauma.

motility abnormality and is present in as many as 53% at some point in their illness.[8] Patients presenting with an INO should be urgently sent for imaging and bloodwork.

Cranial nerve palsies. CN III, IV and VI palsies share many of the same underlying etiologies such as microvascular CN palsies, intracranial aneuryms and neoplasms.[10,20] Trauma can impair the function of any nerve, but CN IV in particular is more susceptible to trauma. Microvascular disease accounts for many CN palsies in patients older than 50, especially in those with known microvascular disease. Pain and rapidity of onset provide less definitive clues about cause, should a cerebrovascular accident be suspected. Pain can be severe or absent in aneurysmal CN III palsies and ischemic events, though a significant headache and CN III palsy requires careful pupil testing and referral to an emergency room. Research suggests acute onset is associated with ischemic events while slow onset is associated with compressive cases.[20] A CN VI palsy is the most common, followed by CN IV and CN III.[7] In all cases of nerve palsies, evaluation must carefully determine if single or multiple nerves are involved, as imaging is most often warranted, particularly when multiple cranial nerves are involved.[21]

CN VI innervates only the lateral rectus muscle, and paralysis causes an estropia from an abduction deficit. It is the most common isolated ocular motor palsy and accounts for 50% of them.[8] The patient reports horizontal diplopia that is worse at distance and worse when looking in the direction of the affected muscle. Microangiopathic disease causes up to 36% of isolated, acute CN VI palsies in patients older than age 50 with vascular risk, and diplopia spontaneously resolves within two to three months.[2,8] Wernicke's encephalopathy, MS and Duane's retraction syndrome may be mistaken for a CN VI palsy and should be considered in the differentials.[2,5,7] In all other patients with an acute CN VI palsy, assessment for causes such as GCA, tumors, intracranial hemorrhage and trauma warrant referral to the emergency room for evaluation and imaging studies.[8,17] If increased intracranial pressure is the underlying cause of a CN VI palsy, a thorough evaluation should include optic nerve head assessment for the presence of associated papilledema.[8,17]

A CN IV palsy affects the function of the superior oblique muscle, resulting in a vertical oblique diplopia more noticeable in downgaze.[9] Trauma in the most common due to the long course of the nerve around the midbrain.[5] In the absence of trauma, clinicians should test to rule out TED and MG.[7] As with CN VI palsies, microangiopathy is the major cause of a CN IV palsy in patients older than 50.[7,8] The pneumonic GOTS—gaze opposite, tilt same—

indicates there is a greater vertical deviation when the patient looks to the opposite side of the affected muscle or tilts their head to the same side. For example, a right superior oblique impairment will have a right hypertropia: greater diplopia with a right head tilt and when looking to the left. The patient will have a left head tilt to minimize their diplopia.[5,7]

A decompensated congenital CN IV can be distinguished from an acute CN palsy by evaluating vertical fusional amplitudes with prism bars or the amount of ocular rotation between the eyes along with the size of the vertical deviation.[10] The prism bar test is performed by measuring the range of prism that will eliminate the diplopia. Normal vertical fusional amplitudes range from one to four prism diopters, whereas patients with congenital strabismus may demonstrate up to six prism diopters of vertical fusion amplitude.[10,20] A review of old photos may also help to identify those patients with longstanding congenital palsies.

CN III innervates the inferior oblique and the superior, inferior and medial recti muscles. A complete oculomotor palsy results in complete ptosis, a mid-dilated pupil and an eye that appears "down and out." Patients report an oblique diplopia when the eyelid is lifted. Any CN III palsy needs immediate imaging, including CTA or MRA, as compression from an aneurysm of the posterior communicating artery is the most common etiology of a complete palsy with pupil involvement and is life threatening. Ischemic or microvascular causes are more common, and the diplopia often improves during the recovery from the event. The presence of pain may occur in both scenarios but does not help to differentiate between them.

However, most CN III palsies are not complete, and clinicians must use cover testing to catch a subtle signs.

Patients with palsies with ischemic causes are usually older with risk factors such as diabetes, hypertension, hyperlipidemia and tobacco use. Pupil evaluation may help narrow the differential, as the pupillary fibers reside on the dorsomedial aspect of the oculomotor nerve and are affected in 90% of compressive pathologies, causing a fixed, dilated pupil. In contrast, microvascular ischemia causes an infarct in the center of the nerve, which spares the pupil in 70% of ischemic cases. Up to 30% of ischemic palsies will have an anisocoria of 1mm to 2mm. Ischemic palsies usually improve within three months, and never demonstrate aberrant regeneration. The pupil rule cannot be applied to rule out a compression lesion when the palsy in incomplete.[2,5,9,20]

In addition, all patients with a new onset diplopia and a history of cancer require urgent imaging studies to rule out a metastatic lesion.[17]

Researchers have debated the use of imaging in all CN palsies for some time, and most agree those with an acute isolated CN III palsy need urgent imaging to rule out a compressive aneurysm or suspected cavernous sinus thrombosis. Imaging of the brain and orbits is appropriate in suspected retro-bulbar mass, TED or orbital trauma. In patients older than 60, referral for urgent bloodwork is indicated to rule out GCA.[8]

However, a literature review shows that, for CN VI palsies, no definitive answer for imaging exists, as both prospective and retrospective cohorts had valid arguments for their conclusions of imaging all patients with isolated CN VI palsies. Thus, clinicians should always consider imaging CN palsies, especially when presenting with other neurological signs and symptoms.[21]

Diplopia can be a concerning condition for any clinician to address. The key to following the right course of action is determining the underlying etiology. Primary care optometrists often have patients complain of diplopia, and with the right tools and skills, every OD can properly treat, coordinate a proper referral and often reassure the patient with a benign presentation. ■

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1. Mashige KP, Munsamy AJ. Diplopia. South African Family Practice. 2015;58(sup1):S12-17.
2. Rucker JC, Tomsak RL. Binocular diplopia. The Neurologist. 2005;11(2):98-110.
3. Dinkin M. Diagnostic approach to diplopia. Continuum (Minneap Minn). 2014;20:942-65.
4. Low L, Shah W, MacEwen CJ. Double vision. BMJ. 2015;351:h15385.
5. Aminoff MJ, Josephson SA, eds. Neuro-Ophthalmology in Medicine, 5th ed. Philadelphia: Elsevier Science; 2014:487-99.
6. Alves M, Miranda A, Narciso MR, et al. Diplopia: a diagnostic challenge with common and rare etiologies. Am J Case Reports. 2015;16:220-23.
7. Iliescu DA, Timaru CM, Alexe N, et al. Management of diplopia. Romanian J Ophthalmol, 2017; 61(3):166-170.
8. Margolin E, Lam CTY. Approach to a patient with diplopia in the emergency department. J Emerg Med. 218;54(6):799-806.
9. Friedman D. Pearls: diplopia. Sem Neurol, 2010;30(1):54-65.
10. Peragallo J, Newman N. Diplopia—an update. Sem Neurol, 2016;36(4):357-61.
11. Alao A, Lewkowicz C. Seeing double: sertraline and diplopia: a case report. Internat J Psychiatry in Med. 2015;49(1):107-10.
12. O'Colmain U, Gilmour C, MacEwen CJ. Acute-onset diplopia. Acta Ophthalmologica. 2013;92(4):382-86.
13. Pelak VS. Evaluation of diplopia: an anatomic and systematic approach. Hospital Physician. 2004;40(3):16-25.
14. Keane JR. Internuclear ophthalmoplegia. Arch Neurol. 2005;62(5):714.
15. Hammond D, Grew N, Khan Z. The white-eyed blowout fracture in the child: beware of distractions. J Surg Case Rep. 2013;(7):rjt054.
16. Grob S, Yonkers M, Tao J. Orbital fracture repair. Seminars in Plastic Surg. 2017;31(1):31-39.
17. Kirsch CFE, Black K. Diplopia: what to double check in radiographic imaging of double vision. Radiologic Clinics of North America. 2017;55(1):69-81.
18. Saffra N, Rakhamimov A, Saint-Louis LA, Wolintz RJ. Acute diplopia as the presenting sign of silent sinus syndrome. Ophthalmic Plast Reconstr Surg. 2013;29(5):e130-1.
19. Gupta PK, Bhatti MT, Rucker JC. A sweet case of bilateral sixth nerve palsies. Surv Ophthalmol. 2009;54(2):305-10.
20. Cornblath WT. Diplopia due to ocular motor cranial neuropathies. Continuum (Minneap Minn)., 2014;20(4 Neuro):966-80.
21. Murchison AP. Neuroimaging and acute ocular motor mononeuropathies. Arch Ophthalmol. 2011;129(3):301-5.
22. Elder C, Hainline C, Galetta SL, et al. Isolated abducens nerve palsy: update on evaluation and diagnosis. Curr Neurol Neurosci Rep. 2016;16(8):69.

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Perfecting Prism

Don't back down from this life-changing treatment. Here's where to start.

By Erin C. Jenewein, OD

Prism can be a powerful treatment for many conditions and for many patients, specifically for those who suffer from asthenopia or diplopia. Although successfully prescribing it can sometimes seem like a time-consuming and daunting task, the following tips can help you smoothly incorporate the use of prism for binocular vision conditions into your practice for the best outcomes.

Identifying Prism Candidates

One of the most important aspects of successfully prescribing prism is choosing patients who will benefit the most from it. The first hurdle is ensuring your patient is comfortable with wearing spectacles. Although small amounts of vertical prism can be prescribed in contact lenses, patients who need prism usually have to wear it on a spectacle lens. Although this seems like a very basic requirement for prism use, it can present an obstacle if not done appropriately or at all. Patients who wear contact lenses the majority of the time or who have undergone refractive, cataract or strabismus surgery may not be happy with the idea of moving or going back to spectacle wear even if it relieves asthenopia or diplopia. Educating patients on the benefits of prism wear is key in helping get them on board in these cases.

Use Wesson cards to measure fixation disparity and the associated phoria.

Patients with non-strabismic binocular vision disorders—such as divergence insufficiency, basic esophoria and vertical heterophoria—can often benefit from treatment with prism, particularly relieving prism, while others—including those with convergence excess, convergence insufficiency, divergence excess or basic exophoria—are better managed with other treatment modalities, such as vision therapy or lenses.[1] An option for prism patients is prescribing prism to relieve diplopia and asthenopia and concurrently having the patient do vision therapy to attempt to decrease or eliminate the amount of prism needed.

Divergence Insufficiency. Divergence insufficiency patients are often symptomatic for diplopia and asthenopia at distance and may present with a decompensated distance phoria. Divergence insufficiency is associated with systemic and neurological disorders, so we must rule out any underlying conditions that may be causing it. Any patient presenting with divergence insufficiency and neurological symptoms should undergo a full neurological evaluation and imaging.[2]

Vision therapy aims to decrease symptoms of vergence anomalies by increasing the compensating fusional vergence range (base-in for esophoria and base-out for exophoria). The normal amount of fusional divergence ability at distance is lower than that of fusional convergence ability, making it challenging to sufficiently increase ranges to compensate for esophoria or esotropia at distance. This combined with the success of prism treatment in patients with divergence insufficiency makes prescribing prism the ideal initial treatment for this condition. One study successfully treated 87 patients with divergence insufficiency between two and 18 prism diopters (PDs) with prism with none of the patients in the study requiring additional treatment or surgery.[3] Another study found that 100% of patients with divergence insufficiency (30 patients) had success with prism.[4]

Basic Esophoria and Exophoria. Prism can be used alone or in combination with vision therapy to treat basic esophoria and exophoria.

This patient is wearing a Fresnel membrane prism on their glasses.

Vertical Heterophoria. Another condition commonly treated with prism is vertical heterophoria, or vertical strabismus. Vertical deviations often cause patients to experience significant symptoms even when the deviation is small. Our normal supraduction and infraduction abilities are limited, so improving these ranges to compensate for a vertical deviation can be challenging. The difficulty of training vertical vergence ranges along with the success in treating vertical deviations with prism make prism the most appropriate initial management tool in these patients. Retrospective case reviews of patients with an acquired hyper deviation secondary to a superior oblique palsy found that 76% to 92% of participants were successfully treated with prism alone.[5,6]

These patients often present with horizontal and vertical deviations. In many of these cases, the initial management strategy is prescribing vertical prism.[1] Often, if the vertical deviation is decreased or eliminated, the patient may be able to comfortably fuse the horizontal deviation. If the patient is still symptomatic, combining prism and vision therapy may help improve convergence and divergence ranges. When considering whether it is appropriate to prescribe vertical prism for a patient with combined horizontal and vertical deviations, particularly for a patient with intermittent strabismus, first identify whether the vertical deviation is primary or secondary.

A primary vertical deviation is the same in presence and size whether the patient is strabismic or aligned. A secondary vertical deviation, on the other hand, presents when the patient's eyes are strabismic but disappears when the patient aligns their eyes to the ortho position.[7] Secondary vertical deviations are common in strabismic patients, particularly in those with intermittent exotropia. If an intermittent horizontal strabismus patient has a primary vertical deviation, vertical prism may help improve their ability to fuse the horizontal deviation. In patients with a secondary vertical deviation, however, vertical prism is not an appropriate treatment. Rather, treatment for a secondary vertical deviation should aim to improve horizontal sensory and motor fusion through vision therapy so the patient is not in the strabismic position as often, thus eliminating the vertical deviation.

Convergence Insufficiency. Prisms aren't as successful in treating convergence insufficiency and are used less frequently than other treatment modalities. The most effective treatment for this condition is office-based vision therapy, but prism can be considered in patients who are unable to undergo this treatment modality.[1,8] A study did not find a significant difference in the signs or symptoms of children with convergence insufficiency who wore base-in prism compared with children who wore placebo lenses.[9] Although prism has not been shown to be beneficial in children with this condition, adult presbyopic patients with convergence insufficiency may benefit from prism treatment.[10,11]

Strabismus. While patients with strabismus often benefit from prism wear, it is important to first determine whether a strabismic patient is fit for treatment with prism by evaluating their potential for sensory fusion and whether the addition of prism will improve their sensory fusion status. Prism is prescribed to these patients to partially or completely eliminate the motor demand so they are able to fuse. If a patient does not have good potential for sensory fusion, then the assistance that prism gives to the motor system won't allow for normal fusion, and it doesn't make sense to prescribe it.

Sensory Anomalies. Patients with sensory anomalies, such as suppression or anomalous correspondence, cannot be treated with prism until they have been eliminated. Testing for suppression and anomalous correspondence can be easily done with the Worth Dot test. The test can identify and characterize suppression based on the room's illumination and the distance of the test from the patient. If a patient sees four dots in free space or gives a diplopia response but is able to fuse with prism, perform a unilateral cover test. If no movement is seen on the unilateral cover test, the patient has normal correspondence. If the patient gives a fusion response with or without prism but movement on the unilateral cover test is seen, then anomalous correspondence is suspected and relieving or corrective prism is not an appropriate management option. The most successful prism cases are often patients who have intermittent strabismus and good potential for normal fusion or patients with strabismus who have not developed any sensory anomalies. They tend to be older pediatric

Use the Fresnel prism trial set for patients with larger amounts of prism.

or adult patients with new-onset or decompensated strabismus. After these patients are evaluated for any underlying systemic or neurological disease, which must always be ruled out in any case of new-onset strabismus, they are often good candidates for a prism prescription.

Prescribing Prism

After identifying a patient who stands to benefit from a prism prescription, the next step is deciding how much prism to prescribe. There are many different ways to do this, but the best method to use depends on the type of binocular vision disorder for which you are prescribing.

Prescribing relieving prism for horizontal, non-strabismic, binocular vision disorders can be done by calculating Sheard's or Percival's criteria by using clinical data or analyzing fixation disparity and determining the associated phoria.[1] At near, fixation disparity and the associated phoria can be found with a Wesson card. While viewing the card, the patient reports what color line the black arrow is aligned with. The patient is also instructed to keep the words around the lines clear to control accommodation. To determine the associated phoria, add prism in the appropriate direction until the patient reports that the arrow is aligned with the center line. At distance, the American Optical vectographic slide is commonly used to determine the associated phoria. Electronic charts are now routinely used in practice, and many of them have an available distance target for determining fixation disparity and the associated phoria.

The preferred method for prescribing prism for vertical heterophoria is determining the vertical associated phoria.[12] This can be done at near using the Wesson card or at distance.

Case #1

A 25-year-old female presented complaining of double vision and headaches that worsened with prolonged near work. Her medical history was unremarkable, and her ocular history was remarkable only for low myopia, for which she wore glasses.

Upon examination, she had a small exophoria and a 3 PD right hyperphoria at distance and near. Her vertical associated phoria, which I determined using the Wesson card, was 2 PD right hyperphoria. I trialed 2 PDs of base-down prism using a Fresnel prism over the right eye and dispensed at the initial visit. During a follow-up examination three months later, the patient noted increased comfort and resolved diplopia and headaches while wearing the Fresnel prism. A new prescription for prism lenses was dispensed to the patient at the follow-up examination.

Patients with constant strabismus may need corrective prism, or an amount of prism that completely neutralizes their strabismus, in order to obtain good levels of fusion. Relieving prism is often prescribed for patients with intermittent strabismus and sometimes for those with constant strabismus. This decreases the motor fusion demand, allowing the patient to fuse more comfortably. Prescribing for some patients with intermittent strabismus can be done by using Sheard's or Percival's criteria or determining the associated phoria as with heterophoric patients.

Another method used for prescribing for patients with intermittent strabismus, particularly for those who have difficulty with fusion in free space, is Caloroso's Residual Vergence Demand (RVD).[7] RVD criteria look at the direction and size of the deviation and determine how much residual vergence demand the patient should have after prescribing relieving prism. RVD states that esotropic patients of magnitude 6 to 20 PDs should be left with 4 to 6 PDs of residual vergence demand. Patients with 20 to 30 PDs of exotropia should be left with 10 to 15 PDs of residual vergence demand, and patients with a vertical strabismus of 3 to 10 PDs should be left with 2 to 4 PDs of residual vergence demand.[7] RVD is best used in patients who have vergence ranges that have been maximally trained through vision therapy but still need prism to maintain binocular vision in free space.[12]

Determining how much prism is required for improved fusion, or "fusion prism," is another method for prescribing prism for your strabismic patients. Fusion prism is the minimum amount of prism needed to see a change from diplopia or suppression to normal binocular vision.[12] To determine prism using this method, use the Worth Dot test to find a preliminary prism amount. While viewing the Worth Dot test, prism is gradually increased until the patient reports fusion. You can also use Random Dot Stereo (RDS) testing to determine fusion prism. Prism is gradually increased until a patient is able to appreciate the forms on the RDS test. After a preliminary prescription of fusion prism is determined, it is recommended that you trial frame the patient and have them

look around to see if they experience any diplopia when viewing objects in the room. If your patient is still experiencing diplopia, additional prism may be needed to help them achieve fusion.

Case #2

A 5-year-old female initially presented for a strabismus and amblyopia evaluation. She had been previously diagnosed with esotropia and amblyopia but was not currently wearing any correction.

On initial presentation, her best-corrected visual acuities were 20/40 OD and 20/25 OS. Her cover test revealed a 25 PD constant right esotropia with a 2 PD constant right hypotropia. Her cycloplegic retinoscopy was +3.00sph OD and +2.25sph OS. I prescribed glasses (+3.00 sph OD, +2.25 sph OS) for the patient, and she returned for follow-up care, eventually patching and undergoing vision therapy for her amblyopia.

Through her full plus spectacles, she still had a 14 PD constant right esotropia and a 2 PD constant right hypotropia. Beginning treatment, the patient suppressed on Worth Dot testing and had no RDS stereoacuity, even with corrective prism in place. As her vision improved with amblyopia treatment, I continued to monitor her sensory fusion.

At the follow-up examination after nine weeks of patching and vision therapy for amblyopia, her visual acuities were equal in both eyes, and all testing showed normal correspondence. She was able to fuse on the Worth Dot test with 12 PDs base-out and 3 PDs base-up OD. With a trial frame, she was not able to appreciate RDS stereoacuity in-office. I prescribed 12 PDs base-out and 3 PDs vertical prism, split between her eyes.

At the follow-up examination, she reported no diplopia in her glasses, and, eventually, she was able to see 250 seconds of arc RDS stereoacuity. This patient continued with vision therapy to help improve her sensory and motor fusion with the hope of eventually titrating down the amount of prism she wears.

Considering Options

Before settling on a final prism prescription, it is often helpful to trial frame the amount of prism you are about to prescribe to ensure that it will help you achieve your goals. Most trial lens sets come with prism lenses, but for larger angles of strabismus, it may be helpful to use a Fresnel prism trial set. Although younger patients may not be able to give good, subjective feedback, older children and adult patients should wear the trial prism set while engaging in an activity that normally causes them to experience diplopia or asthenopia (reading or distance viewing) to see if it eliminates diplopia and improves comfort.

Fresnel membrane prisms can be very useful for many aspects of prescribing prism. They are low in cost and can be easily applied to a pair of spectacle lenses in-office to try out on a patient for several days or weeks, and refining the prism prescription is inexpensive and simple. Fresnel prism, however, degrades visual acuity and contrast sensitivity, and some patients may not find it cosmetically appealing.[12] A significant decrease in visual acuity through the Fresnel lens can be seen at larger prism powers, particularly at those greater than 12 PDs.[13] When trialing Fresnel prism, it is advisable to only place the Fresnel over one eye so that the patient retains good binocular visual acuity with the prism in place.

Although cosmesis can be an issue with prism glasses, educating your patients on proper frame and lens selection will help them choose a frame and lens combination for the best cosmetic outcome. When prescribing ground-in prism, high index lenses, plastic frames and frames that are smaller in size help improve the weight and cosmesis of the lenses. Antireflective coating is also beneficial for patients wearing prism lenses.

Prism is a powerful tool that can be used to successfully treat a variety of binocular vision conditions. Prism prescriptions often greatly improve the quality of life of your patients by reducing asthenopia and diplopia. Although prescribing it can seem intimidating at first, with practice and a bit of trial and error, you can perfect your ability to prescribe prism for your patients in no time. ■

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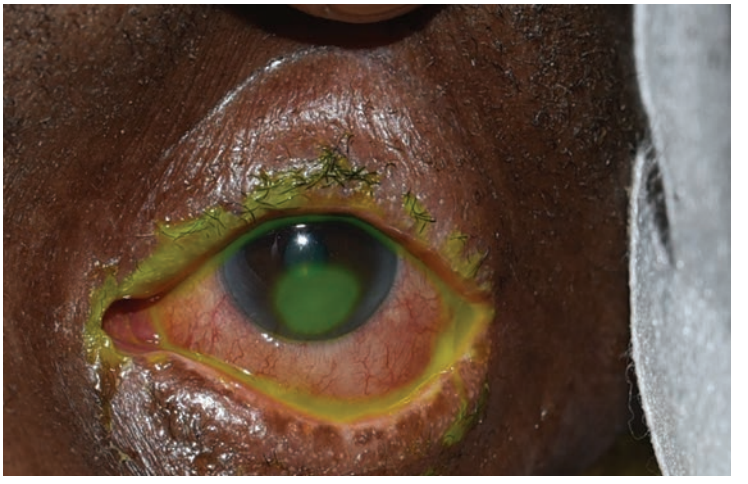
1. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. 1994, Philadelphia, PA: Lippincott Williams & Wilkins. 632.
2. Jacobson DM. Divergence insufficiency revisited: natural history of idiopathic cases and neurologic associations. Arch Ophthalmol. 2000;118(9):1237-41.
3. Godts D, Mathysen DG. Distance esotropia in the elderly. Br J Ophthalmol. 2013;97(11):1415-9.
4. Tamhankar MA, Ying GS, Volpe NJ. Effectiveness of prisms in the management of diplopia in patients due to diverse etiologies. J Pediatr Ophthalmol Strabismus. 2012;49(4):222-8.
5. Tamhankar MA, Ying GS, Volpe NJ. Success of prisms in the management of diplopia due to fourth nerve palsy. J Neuroophthalmol. 2011;31(3):206-9.
6. Neena R, Giridhar A. Effectiveness of prisms in relieving diplopia in superior oblique palsies. Kerala J Ophthalmol. 2016;28(1):38-42.
7. Caloroso E, Rouse MW. Clinical management of strabismus. 1993, Oxford, UK: Butterworth Heinemann Books.
8. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol. 2008;126(10):1336-49.
9. Scheiman M, Cotter S, Rouse M, et al. Randomised clinical trial of the effectiveness of base-in prism reading glasses versus placebo reading glasses for symptomatic convergence insufficiency in children. Br J Ophthalmol. 2005;89(10):1318-23.
10. Teitelbaum B, Pang Y, Krall J. Effectiveness of base in prism for presbyopes with convergence insufficiency. Optom Vis Sci. 2009;86(2):153-6.
11. Pang Y, Teitelbaum B, Krall J. Factors associated with base-in prism treatment outcomes for convergence insufficiency in symptomatic presbyopes. Clin Exp Optom. 2012;95(2):192-7.
12. Cotter SA. Clinical uses of prism: a spectrum of applications. Mosby's Optometric Problem Solving Series, ed. R. London. 1995, St. Louis, Missouri: Mosby.
13. Véronneau-Troutman S. Fresnel prisms and their effects on visual acuity and binocularity. Trans Am Ophthalmol Soc. 1978;76:610-53.

When Corneal Wounds Won't Heal

Timely intervention can keep a bad situation from spiraling out of control.

By Alison Bozung, OD, and Paul Hammond, OD

Corneal epithelial regeneration is constantly taking place. The limbus houses the palisades of Vogt—a specialized niche for maintenance and development of corneal stem cells that differentiate to form the basal cells of the epithelium. From this region, the cells migrate centrally then move anteriorly, transforming into wing cells and then superficial squamous cells. The superficial cells desquamate and are taken away by the tear film.



This patient displays an inferior corneal epithelial defect seen in neurotrophic keratopathy.

Wound Healing Up Close

A corneal insult triggers a cascade of healing mechanisms. Epithelial injury stops basal cell mitosis and causes the release of cytokines and growth factors. Basal cells develop filopodia, which enable them to migrate across the wound. Adhesion molecules expressed by the basal cells allow for adherence to the underlying basement membrane. Once the defect is covered with a single basal cell layer, mitosis resumes and cells proliferate to re-establish regular epithelial stratification. Basal hemidesmosomes slowly re-form to replace the weaker adhesion molecule connections.

In cases of deeper stromal trauma, additional molecules are released, including prostaglandins, platelet-activating factors, cytokines and various growth factors.[1] These substances potentiate transformation of keratocytes into myofibroblasts—contractile cells capable of migrating and filling the wound. The myofibroblasts arrange into a network, secrete extracellular matrix, then apoptose.[2] Unfortunately, residual haze is common as a result of increased collagen fibril diameter and less precise organization.[3]

Impaired Corneal Healing

The procedure described above, while elegant, is fallible. Several conditions can lead to the corneal healing process failing, forming persistent epithelial defects (PED) and possibly underlying ulceration.

Neurotrophic keratitis (NK), for example, compromises corneal healing by reducing nerve function.[4] Diabetes, herpetic keratitis, corneal surgery, topical drug toxicity and trigeminal nerve damage are all among the leading causes of neurotrophic PEDs.[4]

Limbal stem cell deficiency (LSCD) is defined by inhibited proliferation of epithelial basal cells leading to conjunctivalization of the limbus and adjacent cornea. LSCD may result from chemical or thermal burns, topical drug toxicity, a history of ocular surgery, Stevens-Johnson syndrome or ocular cicatricial

pemphigoid. Additionally, severe autoimmune-related dry eye or cicatricial exposure of the ocular surface can result in poor epithelial health.[4]

Clinical Evaluation

A comprehensive approach that includes both history and slit lamp exam is integral to determine a PED's etiology. Accurate history can aid in determining risk factors for poor corneal healing. Conditions such as diabetes, history of ocular or neurologic surgeries, contact lens wear or use of topical ophthalmic medications should all raise red flags.[4]

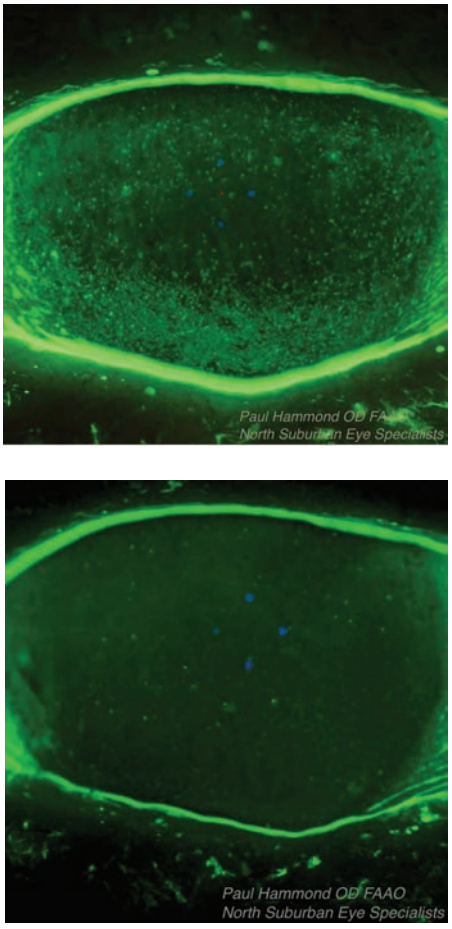
On clinical examination, perform a thorough inspection of the ocular adnexa, tarsal and bulbar conjunctiva, cornea and anterior chamber. In the ocular adnexa, don't overlook eyelid malposition, as this can be a component of poor ocular surface health. Poor lid closure, entropion, ectropion and poor blink through lateral canthal tendon disinsertion may lead to exposure keratopathy. Examination should include the lid margin for signs of keratinization, meibomian gland dysfunction or trichiasis.

Next, turn your attention to the conjunctiva for signs of subepithelial tarsal fibrosis, symblepharon, bulbar conjunctival scarring or forniceal shortening. These may provide insight into a history of prior ocular surface inflammation/infections or prior ocular surgeries. Careful observation at the limbus can provide clues about LSCD, such as pannus or abnormal peripheral corneal epithelium.

On corneal examination, infectious etiologies such as a bacterial corneal ulcer or herpetic keratitis must be ruled out by noting pertinent negatives, which include absence of a stromal infiltrate, anterior chamber reaction or keratic precipitates. Consider the appearance of the corneal epithelial defect; neurotrophic corneal defects typically present as inferior ovoid epithelial defects with rolled edges. Photo documentation is quite helpful in many cases. You should assess decreased corneal sensation before making an NK diagnosis. This can be done quantitatively with Cochet-Bonnet esthesiometry or qualitatively with the "cotton wisp" test.

A Graduated Approach

Corneal healing can often be aided by the placing an oxygen-permeable bandage contact lens with frequent preservative-free artificial tears and prophylactic antibiotic drops. In cases where inflammation is not a major factor, consider early placement of punctal plugs to augment natural tear retention.



This Sjögren's syndrome patient's associated dry eye was treated with Prokera, an overlay that protects the surface and releases nutrients and growth factors while absorbing inflammatory debris as the epithelium grows in underneath it. The above photo shows the patient before Prokera placement, the lower one is after placement.

In the event that an epithelial defect will not heal with basic treatment strategies, more aggressive therapies are indicated. Though scleral lenses may be considered at any stage of impaired ocular surface, severe or recalcitrant PEDs often require their use to protect the ocular surface. Patients with deep-set eyes, small lid apertures or poor dexterity may not be able to successfully use a scleral lens. Amniotic membrane tissue or autologous serum eye drops may be used when more conservative measures fail. In these cases, moving toward early tarsorrhaphy may be the best option.

Custom-fit Sclerals

For patients who are able to tolerate daily contact lens wear, scleral lenses can provide significant benefit in corneal healing.[5] These devices serve as a physical barrier, decrease evaporation from the ocular surface, increase tear-cornea contact and provide a smoother refractive surface. In chronic NK or LSCD, they are a great first-line treatment strategy.

In the event that a cornea or sclera is quite irregular,

consider fitting or referring for a more customizable device. Unlike most other scleral lens devices, the PROSE (Prosthetic Replacement of the Ocular Surface Ecosystem, BostonSight) employs a computer-aided design system to allow for more precise fitting. The EyePrintPro (Eyeprint Prosthetics) uses 3D scanning of a patient's ocular surface impression to create a device that is precisely contoured to the individual eye.

The Serum Solution

ASEDs, also known as autologous serum tears, are becoming increasingly popular amongst eye care professionals. ASEDs contain numerous growth factors, immunoglobulins, fibronectin and vitamin A, which promote cell proliferation and migration.

The first step in producing ASEDs is a blood draw. After blood is harvested from an individual, it is then centrifuged and the serum portion is retained. Depending on the compounding pharmacy's regulations or the doctor's specifications, the percent of autologous serum may vary. Many pharmacies start at 20% serum, but this may be modified based on response to treatment. Traditionally, we have relied on local or hospital compounding pharmacies to produce ASEDs for our patients. More recently, companies such as Vital Tears, which provides a mobile phlebotomy service, allow for a convenient option for both the physician and patient.

Additional autologous hemoderived products include plasma rich in growth factors (PRGF) and platelet-rich plasma (PRP). Both of these products are obtained in a similar fashion, and the difference in production occurs with the speed and duration of centrifugation. PRP and PRGF have proven advantages over ASEDs, due to their increased concentration of growth factors, anti-inflammatory cytokines and other platelet derivatives since they are not lost in the centrifugation process.[4]

Both PRGF and PRP have been successful in treating dry eye disease, post-LASIK ocular surface syndrome, and PRP specifically has also been used for glaucoma-associated ocular surface disease and recurrent corneal erosions.[6-10] Experimental research may support reduced inflammation and likelihood of stromal scarring with PRP vs. ASEDs, though no prospective randomized clinical trials have compared the efficacy of these three hemoderived ophthalmic treatments.[11]

Allogeneic, as opposed to autologous, platelet-derived eye drops are also being evaluated in clinical studies. One such therapy, Elate Ocular (Cambium

Medical) is seeking FDA approval for ocular graft vs. host-disease. FDA-approval could allow insurance coverage for this therapy in the future. These drops would alleviate the need for patient blood draws, as the platelets are sourced from healthy donors.

Typically, autologous eye drops are started four to six times daily in the affected eye, with the percent and dosage titrated upward according to clinical response. Each of the aforementioned autologous drops must be kept in the freezer until ready to be used, then stored in a fridge or on ice if the patient is traveling. This, along with the lack of insurance coverage, must be discussed with the patient prior to starting treatment. Unfortunately, these factors may sometimes be a deterrent for those who need this therapy.

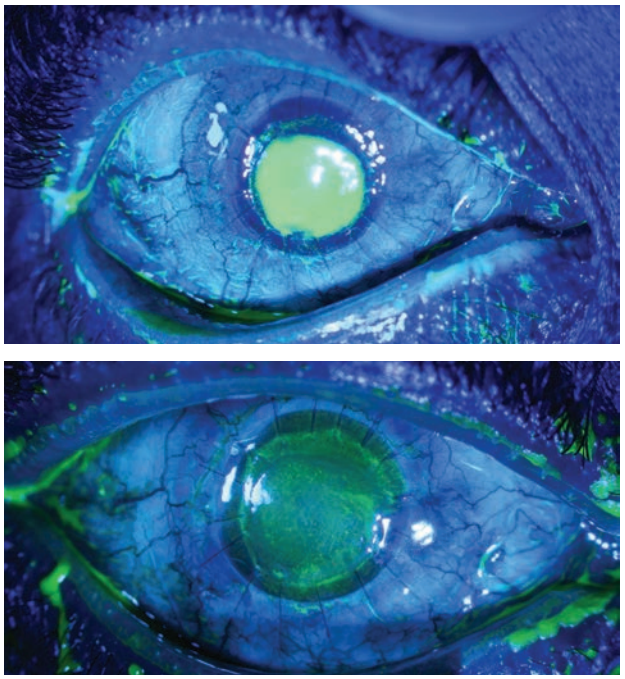
Amniotic Options

Ocular surface grafting via amniotic membrane (AM) application has been reported in ophthalmic literature as far back as 1940, but has gained significant popularity in the last 20 years as cryopreserved and dehydrated products have become widely available for in-office use.[12] The donor graft material is harvested from placental tissue obtained during elective Caesarean sections, screened for transmissible diseases and preserved. Prokera (Biotissue), a cryo-preserved product, is held in position by a polycarbonate ring. Two dehydrated options, AmbioDisk (Katena) and BioDOptix (Integra LifeSciences), are placed under a bandage contact lens.

AM provides many proven beneficial properties to accelerate ocular surface healing beyond acting as a physical barrier and reducing frictional microtrauma from the eyelids. These include anti-inflammatory, anti-scarring, antimicrobial and anti-angiogenic effects, as well as inherent limited immunogenicity.[13] AM use can promote corneal nerve regeneration and increase corneal sensitivity, which supports corneal re-epithelialization.[14,15]

The most common conditions treated with AM include NK, ulcerative keratitis, filamentary keratitis, recurrent corneal erosions, refractory dry eye disease, acute chemical/thermal burns and LSCD.[16] Cases of infectious keratitis have also been treated with AM, but only after appropriate antimicrobial treatment has had time to sufficiently sterilize the ulcer.[17] The purpose of AM in these cases is to decrease inflammation, pain and scarring and promote epithelialization.

Starting to use amniotic membranes may seem daunting, but company representatives will help



This patient with neurotrophic keratitis underwent penetrating keratoplasty. Here, they're seen before (above) and after (below) plasma rich in growth factor eye drop application.

clinicians prepare the materials and instructions for your first patient. Our experiences have left us with two primary strategies for applying AM: as a bandage to protect the ocular surface while it regenerates or as scaffolding for the new growth itself. Prokera works well as an overlay, protecting the surface, releasing nutrients and growth factors, and absorbing inflammatory debris as the epithelium grows in underneath it. Conversely, AmbioDisk adheres directly to the corneal surface and can serve either as an overlay bandage or can be trimmed to inlay healthy basement membrane scaffolding for epithelial cells to adhere to and migrate across.[18] The AM will ultimately integrate into the host tissue when used as an inlay, or slough off as an overlay when epithelial healing is complete.

The primary complication for Prokera is patient discomfort due to the polycarbonate ring, but in our experience, this is less common with the Prokera Slim and improves after the first 24 hours. The dehydrated AM products tend to have issues with adherence and may slip out from under the bandage lens. We've found this issue can be avoided by using a temporary hydrogel adhesive like ReSure Sealant (Ocular Therapeutix) around the periphery of the graft before placing the bandage contact lens.

The recent success of AM in treating the ocular surface has stimulated interest in amniotic-derived eye drops for similar indications, including Regener-Eyes (RNI Solutions) and Genesis (Ocular Science).[19] These drops have many of the same cytokines, growth factors and nutrients as amniotic membrane grafts, and select case reports and retrospective series show they can improve corneal epithelial healing and limbal stem cell proliferation.[20,21] Currently, the lack of standardized clinical trials leaves much to be learned about nuances and efficacy, but amniotic drops hold promise as topical biologic treatments for ocular surface disease while avoiding the blood draw process necessary for ASED/PRP/PRGF.

Oral Medications

Tetracyclines exhibit anti-collagenase activity by reducing levels of matrix metalloproteinases and pro-inflammatory interleukins. For this reason, research shows they are beneficial in epithelial basal cell migration and stromal healing, and are commonly used in cases of stromal ulceration or severe chemical burns. Typically, doxycycline is dosed at 100mg twice daily. Though not a stand-alone therapy, tetracyclines may confer additional benefit in recalcitrant PEDs.

Oral antivirals are also an important consideration when a PED is not improving as expected. Underlying herpetic keratitis is a common culprit in these cases, and starting oral acyclovir or valacyclovir is a safe adjunctive medication to prescribe that may easily resolve the problem.

Lid Closure

One of our mentors once said, "a closed eye is a happy eye." He was referring to how ambient air can desiccate the ocular surface in cases of severe dry eye, but it's a mantra we can apply even with our neurotrophic patients. However, closure of the lids by surgical means can impact an individual's psychosocial health due to altered cosmesis. That being said, it remains a viable option for PEDs that prove recalcitrant to other, more accepted means.

Tarsorrhaphies can be temporary or permanent. A great in-office option is a 'tape tarsorrhaphy'—a very short-term approach that will allow for near total lid closure. This can be done by trimming a 1x1" square of plastic surgical tape, having the patient close their eyes, pulling their brow upwards to smooth any lid creases, then applying the tape with the inferior edge juxtaposing the eyelashes. It will likely need to be reapplied daily by the patient at home, and over the long term may cause a skin reaction underlying the tape.

Chemical tarsorrhaphy, performed by injecting botulinum toxin into the levator muscle, is an alternative that lasts approximately three months. Alternatively, a suture tarsorrhaphy may be done by select placement of sutures and bolsters through the upper and lower eyelids and may quickly be removed following resolution of the corneal defect. Permanent tarsorrhaphies employ excision of the lid margin epithelium prior to suturing, to allow the upper and lower eyelids to "stick" together once they heal, providing a longer term option for those with chronic disease.

Based on the severity of ocular surface disease, a permanent tarsorrhaphy may be performed and is typically closed up to the lateral limbus as a starting point.

New Drugs and Future Directions

It's an exciting time in the eye care field, as new therapies are being developed to help us treat our patients. Oxervate (cenegermin-bkbj, Dompé Pharmaceuticals) is the only FDA-approved topical therapeutic drop indicated for NK. It contains cenergermin-bkbj, a recombinant form of human nerve growth factor that supports differentiation and maintenance of corneal nerves. Nexagon (Eyevance Pharmaceuticals)—the active ingredient is CODA001—is a topical drug that inhibits cell membrane hemichannel formation, decreasing proinflammatory cytokine release and tissue ischemia. Its primary indication is the treatment of PEDs, and clinical trials are underway as of this year.

Surgical treatment for NK has also gained popularity recently through a procedure called corneal neurotization, which involves adjoining a healthy sensory nerve graft from the contralateral supraorbital or supratrochlear nerve, and placing it into the peripheral cornea.[22]

Corneal healing is a complex process, and we need to be ready to step in when the body's own mechanisms aren't adequate. From treatment of simple epithelial abrasions to managing neurotrophic corneal disease, each of us will face cases when our clinical expertise will be called upon. With new advances in both medications and surgical options, we can rise to meet the challenge. ■

Dr. Bozung practices at Bascom Palmer Eye Institute in Miami.

Dr. Hammond is a consultative optometrist at North Suburban Eye Specialists in Minneapolis.

Platelet-rich plasma can help repair recurrent corneal erosions. Here it is after centrifugation (top left), mixing with normal saline (top right) and, finally, bottled.

1. Jester J, Ho-Chang J. Modulation of cultured corneal keratocyte phenotype by growth factors/cytokines control in vitro contractility and extracellular matrix contraction. Exp Eye Res. 2003;77(5):581-92.
2. Myrna KE, Pot SA, Murphy CJ. Meet the corneal myofibroblast: the role of myofibroblast transformation in corneal wound healing and pathology. Vet Ophthalmol. 2009;12 Suppl 1:25-7.
3. Remingto, L. Cornea and Sclera. In:Clinical Anatomy of the Visual System. 3rd ed. St. Louis, Mo: Elsevier, 2012.
4. Alio J, Rodriguez A, Ferreira-oliveira R, et al. Treatment of dry eye disease with autologous platelet-rich plasma: a prospective, interventional, non-randomized study. Ophthalmol Ther. 2017;6(2):285-93.
5. Alio JL, Rodriguez AE, Abdelghany AA, Oliveira RF. Autologous platelet-rich plasma eye drops for the treatment of post-LASIK chronic ocular surface syndrome. J Ophthalmol. 2017;2017:2457620.
6. Anitua E, Muruzabal F, De la fuente M, et al. plasma rich in growth factors for the treatment of ocular surface diseases. Curr Eye Res. 2016;41(7):875-82.
7. Sánchez-avila RM, Merayo-lloves J, Fernández ML, et al. Plasma rich in growth factors eye drops to treat secondary ocular surface disorders in patients with glaucoma. Int Med Case Rep J. 2018;11:97-103.
8. Lee JH, Kim MJ, Ha SW, Kim HK. Autologous platelet-rich plasma eye drops in the treatment of recurrent corneal rrosions. Korean J Ophthalmol. 2016;30(2):101-7.
9. Anitua E, De la fuente M, Muruzabal F, et al. Plasma rich in growth factors (PRGF) eye drops stimulates scarless regeneration compared to autologous serum in the ocular surface stromal fibroblasts. Exp Eye Res. 2015;135:118-26.
10. de Rotth A. Plastic repair of conjunctival defects with fetal membranes. JAMA Ophthalmol. 1990;83(3):522-25.
11. Nakamura T, Yoshitani M, Rigby H, et al. Sterilized, freeze-dried amniotic membrane: a useful substrate for ocular surface reconstruction. Investig Ophthalmol Vis Sci. 2004;45(1):93-9.
12. John T, Tighe S, Sheha H, et al. Corneal nerve regeneration after self-retained cryopreserved amniotic membrane in dry eye disease. J Ophthalmol. 2017;2017:6404918.
13. Morkin MI, Hamrah P. Efficacy of self-retained cryopreserved amniotic membrane for treatment of neuropathic corneal pain. Ocul Surf. 2018;16(1):132-8.
14. Cheng A, Zhao D, Chen R, et al. Accelerated restoration of ocular surface health in dry eye disease by self-retained cryopreserved amniotic membrane. Ocul Surf. 2016;14(1):56-63.
15. Sheha H, Liang L, Li J, Tseng S. Sutureless amniotic membrane transplantation for severe bacterial keratitis. Cornea. 2009; 28(10):1118–23.
16. Malhotra C, Jain AK. Human amniotic membrane transplantation: Different modalities of its use in ophthalmology.World J Transplant. 2014;24;4(2):111-21.
17. Murri M, Moshirfar M, Birdsong O, et al. Amniotic membrane extract and eye drops: A review of literature and clinical application. Clin Ophthalmol. 2018;12:1105-12.
18. Asl NS, Nejat F, Mohammadi P, et al. Amniotic membrane extract eye drop promotes limbal stem cell proliferation and corneal epithelium healing. Cell J. 2019;20(4):459-68.
19. Gupta PK. Clinical outcomes of amniotic cytokine extract in the treatment of dry eye disease. Presented at the 2017 American Academy of Ophthalmology Annual Meeting.
20. Terzis JK, Dryer MM, Bodner BI. Corneal neurotization: a novel solution to neurotrophic keratopathy. Plast Reconstr Surg. 2009;123(1):112-20.

PEER • REVIEWED • PRA • ARTICLE

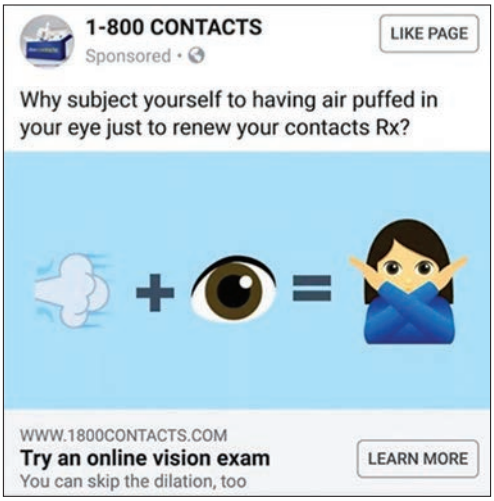
Optometry in the Age of Disruption: Doctors vs. Online Vendors

These companies prioritize cost and convenience over quality eye care and the doctor-patient relationship. Here's how to defuse the threat they pose. By Jeffrey Sonsino, OD

There has never been a better time in America for entrepreneurs to create start up companies. Online platforms, such as Shopify, Square and Amazon Web Services, make it easy to interact with the companies they are associated with, and funding mechanisms, including Kickstarter and IndeGoGo, make it easy to raise money to fund business endeavors.[1] Start ups are everywhere you look. Consequently, our culture has evolved to prioritize convenience over value.

A Threat to Patients

Overnight, three scooter companies recently dumped thousands of scooters onto the streets of my hometown, Nashville. Initially, people loved the convenience, low cost and accessibility of this mode of transportation. Over time, however, it became clear that the scooter companies were not working with local leadership to enforce the safety of scooter riders. Their half-hearted attempts to ensure riders used helmets, drove safely and followed the rules of the road fell on deaf ears. Every night on the news, I watched videos of drunk scooter riders flying over car hoods, hitting pedestrians on sidewalks and blowing through red lights into cross traffic. The local academic medical center started reporting astronomical increases in rates of head injuries due to scooter accidents. Only after a well-publicized death from a scooter accident did the city start to consider banning scooters. This problem is characteristic of similar concerns occurring in health care, specifically in optometry, that are leaving patients equally vulnerable to safety issues.



This is an example of a Facebook advertisement used by 1-800 Contacts to promote the company's online contact lens prescription renewal platform.

In the health care sector, a growing tide of websites and apps allows the public to diagnose and treat themselves without doctor oversight. Some areas of medicine are pushing back, citing the dangers of unmonitored self care as reasons for their resistance. The FDA is telling the public not to use untested and unapproved apps to diagnose concussions without assistance from a doctor.[2] Midwives are cautioning expectant mothers to stay away from baby heart monitor apps.[3] Physicians are warning patients to steer clear of direct-to-consumer websites supplying drugs without requiring a prescription.[4]

In the contact lens field specifically, there is no shortage of companies not abiding by or adhering to the law. For years, optometrists have complained that 1-800 Contacts uses loopholes in passive verification methods to accommodate expired prescriptions and provide lens quantities in excess of prescription durations. With more severe consequences than ever before, the online vendor is now actively marketing the

ability to skip in-office eye assessments by encouraging consumers to renew their contact lens prescriptions online.[5]

Making it possible to renew contact lens prescriptions online, 1-800 Contacts partnered with Visibly.[5,6] Visibly operates by obtaining a patient's prescription, giving them a do-it-yourself sight test and renewing their prescription. Ophthalmologists work with the company to issue these renewals. Most ophthalmologists and consultants who understand why contact lenses are class II and III medical devices would agree that this is not a good idea. But there are those who tend to value convenience and cost over annual exams and eye care.[7]

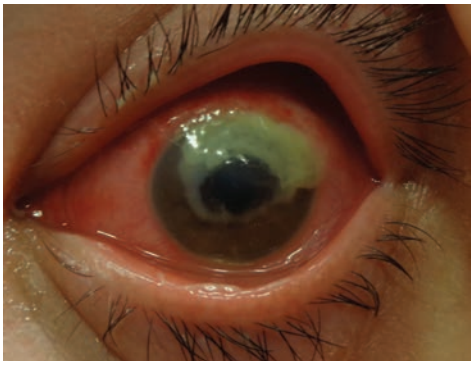
These direct-to-consumer contact lens companies exploit loopholes in the passive verification process as part of their business strategy, essentially working around lens prescriptions altogether. Many optometrists criticize Hubble as the main culprit and are unwilling to prescribe the company's lenses due to the poor quality of their material, methafilcon A.[8,9] Third-party Vision Path "verifies" Hubble's prescriptions but prefers leaving one-way voicemails and not providing prescription verification information directly to office staff, likely because the company knows its requests will be denied.

Advocacy Efforts

Many organizations are fighting to counter the backwards narrative online contact lens sellers are feeding to consumers that convenience and cost should supersede eye care and the doctor-patient relationship.

The American Optometric Association (AOA) is leading the fight on behalf of optometrists and our patients. Joining the AOA's unparalleled advocacy efforts helps ODs protect patients from insurance companies, corporations and even themselves. The AOA encourages those with questions about contact lens violations to submit them to stopillegalcls@aoa.org and those with complaints to report them to the FTC or FTA at www.accessdata.fda.gov/scripts/medwatch or www.accessdata.fda.gov/scripts/email/oc/buyonline/english.cfm.[10]

The Health Care Alliance for Patient Safety (APS) is a newer advocacy organization that promotes the doctor-patient relationship and consists of AOA members specifically and the contact lens manufacturing industry in general as well as a vision insurance plan and a technology start up. The organization is supporting two bills in 2019 designed to shift the focus of contact lens sales back to being patient-centric. The APS is also working to educate lawmakers on the hazards of lax oversight of contact lenses.



This patient suffered a corneal perforation due to a *Pseudomonas* ulcer from ordering contact lenses online without doctor oversight.

Fighting Back

Every optometrist can make several changes in their practice to ensure they are providing the best care to their patients: offer convenience, provide patient education and differentiate their services and products.

Online sellers may have an edge when it comes to convenience, but it is time to close the gap. We can no longer continue to do business the same way we have done it for the past 20 years. If your staff is communicating with patients by calling them, then it may be time to modernize your practice. Text messages outrank phone calls as the dominant form of communication with millennials.[11] If patients are unable to order contact lenses through your website, it may be time to look into adding this service.

These additions, plus allowing patients to conveniently schedule their appointments online, making

ODs Take Charge

In an effort to equip optometrists with tools to compete from a business perspective, I teamed up with a business strategist/entrepreneur to launch Eyeris. The company's aim is to help optometrists go head-to-head against disruptors by offering services that seek to neutralize their advantages. Patients want quick access to eye care, so we created software that matches people seeking same- or next-day appointments with practitioners who have openings due to cancellations or no-shows. This safer alternative to online vision tests provides patients with quality care, but on their schedules. Patients also want affordable lenses they can order online, so, later this year, Eyeris will debut a daily disposable that will only be available with doctor oversight and cannot be substituted for another lens. Patients can order it online from Eyeris, but the doctor maintains control over the process—and the margins, just as if they sold it in their office. We also believe our pricing advantage will keep patients from looking to other online outlets. Only time will tell, but we feel Eyeris can shift the way optometrists do business and help them compete with the tide of disruption.

Aveo Chat Transcript with Andrew [redacted]

Chat started on 05 Oct 2018, 07:37 PM (GMT+0)

(07:37:43) *** Andrew [redacted] joined the chat ***
(07:37:43) **Andrew** [redacted]: Can my Optometrist fit me for these lenses? I'm in Nashville, TN.
(07:37:46) *** Kimberly joined the chat ***
(07:38:29) **Kimberly:** Thank you for reaching out to Aveo, my name is Kimberly.
(07:40:47) **Kimberly:** Hi Andrew! Your doctor would need to request a fitting kit from us before they would be able to fit you for our lenses. We do provide an online vision test that is often cheaper than having your doctor fit you for contacts and you will see that option when placing your order. You can also just submit your current contact rx and we will attempt verification using that!
(07:42:53) **Andrew** [redacted]: So you you would be able to substitute my current prescription? I have Accuvue 1-Day Moist
(07:43:36) **Kimberly:** Some doctors don't mind allowing substitutions so we always like to check first before having you take an unnecessary step like an additional vision test or fitting!
(07:43:59) **Kimberly:** Just make sure to include correct contact information for your doctor when you place your order and we'll take care of the rest!
(07:45:10) **Kimberly:** If your doctor denies our request to verify your prescription or there is any other issues we would reach out to you by email.
(07:45:34) **Andrew** [redacted]: Should I ask my Doctor if they are ok with the substitution first?
(07:45:48) **Kimberly:** That's really not necessary!
(07:47:04) **Kimberly:** You can place your order and we will use the information you provide to contact your doctor. If there is any issues we will place the order on hold while we let you know what additional steps are needed, such as a new fitting or the option to take our vision test online.
(07:50:16) **Andrew** [redacted]: I'd rather you reached out first if that's ok. I've run into issues like this before. His email address is optique@optiquenashville.com
(07:51:00) **Kimberly:** We can't reach out to your doctor without an order being placed but if you would like to you can reach out to him.

Read this transcript of an interaction between a patient and direct-to-consumer contact lens company Aveo.

sure you are not running behind and maximizing chair time, will hopefully sway patients away from taking the online vendor route. Implementing these strategies into your practice is easy with optometry-friendly companies that can help you advertise these features on your website and in your practice.

Optometrists used to perform a battery of tests without explaining what they were or why they were doing them. Those days are long gone. Now, patients have options, and if they are not getting what they need or want from you, they will go elsewhere to find it. Patients must be armed with extensive, comprehensive eye care knowledge so that when 1-800 Contacts calls for them to "skip the air puff test," they know what the test is and why it is important and can make an educated decision for themselves. It may seem like some patients do not care about or understand the information we have to offer.

However, how you communicate with them makes a huge difference—if it is not effective or clear, you are right back to square one. Sometimes patients need to hear the consequences of skipping a test or exam, turning down treatment or misusing a service or product for the importance of annual eye care to truly sink in. Make every visit meaningful for patients so that they have something to take away and are that much more likely to return next time.

Not every patient is an optimal candidate for off-the-shelf contact lenses. Patients with high ametropia or high astigmatism may be better suited for hybrid lens wear, for example, due to their higher Dk, customizable base curve, centration abilities and lack of a need for rotational stability. Patients with specific ocular problems require specific solutions that online vendors cannot fulfill. If you offer a service or product that can not be or is not easily replaced or replicated, patients will seek you out when they are in need. Set yourself apart from others and give patients a reason to go to you first, every time.

Many companies have priorities that compromise consumer safety, as was the case with the scooters in Nashville. Many organizations do not understand or respect quality eye care; but, the good news is that many organizations also advocate for the doctor-patient relationship and the highest standard of clinical eye care. The OD is at a turning point in the evolution of eye care. Instead of succumbing to a future narrative dictated by companies that do not have patients' best interests in mind, we should adapt the way we practice to stay ahead. As long as what we have to offer is valuable and unique to patients, we will remain a central part of the equation. ■

Dr. Sonsino is a partner in a specialty contact lens and anterior segment practice in Nashville, Tenn. He is a diplomat of the Cornea and Contact Lens section of the American Academy of Optometry and the past chairman of the Cornea and Contact Lens section of the American Optometric Association. He co-founded Eyeris, which launched at the 2019 AOA conference.

1. Reeves J. Why it's easier than ever to be an entrepreneur. Business Insider. www.businessinsider.com/why-now-is-the-best-time-to-start-a-company-2014-10. October 22, 2014. Accessed June 25, 2019.
2. Wicklund E. FDA targets DIY mHealth devices aimed at concussions, head injuries. mHealth Intelligence. mhealthintelligence.com/news/amp/fda-targets-diy-mhealth-devices-aimed-at-concussions-head-injuries. April 11, 2019. Accessed June 25, 2019.
3. Coates L. Senior midwife begs pregnant women not to use 'extremely dangerous' baby heart monitor apps. Eastern Daily Press. www.edp24.co.uk/news/health/jpuh-midwife-warning-over-extremely-dangerous-app-1-5962093. March 27, 2019. Accessed June 25, 2019.
4. Yarbrough JL. The dangerous side effect of digital wellness. Fashionista. fashionista.com/2019/03/digital-health-wellness-companies-government-regulations. March 20, 2019. Accessed June 25, 2019.
5. 1-800 contacts launches online eye exam powered by Opternative. Vision Monday. www.visionmonday.com/latest-news/article/1800-contact-launches-online-eye-exam-powered-by-opternative-1/. August 1, 2016. Accessed June 25, 2019.
6. Opternative changes name to 'Visibly' to better reflect a move toward partnerships. Vision Monday. www.visionmonday.com/latest-news/article/opternative-changes-name-to-visibly-to-better-reflect-a-move-toward-partnerships. December 11, 2018. Accessed June 25, 2019.
7. Federal Trade Commission. The contact lens rule and the evolving contact lens marketplace. www.ftc.gov/system/files/documents/public_events/1285493/panel_ii_contact_lens_health_and_safety_issues.pdf. Accessed June 25, 2019.
8. American Optometric Association. Concerns validated, AOA stands ground amid Hubble row. www.aoa.org/news/advocacy/hubble-update. Accessed June 25, 2019.
9. American Optometric Association. A Letter to the Honorable Chairman Joseph Simons. www.aoa.org/Documents/CLCS/Hubble-April-2019(0).pdf. April 23, 2019. Accessed June 25, 2019.
10. American Optometric Association. Reporting contact lens violations to the FDA and FTC. www.aoa.org/advocacy/federal-advocacy/regulatory-issues/fclca/report-cls-violation/report-to-fda-and-ftc. Accessed June 25, 2019.
11. Howe N. Why millennials are texting more and talking less. Forbes. www.forbes.com/sites/neilhowe/2015/07/15/why-millennials-are-texting-more-and-talking-less/#2c2b300d5975. July 15, 2015. Accessed June 25, 2019.

To listen to a call between a Hubble employee and a member of my staff, read this article online at www.reviewofoptometry.com or scan the QR code.

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Provide Specialty Contact Lenses and Thrive

You can build doctor-patient loyalty and keep patients from shopping around by offering something your competitors don't have. **By Jane Cole, Contributing Editor**

Today, patients have an ever-increasing list of inexpensive options to choose from when buying their contact lenses—from online behemoth 1-800 Contacts to the recently launched Hubble that promises the first box of daily disposable lenses will be free. And most optometrists agree that trying to compete with these bargain venues on price alone is a losing battle.

Instead, experts say you should focus on what these retailers can't provide: quality care. For contact lens fitters, this includes providing specialty contact lenses that require more time and attention to fit, such as torics, multifocals, toric multifocals, sclerals, gas permeables (GPs) and hybrids. If you build a thriving practice of patients wearing these lens types, those patients will feel more loyal to you and will be less inclined to shop around for the best deal.

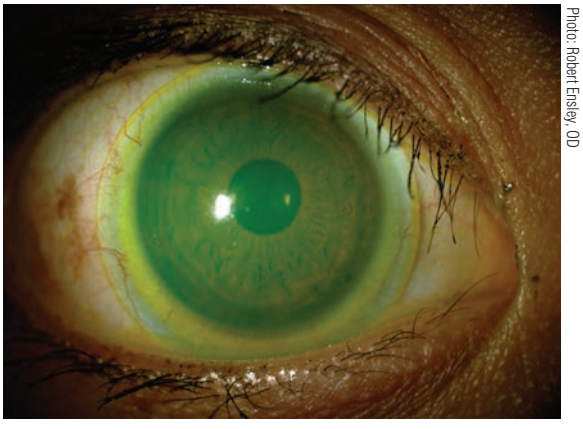


Photo: Robert Ensley, OD

A specialty lens, such as this mini-scleral, may be a good option for your patient with high corneal astigmatism—and it's one only you can prescribe.

"In the last decade, there has been an incredible increase in lens parameters that are available for our patients, but I often hear optometrists mention they are hesitant to start the fitting process with these lens types because they are viewed as more difficult or more time consuming," says Andrew Fischer, OD, of Specialty Eyecare Group in Kirkland, WA. "This may be true, but investing the time to achieve a successful fit with a multifocal or toric lens can pay off many times over."

Specialty lens wearers, especially multifocal-wearing presbyopes, are often long-term patients, and they are a "fantastic referral source" as well, Dr. Fischer explains.

Still, specialty lenses remain an untapped opportunity for many. A 2015 Gallup study of the US multifocal contact lens market found 42% of patients between the ages of 40 and 54—and 38% of those aged 55 to 64—have expressed some interest in multifocal contact lenses.[1] Despite this growing need, just slightly more than half of eye care professionals start a conversation about innovative contact lenses, and 91% of practitioners fit less than 20% of soft multifocal lenses.[1,2] Even during a multifocal lens conversation, only 15% of clinicians reported they present the lenses

enthusiastically to their patients, while 48% said they offer warnings about the lenses' downsides before the patients have even tried them.[2,3]

Here, your colleagues offer insight on how specialty lenses can help your practice gain a leg up on the competition, in addition to some "do's and don'ts" on how to best get your patients into a specialty lens.

Play the Loyalty Card

Patients in specialty lenses are more apt to purchase lenses from their doctor, says Jason Miller, OD, of EyeCare Professionals of Powell, Ohio. The fact that the doctor has taken more time to find the best fitting lens or the right combination likely weighs into a patient's decision on whether or not to search for a better price, he adds.

"I don't really like to consider I'm competing with companies like 1-800 just in price, because I can beat them with in-person service," he explains. "By offering great service and advanced technologies like specialty lenses at a good price, it certainly helps swing that competitive edge toward the practice."

Specialty lenses—such as sclerals, GPs, custom torics and hybrids—require a greater emphasis on the relationship between the patient and the doctor, not the product, says Brian Chou, OD, owner of a specialty contact lens practice, ReVision Optometry, in San Diego.

"The commodity world of disposable contacts is inhabited by a very strong brand-centric emphasis on the product, where the patients develop a loyalty to the brand and the product, not the services or the doctor," Dr. Chou says. If patients see their contact lenses as a commodity, online marketers are likely to amplify the perception that the doctor is not a necessary part of the puzzle, he adds.

Stand Out With a Specialty Lens Practice

After selling his practice to a private equity affiliate more than two years ago and buying an existing practice in central San Diego, Dr. Chou decided to shift his focus to specialty contact lens prescribing. Since then, he's grown to love Walmart, Costco, LensCrafters and other retail competitors he previously faced at his former group optometric practice.

Because Dr. Chou's new office caters to specialty lens patients, with an emphasis on scleral lens prescribing and keratoconus and irregular cornea patients, the big box stores and retail chains refer him their hard-to-manage contact lens patients, and the relationship is paying off in dividends.

He still does general eye exams, but most of his patients are wearing specialty lenses. Dr. Chou treats any and all corneal complications, including keratoconus and corneal transplant patients, and he fits scleral and prosthetic contact lenses in addition to impression scleral cover shells with EyePrint Pro (EyePrint Prosthetics) and corneal refractive therapy.

Although he doesn't think there are enough patients with irregular corneas to support a thriving specialty contact lens practice for every OD, Dr. Chou still encourages others to delve into a specialty, such as myopia control or dry eye, to diversify their practice from mercantile optometry

"I am pleased with the outcome. Most optometrists will admit there are a lot of competitive threats ranging from online refraction to Hubble. Now my practice coexists in this ecosystem where all these other entities are too," Dr. Chou says.

A Caveat

Specialty fits may help differentiate your practice, but this scenario may not always equate to a large volume of patients, some experts warn.

"While a specialty practice is ideal, it is impossible for the average OD to have one," says Justin Bazan, OD, of Park Slope Eye in Brooklyn, NY. "You add specialty fits to your existing contact lens services, but there simply aren't enough specialty patients to sustain a practice that only does specialty fits. You will have to compete with online retailers, not necessarily on price, but you will have to compete for the sale of soft contact lenses."

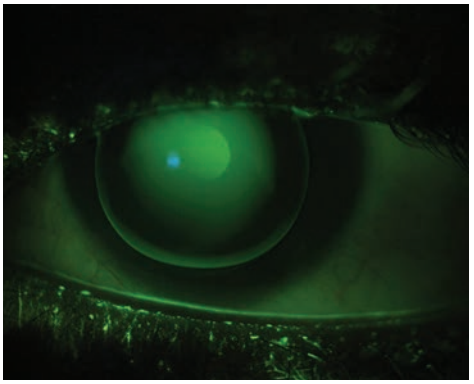
Still, expertise in this area can give you a competitive edge over other practices, as many patients, even those with general needs, may feel like they are in better hands when seeing a "specialist."

Photos: Brian Chou, OD

Providing custom medical contact lenses, such as EyePrint Pro, has led to OD and MD referrals, including from big box and retail stores, Dr. Chou says.

Dr. Chou gives the example of a patient who wears a popular single-vision daily disposable. "If you ask the patient the reason behind their wearing success in many of these disposable lenses, the patient will often say it's the brand, and in this case, it doesn't matter who prescribes it. The patient will just go to the lowest cost provider."

Photo: Irene Frantzis, OD



Patients with a high amount of astigmatism may not know they can achieve better vision with a well-fitting GP until you educate them.

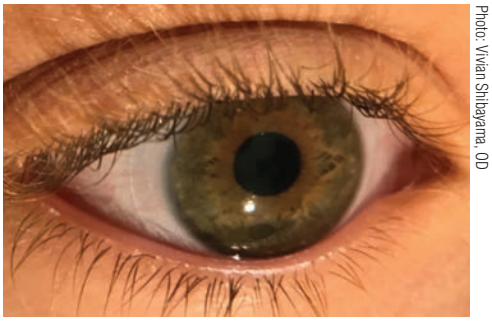


Photo: Vivian Shibayama, OD

This patient's larger-than-average horizontal visible iris diameter led to contact lens intolerance with traditional soft lenses. Her OD took the time to design a toric lens with an increased diameter, which did the trick.

In this scenario, patients see the doctor as a third wheel interfering with their access, because the focus is on cost and convenience, not the care, Dr. Chou says.

Specialty contact lenses, however, often require tremendous expertise, beginning with the identification of good candidates.

"Patients won't come into the office asking for a front-surface toric prism ballasted scleral contact lens," Dr. Chou explains.

Instead, they usually say they want a certain brand of soft lenses. It's up to you to notice they might do well in another, more specialized, lens and offer that option.

Dr. Fischer finds patients will acknowledge and appreciate the extra time it takes to achieve a perfect fit in more complicated lenses, which often helps with loyalty. Custom soft lenses, sclerals, corneal GP lenses and orthokeratology designs often need two or more lens orders and evaluations on the eye before finalizing a lens prescription, he explains.

The Price Isn't Always Right

If practitioners are tempted to compete on price with online entities and big box stores, they should know it may be an unwinnable fight, the experts warn.

Optometrists can address three key features when structuring their contact lens practice, according to Dr. Fischer: fast, cheap and quality. "The catch is, we can only choose two. If we choose fast and cheap, we sacrifice quality," he says. "As a business model, fast and cheap can be successful; however, as a medical provider dealing with something as vital as my patients' eyes and vision, quality eye care is not something I am willing to sacrifice."

Remember that someone else will always be faster and cheaper than you, says Stephanie Woo, OD, of Havasu Eye Center in Arizona. Additionally, you will lose if you're trying to compete for patients who are looking to get the cheapest price. Instead, be known for your high level of customer service and instrumentation, and patients will find great value in that, she says.

Dr. Miller agrees that it's a mistake to chase online outlets. "That's a bad race to the bottom, and patient service has to be sacrificed in order to chase that lower price."

When price really is an issue, patients are more inclined to try specialty lenses if they can get help from their insurance, so Dr. Fischer suggests becoming familiar with the rules of individual insurance plans.

Find Your Followers

One of the simplest ways to open your patients' eyes to specialty lenses is just by mentioning the option when they are in the chair. Once you have their interest, you have to demonstrate that you are well versed on the latest lens technologies.

"Talk about the lenses and always do what's best for the patient, regardless of your perception of the patient's ability or willingness to pay for them, which is often a barrier to doctors having the discussion in the first place," says practice management expert Gary Gerber, OD, of

Think Like Amazon

Savvy doctors should expand their thinking on ways to compete with 1-800 Contacts other than price, Dr. Gerber says.

Instead of focusing on the lenses, the practice management expert suggests paying attention to the event, experience and everything else that surrounds the delivery of the contact lenses.

"Think about how online retailers compete with each other. Amazon isn't always the lowest price provider, but they make it so easy to order and reorder and return things, that it's tempting to not even bother shopping for a better price," Dr. Gerber says. Doctors should make their patients' contact lens experience—both online and in the office— "Amazonian" to mitigate price pressure from competitors.

"This goes for everything in their offices, not just specialty lenses," he says.

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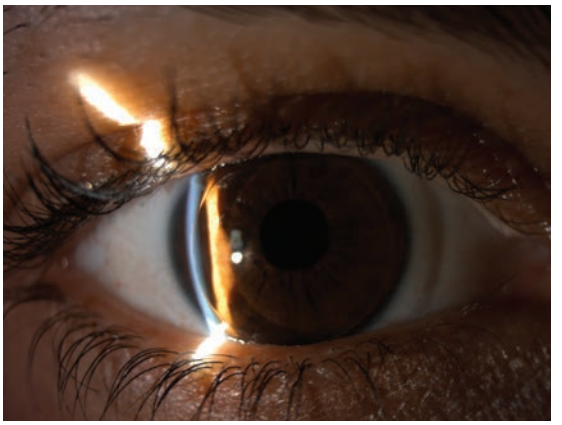

Photo: Suzanne Sherman, OD

A smart OD resolved this patient's daily rigid GP lens awareness by piggy-backing a GP lens on top of a silicone hydrogel lens.

The Power Practice.

When fitting multifocals, for example, Dr. Woo will ask patients if they would be willing to try a lens that allows them to see distance and near without reading glasses, and she finds most of the time, patients are excited to try them.

Mentioning specialty lens options in the exam room is part of the routine for Dr. Fischer. "Specialty lenses do not require 'special' eyes. Specialty lenses are great for normal corneas and those wanting higher quality vision. Patients with moderate to high astigmatism and higher prescriptions make great specialty lens candidates."

He also encourages optometrists to get outside of their comfort zone and take the plunge into specialty lenses. "Being able to fit contact lenses well comes with experience, and the more specialty lenses they fit, the more comfortable they become. There are many tools, webinars, resources and conferences that provide fantastic guidance on how to succeed with specialty lenses.

Troubleshooting

The number one fitting mistake is not following the proper fitting guides, according to Dr. Miller. Many doctors try to use their experience and make adjustments that could lead them in the wrong direction, he says. Easy steps doctors can take to better ensure their success with specialty lenses is being up-to-date with the newest technologies, not masking astigmatism and being a strong advocate of the newest lens options, he says. "Additionally, many doctors remember that one patient who was a challenging multifocal fit instead of remembering the many other patients who loved the technology."

Also, be crystal clear on who would be a good candidate. Poor candidates, Dr. Woo says, include patients who want pristine vision at all distances; have unrealistic expectations such as demanding 20/15 vision at far and near but never want to wear glasses again; have failed in soft contacts due to dryness; or have ocular surface disease or other ocular pathologies.

Jump into Multifocals

For early presbyopes who haven't worn contacts, optometrists may be more inclined to first introduce them to a single vision lens before graduating them into a multifocal. But experts say this may be a misguided approach that could hamper contact lens success.

"I jump right into multifocal lenses," Dr. Miller says. "It is easier to introduce multifocal contact lenses when patients first start noticing symptoms."

Dr. Fischer follows the same approach. "These patients are in our chair with near complaints, and fitting them into distance contacts does not solve that problem. Multifocals provide an option to correct for both distance and near. Additionally, I find transitioning a patient into multifocals as an emerging presbyope is much easier than waiting until they have no accommodation left. For me, the earlier, the better."

Early presbyopes are some of the easiest patients to fit with multifocals, with the exception of those patients who are in "presbyopic denial," Dr. Gerber adds.

As 1-800 Contacts and other discount lens entities continue to infiltrate the marketplace, ODs have an opportunity to differentiate themselves by focusing on what they do best: providing quality, specialized eye care. Given today's contact lens advancements, that includes offering access to a variety of specialty lens modalities to meet the visual needs of each and every patient. ■

1. Multi-sponsor Surveys, Inc. 2015 Gallup Study of the U.S. Multi-Focal Contact Lens Market. October 2015; Princeton, NJ.
2. Bausch & Lomb's Innovation Index, 2015.
3. Jobson Optical Research 2015.

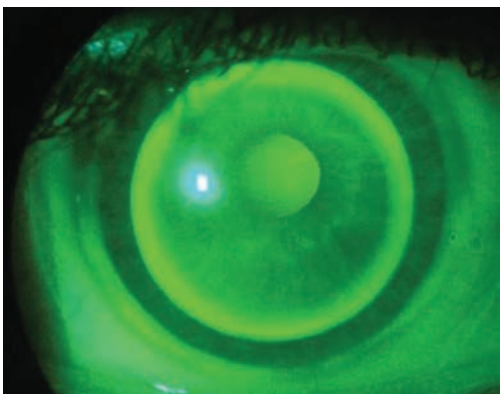

Photo: Robert Ensley, OD

This competitive athlete was not happy with his fluctuating vision in standard soft toric lenses. A hybrid lens that provided GP optics without lens rotation or dislodgement gave him comfortable 20/15 vision in each eye.

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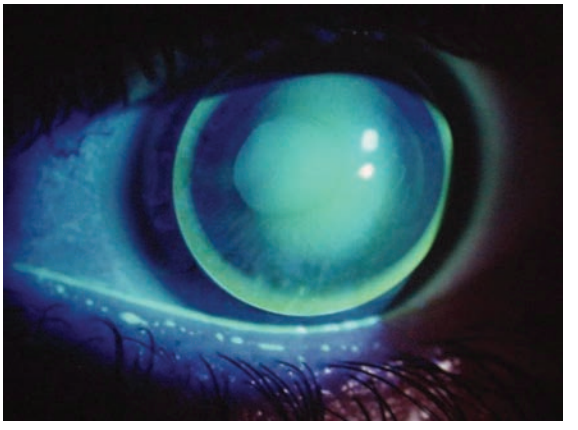
PEER • REVIEWED PRA ARTICLE

Don't Miss Out On Multifocals

These devices can be a practice builder, not a spirit breaker. Here's how.

By Mark De Leon, Associate Editor

A recent study estimates 1.8 billion people globally have presbyopia, 826 million of whom have near vision impairment because they have inadequate vision correction—or none at all.[1] This staggering number is on the rise, the study authors note.[1] Many attribute the global high prevalence of unmanaged presbyopia to a lack of awareness and access to affordable treatment in the developing world, but research identifies the same unmet need in developed countries, too.[2]



Discussing presbyopia's impact on your patient's vision early will reap many benefits.

Multifocal contact lenses can—and should—be the answer to many presbyopes' vision concerns, yet one study found contact lens prescribing to patients 45 years and older was only 37%.[3] In addition, about 42% of spectacle wearers are presbyopes, compared with just 24% of contact lens wearers. Still, spectacle-wearing presbyopes prefer contact lenses as often as non-presbyopes and demonstrate an interest in contact lens wear.[4] This disparity between patient interest in contact lens wear, the number of multifocal prescriptions and the growing number of presbyopes is a critical opportunity for optometry.

Here is a look at how optometrists are overcoming the multifocal prescribing hurdles to provide their presbyopic patients the best vision correction options available.

Know Your Audience

Although presbyopes are still patients in their 40s and older, they aren't the same patients they once were. Retirement-age Americans are feeling healthier than ever, with more than 75% aged 65 or older reporting being in good, very good or excellent health—a demographic that has grown steadily over the past 35 years.[5] Of workers older than 65, 77% said they had no limitations on the kind of work they can do.[5]

"Today's presbyopes are active folks who lead demanding schedules filled with travel, fitness and fun," Pamela Lowe, OD, of Professional Eye Care Center in Illinois, says. "They are and want to look and feel more youthful."

These presbyopes make the best multifocal lens candidates because "those who work in a dynamic environment with various working distances will appreciate the freedom from reading glasses," according to Robert Ensley, OD, of Gaddie Eye Centers in Kentucky. Patients with an active lifestyle who don't have an unrealistic demand for their vision will make excellent patients, he adds.

According to Shalu Pal, OD, who practices in Yorkville, Toronto, ideal patients include those who are frustrated with reading glasses or progressives and active patients who need hands free and head-tilting free vision—patients who want easy, functional vision at all ranges and all angles.

"I like to recommend multifocals

to anyone who does outdoor activities, so they don't have to constantly wear glasses," says Stephanie Woo, OD, of Havasu Eye Center in Arizona. Many presbyopic patients in Dr. Woo's practice who wear multifocals use them part-time, only using them when they are at the gym, hiking, boating or playing sports.

"Many times patients feel if you wear contact lenses, it is a full-time commitment," Dr. Lowe adds. "Let patients know they can be a weekend warrior or occasional social wearer, especially with the single-use lenses now available."

"Sometimes these patients slip through the cracks because they have never expressed interest in contact lens wear before or have never been asked," Dr. Ensley points out.

When considering a patient's vision correction options, optometrists should not assume that presbyopic status, wear time, refractive error, or gender are factors that preclude a patient from being interested in multifocal contact lens wear.[4]

Educate Early

When a presbyopic patient is in your chair, you have their undivided attention and a prime opportunity to talk to them about the condition. Having a conversation early about presbyopia's impact on their vision will reap many benefits and can help to assuage their fears.

"Ask about near work demand, eye fatigue and trouble focusing," Dr. Ensley suggests. "Even if the patient isn't struggling at the time, I might mention that when those issues arrive we have plenty of options for them to remain successful with contact lenses."

Dr. Lowe suggests getting into the habit of mentioning multifocal contact lenses as a viable option for every potential presbyopic patient. It should become an important example of how you can correct your patient's distance and near visual needs, she says.

Dr. Geffen discusses all three vision correction options—glasses, contacts or surgery—so the patient knows he is the source for all of their vision-related questions and concerns.

Others such as Dr. Pal help provide a long-term plan at the end of their assessment if they see presbyopia becoming a problem. "I offer them their multifocal options that best match their prescription regardless if they have asked for contact lenses or not," she says.

Temper Expectations

Presbyopia reduces vision-related quality-of-life, and although various vision correction options can improve it, nothing can restore vision to its pre-presbyopic state.[6]

"We give proper expectations to our patients and try to under promise and over deliver," David Geffen, OD, of Gordon Schanzlin New Vision Institute in San Diego, says.

Dr. Woo suggests a handout outlining what to expect could help patients be fully aware of what vision outcomes to expect.

"Once a patient understands the advantages and limitations they will experience with lenses, they become an engaged partner with the doctor in achieving the best outcome," explains Dr. Lowe.

Choose the Right Lens

Multifocal contact lens materials, designs and replacement schedules have come a long way from the time Dr. Lowe started fitting contacts more than 30 years ago. The optics and comfort have improved considerably, and there are lens choices to fit every lifestyle, she says. "Doctors need to stay educated on the latest multifocal advances so they know what's available to fulfill patient needs and expectations," Dr. Lowe says. To choose the right lens, clinicians first need to know about the patient's visual environment.

"Teasing out when they would like to wear lenses, what visual activities they perform and what their visual environment entail are key," she explains.

"We first go through a series of questions about lifestyle and then decide on which distances are most important for the patient," Dr. Geffen explains. He focuses first on providing the patient with excellent distance vision and tells them they can modify the near at the next visit, if necessary. "I like this approach because if the patient is unhappy with the distance they may not even wear the lenses to give them the chance to adapt," he adds.

Dr. Ensley emphasizes the importance of a lens's replacement schedule. "New contact lens wearers almost always get daily disposable lenses to start them off on the path of better compliance," he says. Once they move to a multifocal, daily disposable lenses give patients

The Outliers

There will always be presbyopic patients who won't do well in multifocal lenses or have unrealistic vision correction expectations. While everyone wants be independent of eyeglasses, some might still need it as a supplement.

"Emmetropes or very low hypertropes desperately want to be free from readers, but typically want to compare the distance vision with multifocals to what they are used to unaided," Dr. Ensley notes. Patients with occupations that require extensive distance demands, such as pilots, engineers and truck drivers, might not be the best fit for full-time multifocal lens wear.

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the flexibility to choose part-time wear if they want and still remain in an ideal modality.

However, if the patient is already in a monthly lens and comfortable with the material, Dr. Ensley will start with that brand's multifocal. If that brand is unsuccessful, he will switch to another brand with a different optical design.

Soft vs. GP. A key decision involves whether to use soft or gas permeable (GP) multifocal lenses. Each modality brings benefits to the table, the experts say.

Soft lenses offer good initial comfort, and the daily disposable lenses are a excellent option for the occasional or social wearer, according to Dr. Lowe.

However, "rigid multifocal lens options are great for more customized designs to give the patient better clarity, especially by having options in translating designs vs. the simultaneous vision of soft lenses," she adds.

A patient's prescription will ultimately determine whether they qualify for a standard soft multifocal lens or a custom GP multifocal lens, Dr. Woo says. "Soft lenses are easier to fit, easier to stock in office and require less chair time, so patients can leave wearing them, but they can offer less precise vision and more limited parameters," she notes.

A more customizable GP lens can help patients with higher prescriptions but requires more chair time and a delay for ordering. "GPs are also associated with higher fitting fees and cost," Dr. Woo adds.

Dr. Ensley finds that patients pursuing a GP lens option need to understand comfort requires an adaptation period, but motivated patients will do quite well. "GP lenses also correct astigmatism of 0.75D or more much better than soft multifocals," he explains.

However, if a presbyopic patient interested in multifocals is already wearing GP lenses, offer them an option in that type, Dr. Geffen says. "Most of my patients do not want to go with GPs if they have never worn lenses, so most start with soft," he notes. "If I cannot satisfy their visual need then we try GPs." Dr. Geffen also finds that the older patients have somewhat reduced corneal sensitivity, and often do well with GP multifocals.

Fitting. The entire multifocal process requires a higher level of commitment from both the patient and optometrist. Before she begins a new fit, Dr. Pal provides her patient with all of the details of the process: the time commitment, the length of the visits, vision expectations and costs. If, for any reason, the patient does not want to proceed because one or more of the topics do not resonate with them, her team knows prior to starting the fit.

"If you properly prepare your patients with more information, they are more likely to be on board with your directions and comply because they made an informed decision to proceed," Dr. Pal says.

"At the initial exam we find a pair of lenses that are comfortable and perform well visually in the office, Dr. Ensley says. "Then we send the patient out in the real world to try the lenses." Dr. Ensley then follows up one to three weeks later to troubleshoot, if needed.

Staff, especially contact lens technicians, well educated in fitting multifocals and aware of the options can contribute to a successful fit, according to Dr. Lowe.

She believes that another key factor is having the patient know proper care and maintenance of the lenses they wear. "A well-trained contact lens technician can educate and train the patient efficiently and effectively," she adds.

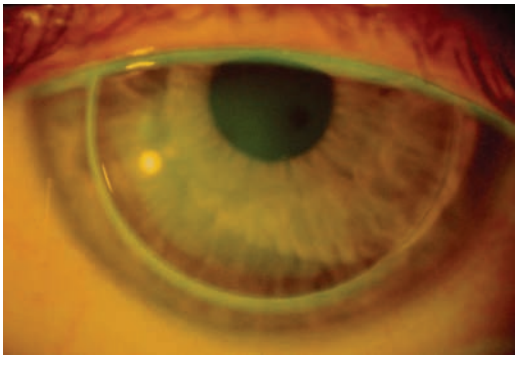
Common Apprehensions

Despite better lens materials and designs, many patients still struggle with multifocal lens wear. One study found 15% of presbyopic patients permanently discontinued contact lens wear, reporting poor vision (38%), discomfort (34%), convenience (20%) and cost (6%) as the primary reasons for discontinuation.[7]

Patients lost to contact lens wear had a worse overall opinion of their distance, intermediate and near vision compared with subjects still wearing their contact lenses.[7] If you address these three issues, you can ease your patients' concerns and boost your multifocal fitting success:

Comfort. Presbyopes are a unique group of contact lens wearers with distinct visual demands compared with non-presbyopic patients. But good vision must be paired with comfort, too. One study found that presbyopes of all refractive errors prefer contact lens correction when they can achieve both good vision and comfort.[4]

"Patients might ask about cost,



Consider GP multifocal lens options for more customized designs that give better clarity.

but if they are happy with comfort and vision, I seldom have patients go back to single vision on the basis of cost alone,"says Dr. Ensley.

"We connect comfort with compliance," Dr. Geffen says. To make sure his patients are as comfortable as possible in their lenses, he focuses on the importance of hygiene and follow-up care in the initial fitting visits.

Discomfort remains the main factor contributing to contact lens discontinuation in all age groups, but if you address it at the outset, the hope of improved vision can motivate a contact lens dropout to try multifocals again.[4]

Anxiety. Some patients stop wearing contact lenses because of an unsatisfactory previous experience, and the thought of trying them again can be daunting. "A past failed experience can take all the motivation out of going down a fitting path again," Dr. Lowe says.

For others, wearing contact lenses is a new experience and they are resistant because they don't understand or are nervous about them, according to Dr. Pal. "Fear of putting lenses in and taking them out is the primary reason," she says.

"Some new wearers really struggle with contact lens handling and are not as patient as kids and young adults," Dr. Ensley adds.

Perhaps the best way to quell a patient's anxiety—whether new to contact lenses or returning to the modality—is to take the pressure off of contact lens wear being a full-time commitment at that moment. "Worst-case scenario, they don't work for you, and you are just back to what you are doing now,"says Dr. Woo.

Dr. Geffen tells his patients that there is no risk in trying the lenses, as his practice's policy is to not charge for lenses until the evaluation period is finalized. They offer a test drive in contacts for a few minutes in the office to give patients the chance to experience how good the lenses have become.

A presbyopic patient may not take on lens wear as easily as a patient who started wearing lenses in adolescence.

Awareness. Some patients are not aware contact lenses are an option for them. "Some do not realize these lenses even exist, and others were told by prior doctors these lenses do not work," Dr. Geffen says. "I let them know my vast experience in using these lenses and my high success rate."

It's the OD's job to educate patients on their many vision correction options, and leaving multifocal contact lenses off the list isn't an option, according to Dr. Pal.

"If a practitioner doesn't fit multifocal lenses, they should still educate patients on multifocals being a source of vision correction, and then refer them to an optometrist who does multifocal lens fittings," Dr. Woo advises.

"If I were a patient and my doctor did not educate me on all of my options, and I found out there were other options, I would be upset and possibly not trust that doctor in having my best interest," she adds.

Pushing Forward

Multifocal lens designs are improving by the day, but they aren't perfect. Optometrists look forward to new soft lenses that improve upon the visual quality of today's options.

"Decentered optics and variable zone sizes can certainly improve the visual quality of a multifocal lens," Dr. Ensley says. "Currently these can only be adjusted in custom lenses."

Dr. Pal also believes that more customization could be cost effective, especially when designing a multifocal lens for patients who are unsuccessful with traditional methods.

Despite the disconnect between presbyopes' interest in contact lens wear and multifocal contact lens prescribing, many optometrists who work with these lenses are optimistic regarding the modality's future.

"As the products continue to improve and the optics become more crisp, I'm excited about being able to help more of our patients," Dr. Pal says.

"We are seeing a great deal of research in the area of multifocals," Dr. Geffen says. "I truly believe that multifocal lenses are best for most of our patients, and this positive attitude pervades our entire office." ■

1. Fricke TR, Tahhan N, Resnikoff S, et al. Global prevalence of presbyopia and vision impairment from uncorrected presbyopia. Ophthalmology. 2018;125(10):1492-9.
2. Wolffsohn JS, Davies LN. Presbyopia: effectiveness of correction strategies. Prog Retin Eye Res. 2019;68(1):124-43.
3. Morgan PB, Efron N, Woods CA. An international survey of contact lens prescribing for presbyopia. Clin Exp Optom. 2011;94(1):87-92.
4. Rueff EM, Bailey MD. Presbyopic and non-presbyopic contact lens opinions and vision correction preferences. Cont Lens Anterior Eye. 2017;40(5):323-8.
5. Plews L. Older americans in the workforce. United Income. unitedincome.com/documents/papers/United_Income_Older_Americans_in_the_Workforce.pdf. Accessed July 8, 2019.
6. McDonnell PJ, Lee P, Spritzer K, et al. Associations of presbyopia with vision-targeted health-related quality of life. Arch Ophthalmol. 2003;121:1577-81.

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The Dangers and the Diagnosis of CLMK

Despite many advances, the threat of contact lens-related microbial keratitis has not retreated. **By Jaya Sowjanya Siddireddy, PhD**

Microbial keratitis (MK) is a rare and acute corneal disease that can lead to severe visual disability.[1] The severity of the infection depends on the underlying condition of the cornea and the pathogenicity of the microbe. Although it used to occur mostly with predisposing factors such as ocular trauma and ocular surface diseases, an increase in contact lens wear over the last decade has caused a dramatic rise in the prevalence of contact lens-related microbial keratitis (CLMK).[2-7] Prognosis of this infectious disease is usually poor if aggressive and appropriate therapy is not initiated promptly.[2,8,9]

Contact lens wear has been identified as one of the major risk factors for MK, affecting almost five in 10,000 wearers.[10] Because of the massive number of contact lens wearers worldwide, the morbidity due to corneal ulcer has public health consequences.[11]

Epidemiology

The incidence of CLMK is two to four per 10,000 contact lens wearers per year for daily soft contact lens wearers and 20 per 10,000 for overnight soft contact lens wearers.[11-15]

Despite the advent of silicone hydrogel lenses that reduce the

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Educational Objectives: After completing this activity, the participant should be better able to:

- Discuss the incidence and types of MK, including bacterial, fungal and *Acanthamoeba* infections.
- Identify the many modifiable and non-modifiable risk factors associated with CLMK.
- Provide prompt and correct diagnosis of MK, followed by effective pharmaceutical therapy or corneal procedure.
- Educate patients about proper contact lens wear, lens replacement, and cleaning and disinfecting lenses and cases.

Target Audience: This activity is intended for optometrists engaged in the care of patients with contact lens-related microbial keratitis.
Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by the Postgraduate Institute for Medicine and Review Education Group. Postgraduate Institute for Medicine is jointly accredited by the Accreditation Council for Continuing Medical Education, the Accreditation Council for Pharmacy Education, and the American Nurses Credentialing Center, to provide continuing education for the healthcare team. Postgraduate Institute for Medicine is accredited by COPE to provide continuing education to optometrists.
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hypoxic stress to the cornea, especially during overnight wear, the risk for CLMK has not decreased.[16] Although daily disposable contact lenses reduce the risk of corneal infiltrative events and severe MK, epidemiological studies have not shown a reduced incidence of MK with daily disposable contact lenses.[11,17-19] Of all the lens types, use of gas permeable lenses on a daily wear schedule have the lowest incidence of MK.

Bacteria, especially *Pseudomonas* species, are the most common pathogens involved in MK, while far fewer—but more severe—infections are caused by *Acanthamoeba* and fungi.[20-23]

The incidence of contact lens-related *Acanthamoeba* keratitis is one to five per one million soft contact lens wearers in Europe and the United States.[24] Epidemiological studies have confirmed the use of non-sterile water to clean or store contact lenses and showering or swimming while wearing contact lenses as risk factors for contact lens-related *Acanthamoeba* keratitis.[25-29] *Acanthamoeba* keratitis has also been linked to both domestic and surface water contamination.[25,30,31]

Fungal keratitis is rare in contact lens wearers, typically accounting for about 5% of all CLMK.[20] *Fusarium* is a filamentous fungus mainly found in soil and plants.[32] Outbreaks of contact lens-related *Fusarium* keratitis were reported between 2006 and 2007, primarily associated with a specific contact lens disinfection solution (ReNu with MoistureLoc, Bausch + Lomb).[33,34] Researchers reported that 60% (10/15) of those with the disease used water to clean contact lens storage cases.[35] Following the withdrawal of this disinfection solution from the market, the rates of disease returned to pre-outbreak levels.[36]

Corneal infiltrative events range from mild asymptomatic corneal infiltrates to severe MK.[37] Sterile keratitis is 200 times more prevalent than MK.[38]

The most prominent contact lens-related risk factor for corneal infiltrative events is extended or overnight lens wear.[13,39-42] Research reports an nnnual incidence for severe keratitis for overnight lens wear at 96.4 per 10,000 wearers compared with 6.4 per 10,000 daily lens wearers.[42] This suggests an 8.4-fold increase in risk for developing corneal keratitis due to overnight lens wear.

Evidence also suggests a higher rate of contact lens contamination with corresponding increases risk for corneal infiltrative events in lens wearers with two or more years of experience compared with neophytes or those with less than two years of lens wear experience.[11,18,19,41,43]

Showering with contact lenses or rinsing contact lenses with tap water is associated with an increased risk of contact lens-related corneal infiltrative events.[40] Most contact lens disease-causing pathogens are waterborne, so water exposure during lens wear is a serious concern.

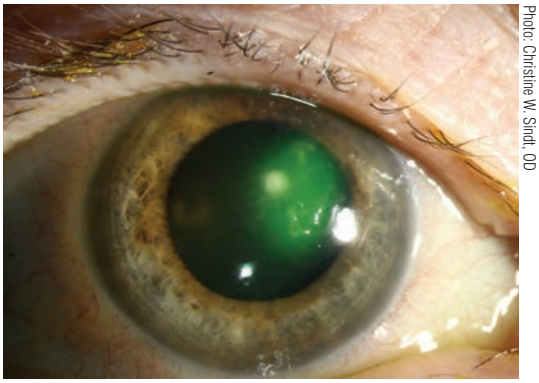

Photo: Christine W. Sindt, OD

***Serratia marcescens* (as well as *P. aeruginosa* and *S. aureus*) can form biofilms on contact lenses that are resistant to contact lens disinfecting solutions.**

Pathophysiology

Research to understand the mechanisms of ulceration is ongoing.[44] Several factors play a crucial role in contact lens-related keratitis:

Bacterial adherence to the lens surface and reduced resistance of the cornea to infection. In bacterial keratitis, bacteria gain access to the corneal stroma through an abnormality or defect in the corneal surface causing an inflammatory response, which results in loss of transparency.[45,46] First, penetration of corneal epithelium (more severe than punctate fluorescein staining) needs to take place in the presence of pathogenic levels of bacteria to initiate MK.[47] In contact lens wear, hypoxic conditions may increase bacterial binding, compromising corneal integrity and impairing wound healing.[11,45,48] Ocular biochemistry changes underneath the contact lens can also predispose the lens wearer to infection.[44] Interaction with the contact lens can override the cornea's defense mechanisms and increase the rate at which the microbes adhere to the ocular surface, leading to MK.[44,48,49] The rate of progression of MK depends on the virulence of the offending pathogen and host factors.[50]

Formation of biofilm on lenses and storage cases. Contact lenses are a fertile surface for bacterial adhesion and biofilm formation.[44,48,49] As such, adhesion of bacteria—particularly *Staphylococcus epidermis* and *P. aeruginosa*—to contact lenses is a major risk factor.[44,49] Contact lens cases are associated with more contamination than lenses or lens care solutions.[48,51] Notably, the same strains found in corneal ulcers have been isolated from contact lens cases.[51] The level of contamination rate is associated with the age of the lens case.[11] The upper rim of the lens case is ideal to harbor gram-negative bacteria due to its air-liquid interface, increasing the likelihood of biofilm

formation.[51] Contact with this area during lens handling can severely contaminate the lens.[51]

Resistance of microorganisms to disinfecting systems. Not performing the "rub and rinse" cleaning technique curtails the removal of microbes and creates a carryover effect from lens case to lenses, leading to an increase in microbial virulence and survival rate.[44,48,51]

Stagnation of tear film behind contact lenses. The presence of debris, toxins and antigens trapped between the contact lens and corneal surface, and their prolonged exposure, can increase the risk of infection.[52] This could be the reason for lower CLMK risk with rigid gas permeable lenses, due to higher post-lens tear exchange, compared with soft contact lenses.[14,53-56] In addition, epithelial cell proliferation and migration are slower in contact lens wearers, so epithelial cells that reside for a longer time on the corneal surface may initiate inflammation.[57-61] Also, tear exchange is drastically reduced in soft contact lens wear.[44,45,62] Although the impact of tear exchange is not completely understood, the mean tear elimination rate is 50% less in eyes wearing conventional contact lenses compared with eyes of non-lens wearers.[44,48] Incidentally, silicone hydrogels provide better tear exchange than conventional lenses.[62]

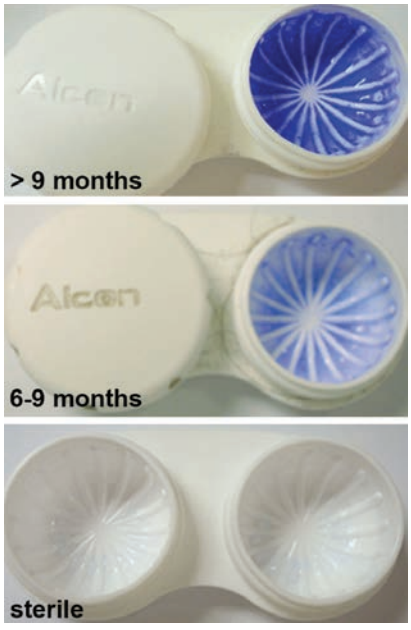

Photo: Danielle M. Robertson, OD, PhD

Staining of a contact lens storage case shows the correlation between age and the level of microbial contamination. Staining was evident as early as six months and showed a dramatic increase in intensity beyond nine months.

Ocular surfaces of contact lens wearers, compared with non-lens wearers, harbor greater numbers of gram-negative bacteria and fewer numbers of gram-positive bacteria.[63,64] Clinicians should consider this evidence and provide adequate prophylactic antibiotic treatment against gram-negative organisms, especially *P. aeruginosa*, which is the most prevalent gram-negative pathogen in CLMK.[63,64]

Gram-positive bacteria are the most common organisms in CLMK, especially in temperate climates.[10,65] These organisms include coagulase-negative *Staphylococcus* (including *S. epidermidis*), *S. aureus* and *Streptococcus pneumoniae*. Coagulase-negative *Staphylococcus* is found on the lid margins and is considered normal flora, but it can become infectious in some instances.[66]

P. aeruginosa, *S. aureus*, and *Serratia marcescens* can form biofilms on contact lenses that are resistant to environmental challenges and contact lens solutions.[67-69] *P. aeruginosa* and *S. epidermidis* are also found to adhere and replicate on contact lenses *in vitro*, on both silicone hydrogel and regular hydrogel materials.[70]

The microbes that are isolated from contact lenses originate from the lid margins, conjunctiva, hands, lens cases, care solutions and the water supply.[71-74] Pathogenic organisms are associated with MK, so it is reasonable to assume that if the lid margins, conjunctiva, tear film, contact lens case, care solutions and the patient's hands are contaminated with these pathogens, the risk of developing MK increases.

Modifiable risk factors. Several modifiable risk factors for MK and corneal inflammatory events are associated with poor compliance. Modifiable risk factors are those that a lens wearer has some control over, as opposed to non-modifiable factors such as age and sex.

The major modifiable risk factors identified in epidemiological studies of CLMK are overnight wear and poor hygiene, including omission of or infrequent lens disinfection, omitted or infrequent lens case cleaning, omission of handwashing before handling lenses and smoking.[11,13,75-78] Research estimates that overnight wear and poor hygiene account for about 43% and 33% of attributable risk for developing CLMK.[79]

Sleeping in contact lenses is another commonly reported contact lens risk behavior and one with a high relative risk for corneal infection.[53,80] Research shows sleeping in lenses is a risk factor regardless of lens material and frequency, with even occasional overnight use conferring risk.[77]

In severe keratitis, contact lens case hygiene (cleaning and replacement) accounts for 63% of the population-attributable risk. In addition, swimming is a risk factor for *Acanthamoeba* keratitis, and travel is a risk for severe infection, thought to be related to disruption of routine.[25,65,81,82]

The risk of infection in extended wear is higher with increased wearing time and less experience.[11,53,55,83] Wearers considering extended wear should be motivated and aware of this increased risk; however, it is important to balance the risk with other lifestyle risks. Some individuals, such as shift workers and those with busy lifestyles, may feel the convenience of extended wear outweighs

the increased risk.

Despite a higher unadjusted incidence rate for daily use of silicone hydrogel contact lenses compared with hydrogel contact lens use, multivariate analyses have not identified lens material as an independent risk factor.[11,53]

Non-modifiable risk factors. Non-modifiable risk factors include younger age, male gender and socioeconomic class.[53,56,78] Systemic risk factors include self-reported poor general health, diabetes and thyroid disease.[83,84]

More recently, an increased exposure (number of days of wear per week) in daily wear, hypermetropia, obtaining lenses via the internet or mail order and the early period of lens wear have been identified as additional risk factors with contemporary lens types.[11,53] Males tend to be more prone to complications in contact lenses, which may be due to increased non-compliance and also a reluctance to seek care.[85,86]

Genetic differences in contact lens wearers can affect the susceptibility and severity of keratitis. Small mutations (single nucleotide polymorphisms) of interleukins, inflammatory mediators and defensin (an antimicrobial peptide) have been isolated as contributory.[87-89] This may mean some people have a degree of innate protection against infection and inflammation when wearing contact lenses, but this protection is much lower than the risk of poor hygiene and overnight wear.

While research shows that risk-taking personality styles are associated with non-compliance in contact lens wearers, no studies indicate whether risk taking is associated with a susceptibility to corneal inflammation and infections.[90]

There are likely to be many drivers for non-compliance, and not being aware of the risks or understanding the consequences of non-compliance are likely to be key factors among some lens wearers.

Water and Contact Lenses Don't Mix

Water exposure during contact lens wear is associated with multiple complications ranging from sterile corneal infiltrates to more severe sight-threatening infections. Clear, unequivocal guidelines/recommendations to avoid all water exposure—including handling contact lenses with wet hands, rinsing/storing contact lenses or storage cases in tap water, and showering with contact lenses—are needed.[133] Swimming with contact lenses should be done with protective goggles or using daily disposable contact lenses, which can be discarded immediately after swimming. Active dissemination of these guidelines to contact lens wearers through all stakeholders—including contact lens manufacturers, professional organizations, and contact lens practitioners—is recommended.[133]

Diagnosis

Bacterial keratitis. A substantial inflammatory response along with replicating necrotic cells and microbes form infiltrates that are mostly irregular and focal, surrounded by diffuse inflammation and edema of the cornea.[22,91] It is unusual to find a bacterial keratitis with no apparent focal epithelial defect.[22,91] In some cases, a focal infiltrate can be absent and an epithelial defect or melting stromal keratitis may be the only signs of infection.[22,91] Due to inflammation of the surrounding cornea, causing scattering of light and photophobia, vision can be affected even if the lesion is not central. Other signs such as lid swelling, conjunctival chemosis and anterior chamber reaction are common in bacterial keratitis.[91]

P. aeruginosa is difficult to neutralize due to its virulent structure, adaptability and high rate of survival under various conditions.[44,45] Along with intense immune response, *P. aeruginosa* also produces enzymes such as protease and elastase, which digest collagen, contributing to corneal melting and perforation.[92] Hallmark signs are corneal edema, a ring abscess (defined as a circular infiltrate with a less dense center) associated with larger lesions and presence of hypopyon.[93]

Acanthamoeba *keratitis.* Subtle corneal signs with or without symptoms of pain can be found in early infection.[94] The early signs include an epitheliopathy with or without a dendritic appearance. Infiltrates running along the nerves from the periphery (perineural infiltrates) are virtually pathologic for *Acanthamoeba* keratitis, occurring in around 60% of cases.[94,95] In later stages of infection, the involvement of central stromal infiltration occurs in around 20% of cases.[94] Scleritis occurs in 15% to 20% of cases.[94] If scleritis develops, patients often report severe, persistent pain.[94] Early diagnosis and prompt, appropriate medical attention improves the prognosis of disease. A delay in effective therapy for more than three weeks will likely worsen the prognosis.[96,97]

Fungal keratitis. Fungi are opportunistic organisms that do not infect a healthy cornea. However, after trauma and inoculation, fungi can proliferate, leading to tissue damage and the disruption of host defenses.[98] Fungi secrete toxins, such as proteases, that aid tissue destruction and allow the fungi to penetrate deep into the cornea. The fungal hyphae and pseudohyphae both form large structures that cannot be fully ingested by polymorphonuclear leukocytes and macrophages. Activation of resident corneal cells and host inflammatory cells that attempt to neutralize the invading organisms adds to the tissue destruction.[98]

The fungal species that cause keratitis are the yeast, *Candida* sp., and filamentary fungi such as *Aspergillus*

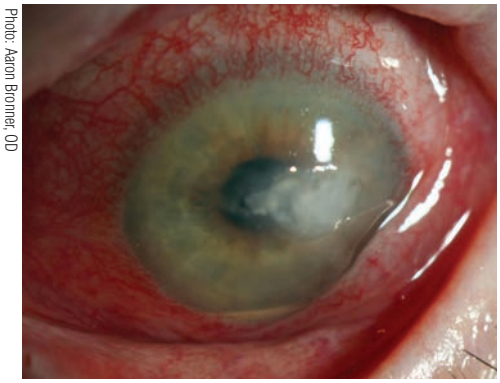
Photo: Aaron Bronner, OD

Differentiating the clinical signs of fungal keratitis (seen here) from more common bacterial ulcers can be difficult, especially with yeast fungi such as *Candida*.

sp. and *Fusarium* sp. Contact lens use and trauma are mainly associated with the filamentary fungi, while ocular surface disease is commonly caused by *Candida* sp.[99]

Research recently shows microsporidia is a parasitic fungus causes keratoconjunctivitis. It has been found in contact lens wearers, but is usually associated with soil/mud and occurs in immunocompromised patients.[100]

Differential diagnosis. Distinguishing between Acanthamoeba keratitis and herpes simplex virus (HSV) stromal keratitis can be difficult in the early stages, and around 50% of *Acanthamoeba* keratitis cases are misdiagnosed as HSV keratitis.[101] Epitheliopathy, pseudodendrites and stromal inflammation in *Acanthamoeba* keratitis are often confused with HSV stromal keratitis. These cases are then treated using corticosteroids. However, use of corticosteroids before the initiation of anti-amoebic drugs is independently associated with a four-fold increased risk of poorer outcomes.[101]

Differential diagnosis of the clinical signs of fungal keratitis from more common bacterial ulcers can be difficult, especially with yeast fungi such as *Candida*. Yeast infections tend to present as discrete infiltrates and overlying defects, but unlike most bacterial infections, the onset of yeast-like infections tends to be slow.[102] The common features of fungal keratitis cases are serrated ulcer margins with raised slough and a dry textured infiltrate that is usually white or gray (not yellow) and satellite lesions.[103] Immune rings (ring infiltrates/Wesley rings) are not pathognomonic for fungal keratitis and can occur in other forms of keratitis, including those caused by *Acanthamoeba* and bacteria.[94,103]

Other corneal inflammatory events, such as infiltrative keratitis driven by microbial products and presumed hypersensitivity reactions, typically present with a non-specific inflammatory response that can be local or general.[37] Sometimes these can mimic the immune corneal response seen later in the course of adenoviral keratoconjunctivitis.[104] Careful history of the redness and symptoms, as well as the swelling of the lymph glands and the more diffuse, fluffy pattern of the infiltrates that are seen in viral conditions, will aid diagnosis.[100]

In localized sterile inflammation of the cornea, such as marginal keratitis and contact lens-related peripheral ulcer, the redness is usually sectorial corresponding to the corneal lesion and associated with lower pain, a staining diameter greater than the infiltrate diameter (generally <1mm), and possibly a mild anterior chamber reaction. However, a case of infection shows diffuse and more intense redness. In moderate/severe infection, the redness is deeper and the lid as well as anterior chamber are likely to be involved.[105]

Diagnostic techniques. In cases of suspected MK, corneal scraping is usually the first step to collect samples containing the causative organisms. These samples are then taken for culture or molecular testing using polymerase chain reaction.[66] Culture results are available usually within two to seven days.[66] Fungi typically take longer than bacteria to grow.

Drug sensitivity testing is performed from the cultured organisms, which is important to guide therapy in nonresponsive cases.[66] If antibiotics have been started in nonresponsive cases, the treatment is stopped for 24 hours to maximize the chance of organism recovery.[22]

Corneal biopsy can be useful for recalcitrant cases.[22] The biopsy is deeper than a corneal scrape and can be done freehand or with a trephine. This is particularly useful in fungal keratitis as filamentary fungi may proliferate in the deeper corneal layers, so surface scrapes may not capture the organism. Typically for a biopsy, half the material is sent for microbial culture while the other half is sent to histology for tissue processing.[22] Culture tends to be positive in around 50% of cases of clinically presumed bacterial keratitis and slightly higher for *Acanthamoeba*.[22]

Management

The goal of treatment is to rapidly eradicate the pathogen, so clinicians should assume CLMK is bacterial unless proven otherwise.[106-108] The gold standard treatment for corneal ulceration is fortified antibiotics, such as cefazolin 5% and tobramycin 1.3%, or fourth-generation fluoroquinolones (either ciprofloxacin or ofloxacin) as monotherapy.[109,110]

Research has observed an alarming trend of increased resistance to antibiotics over the past two decades.[111-113] Pathogenic strains such as methicillin-resistant *S. aureus* (MRSA) and methicillin-resistant *S. epidermidis* (MRSE) are becoming more prevalent, and many strains show multidrug resistance, including resistance to both earlier and current

generation fluoroquinolones.

The most recent addition to topical ocular fluoroquinolones is Besivance (besifloxacin 0.6% ophthalmic suspension, Bausch + Lomb), which decreases resistance due to its unique molecular structure with an increased antibacterial potency.[114]

Bacterial keratitis. Use of topical antibiotics is the standard treatment for most CLMK. For mild to moderate cases, empirical therapy using a broad spectrum fluoroquinolone is generally employed.[22] Ciprofloxacin and ofloxacin are the mainstay fluoroquinolone antibiotics. The therapeutic regimen for bacterial keratitis includes use of a cycloplegic agent and frequent use of ciprofloxacin 0.3% antibiotic drops every 15 minutes for four hours, followed by every 30 minutes for four hours, and then every hour around the clock for at least 24 hours. Depending on the severity of the ulcer and the clinical response, ciprofloxacin ointment can be substituted for the drops at a lower frequency during the night after one to two days of therapy. In a severe ulcer, one to two weeks of therapy may be required for a complete therapeutic response.[115]

The first period is the sterilization phase where the organism is neutralized, followed by the healing phase.[22] The sterilization phase usually takes three to five days. Often a dilating agent is used to relax the ciliary body and prevent ciliary spasm, stabilize the blood aqueous barrier and to help prevent posterior synechiae. Review is recommended within 24 hours with daily follow-up until improvement is clearly established. In a few days, the eye may be more inflamed due to an inflammatory response to dead organisms, but it is important that there is not a dramatic deterioration in status.[22]

Some centers tend to treat severe cases with fortified antibiotics that are compounded by an accredited pharmacy. The combination is often a cephalosporin for gram positive coverage, such as cephazolin, and an aminoglycoside for gram negative activity, either tobramycin or gentamicin. A randomized clinical trial conducted in Australia revealed that monotherapy with ofloxacin 0.3% and moxifloxacin 1% had similar efficacy and safety compared with fortified tobramycin 1.33% and cephazolin 5% antibiotics.[116]

In many cases, topical steroids are used in the management of bacterial keratitis in an attempt to limit scarring as a lot of the damage in keratitis occurs due to the inflammatory response. Topical steroids also decrease pain and may improve quality of life. A typical regimen introduces steroids once the ulcer begins to re-epithelialize, which is an indication that the antibiotic therapy is effective.

Tapering steroids is essential to avoid a rebound effect. The schedule of steroids, concurrent with antibiotics, might be QID for one week, BID for one week, QD for one week, and then cease. The main concerns for using topical corticosteroids in bacterial keratitis are delayed re epithelialization, recurrent infection and increased risk of perforation.[8]

Acanthamoeba *keratitis.* There are no currently approved medications for treating *Acanthamoeba* keratitis. However, biguanides (polyhexamethylene biguanide 0.02% to 0.06% and chlorhexidine 0.02% to 0.2%) and diamidines (propamidine isethionate 0.1% and hexamidine 0.1%) are the most effective cysticidal agents for cases of *Acanthamoeba* keratitis.[117] None of the treatments are licensed for use in the United States but case series have shown good evidence that they are effective *in vivo*.[118]

Fungal keratitis. This is often highly invasive, and antifungal agents tend to be fungistatic, leading to prolonged treatment and often surgical intervention.[119] Natamycin 5% is usually the initial agent of choice. The Mycotic Ulcer Treatment Trial Phase 1 (MUTT I) found natamycin to be more effective than voriconazole for filamentary fungus, such as *Fusarium* and *Aspergillus*.[119,120] Also, MUTT II demonstrated that the addition of oral voriconazole to topical natamycin does not improve outcomes.[121] Oral voriconazole can also be prescribed for deep fungal ulcers and scleritis, although patients commonly suffer from visual disturbances and will also require liver function tests.[122]

Anti-fungals are less effective in deeper layers of the cornea. In early phases, rapid progression of fungal keratitis is due to factors relating to organisms such as large fungal inoculum. In later stages, the combination of agent and host factors lead to resistance to anti-fungals.[99]

Chlorhexidine, an antiseptic for treating *Acanthamoeba* keratitis, can be used as an alternative to natamycin.[121,123] For yeast infections,

Photos: Aaron Bronner, OD

A staphylococcal ulcer typically appears as a discrete infiltrate with well-defined borders and often surrounding edema.

such as *Candida*, which is more common in patients with a history of ocular surface disease and in immunosuppressed patients, topical amphotericin B is recommended.[119] Echinocandins (such as caspofungin and micafungin) can be added.[119] Some evidence suggests that fluoroquinolones may be synergistic with amphotericin.[124] Often multiple agents are used to offer maximum coverage.[119]

Topical steroids should not be used during the treatment of a fungal infection. Corticosteroids induce fungal growth by suppressing ocular immune mechanisms.[125] Topical cyclosporine A, however, may be synergistic to fungal therapy because it inhibits filamentary fungal growth.[126,127]

Therapeutic penetrating keratoplasty. In severe cases of corneal infection, therapeutic penetrating keratoplasty can be considered to prevent the spread of the pathogen to other parts of the eye, especially in filamentary fungal cases. However, this involves the risk of pathogens entering the anterior chamber during surgery.[128] Furthermore, corticosteroids used to limit graft rejection may exacerbate the growth of fungus that may remain in the eye following surgery.[128] In *Acanthamoeba* keratitis, therapeutic keratoplasty is limited to impending or perforated corneas, as recurrence often occurs and outcomes are far better following optical keratoplasty in a quiet eye to restore vision.[129]

Prevention

Contact lens practitioners play a crucial role in the management and education of healthy lens wearers.

Research suggests that 40% to 70% of contact lens wearers are noncompliant.[130] Higher rates of complications have been associated with men, teens/young adults, smokers, longer periods of lens wear, lack of hand washing, and internet purchase of lenses.[49,51,130,131] Non-compliance with manufacturers' recommended frequency of replacement of contact lenses is found to be highest among teenagers and wearers of non-silicone hydrogel lenses.[130]

Contact lens wearers using hydrogen peroxide solution may be more compliant with their lens replacement schedule due to the complex and demanding care regimen.[130] Daily disposable lenses were associated with lowest rate of complications in general.[130] Better storage lens case designs, frequent replacement of the lens case (at least once in three to six months) and improved hygiene of lens cases may decrease the incidence of corneal ulceration.[51]

Timely diagnosis and treatment are of paramount importance as early treatment can limit the scarring and vision loss caused by CLMK.[46,106,109,132] Treatment delayed by more than 12 hours increases the risk for vision loss.[48]

Post-marketing surveillance of drugs and devices is important to the health and safety of the general public. Because contact lenses and accompanying lens care solutions are regulated as medical devices by the Food and Drug Administration (FDA) for patient safety, cases of contact lens-related infections should be reported as adverse events to the FDA Safety Information and Adverse Event Reporting Program (www.fda.gov/safety/medwatch). Using the data accumulated in the adverse event reporting program, contact lens stakeholders—industry, regulatory authorities, eye care providers and public health professionals—can work together to determine what improvements can be made to contact lenses, care products, manufacturer guidelines, and labelling. ■

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1. McLeod SD, LaBree LD, Tayyanipour R, et al. The importance of initial management in the treatment of severe infectious corneal ulcers. Ophthalmology. 1995;102(12):1943-8.
2. McLeod SD, Kolahdouz-Isfahani A, Rostamian K, et al. The role of smears, cultures, and antibiotic sensitivity testing in the management of suspected infectious keratitis. Ophthalmology. 1996;103(1):23-8.
3. Benson WH, Lanier JD. Current diagnosis and treatment of corneal ulcers. Curr Opin Ophthalmol. 1998;9(4):45-9.
4. Musch DC, Sugar A, Meyer RF. Demographic and predisposing factors in corneal ulceration. Arch Ophthalmol. 1983;101(10):1545-8.
5. Dart JK. Predisposing factors in microbial keratitis: the significance of contact lens wear. Br J Ophthalmol. 1988;72(12):926-30.
6. Liesegang TJ. Contact lens-related microbial keratitis: Part I: Epidemiology. Cornea. 1997;16(2):125-31.
7. Schaefer F, Bruttin O, Zografos L, Guex-Crosier Y. Bacterial keratitis: a prospective clinical and microbiological study. Br J Ophthalmol. 2001;85(7):842-7.
8. Miedziak AI, Miller MR, Rapuano CJ, et al. Risk factors in microbial keratitis leading to penetrating keratoplasty. Ophthalmology. 1999;106(6):1166-70; discussion 1171.
9. Vajpayee RB, Dada T, Saxena R, et al. Study of the first contact management profile of cases of infectious keratitis: a hospital-based study. Cornea. 2000;19(1):52-6.
10. Bourcier T, Thomas F, Borderie V, et al. Bacterial keratitis: predisposing factors, clinical and microbiological review of 300 cases. Br J Ophthalmol. 2003;87(7):834-8.
11. Stapleton F, Keay L, Edwards K, et al. The incidence of contact lens-related microbial keratitis in Australia. Ophthalmology. 2008;115(10):1655-62.
12. Poggio EC, Glynn RJ, Schein OD, et al. The incidence of ulcerative keratitis among users of daily-wear and extended-wear soft contact lenses. N Engl J Med. 1989;321(12):779-83.
13. Lam DS, Houang E, Fan DS, et al. Incidence and risk factors for microbial keratitis in Hong Kong: comparison with Europe and North America. Eye (Lond). 2002;16(5):608-18.
14. Cheng KH, Leung SL, Hoekman HW, et al. Incidence of contact-lens-associated microbial keratitis and its related morbidity. Lancet. 1999;354(9174):181-5.
15. Seal DV, Kirkness CM, Bennett HG, et al. Population-based cohort study of microbial keratitis in Scotland: incidence and features. Cont Lens Anterior Eye. 1999;22(2):49-57.
16. Papas EB, Vajdic CM, Austen R, Holden BA. High-oxygen-transmissibility soft contact lenses do not induce limbal hyperaemia. Curr Eye Res. 1997;16(9):942-8.
17. Chalmers RL, Hickson-Curran SB, Keay L, et al. Rates of adverse events with hydrogel and silicone hydrogel daily disposable lenses in a large postmarket surveillance registry: the TEMPO Registry. Invest Ophthalmol Vis Sci. 2015;56(1):654-63.
18. Radford CF, Minassian D, Dart JK, et al. Risk factors for nonulcerative contact lens complications in an ophthalmic accident and emergency department: a case-control study. Ophthalmology. 2009;116(3):385-92.
19. Steele KR, Szczotka-Flynn L. Epidemiology of contact lens-induced infiltrates: an updated review. Clin Exp Optom. 2017;100(5):473-481.
20. Green M, Apel A, Stapleton F. Risk factors and causative organisms in microbial keratitis. Cornea. 2008;27(1):22-7.
21. Stapleton F, Keay LJ, Sanfilippo PG, et al. Relationship between climate, disease severity, and causative organism for contact lens-associated microbial keratitis in Australia. Am J Ophthalmol. 2007;144(5):690-98.
22. Allan BD, Dart JK. Strategies for the management of microbial keratitis. r J Ophthalmol. 1995;79(8):777-86.
23. Schein OD, Ormerod LD, Barraquer E, et al. Microbiology of contact lens-related keratitis. Cornea. 1989;8(4):281-5.
24. Carnt N, Stapleton F. Strategies for the prevention of contact lens-related Acanthamoeba keratitis: a review. Ophthalmic Physiol Opt. 2016;36(2):77-92.
25. Radford CF, Minassian DC, Dart JK. Acanthamoeba keratitis in England and Wales: incidence, outcome, and risk factors. Br J Ophthalmol. 2002;86(5):536-42.
26. Fraser MN, Wong Q, Shah L, et al. Characteristics of an Acanthamoeba keratitis outbreak in British Columbia between 2003 and 2007. Ophthalmology. 2012 Jun;119(6):1120-5.
27. Por YM, Mehta JS, Chua JL, et al., Acanthamoeba keratitis associated with contact lens wear in Singapore. Am J Ophthalmol. 2009;148(1):7-12.
28. Kilvington S, Gray T, Dart J, et al. Acanthamoeba keratitis: the role of domestic tap water contamination in the United Kingdom. Invest Ophthalmol Vis Sci. 2004;45(1):165-9.
29. Butler TK, Males JJ, Robinson LP, et al. Six-year review of Acanthamoeba keratitis in New South Wales, Australia: 1997-2002. Clin Exp Ophthalmol. 2005;33(1):41-6.
30. Ji WT, Hsu BM, Chang TY, et al. Surveillance and evaluation of the

infection risk of free-living amoebae and Legionella in different aquatic environments. Sci Total Environ. 2014;499:212-9.
31. Joslin CE, Tu EY, McMahon TT, et al. Epidemiological characteristics of a Chicago-area Acanthamoeba keratitis outbreak. Am J Ophthalmol. 2006;142(2):212-7.
32. Nelson PE, Dignani MC, Anaissie EJ. Taxonomy, biology, and clinical aspects of Fusarium species. Clin Microbiol Rev. 1994;7(4):479-504.
33. Saw SM, Ooi PL, Tan DT, et al. Risk factors for contact lens-related fusarium keratitis: a case-control study in Singapore. Arch Ophthalmol. 2007;125(5):611-7.
34. Chang DC, Grant GB, O'Donnell K, et al. Multistate outbreak of Fusarium keratitis associated with use of a contact lens solution. JAMA. 2006;296(8):953-63.
35. Gorscak JJ, Ayres BD, Bhagat N, et al. An outbreak of Fusarium keratitis associated with contact lens use in the northeastern United States. Cornea. 2007;26(10):1187-94.
36. Gower EW, Keay LJ, Oechsler RA, et al. Trends in fungal keratitis in the United States, 2001 to 2007. Ophthalmology. 2010;117(12):2263-7.
37. Sweeney DF, Jalbert I, Covey M, et al. Clinical characterization of corneal infiltrative events observed with soft contact lens wear. Cornea. 2003;22(5):435-42.
38. Stapleton F, Keay L, Jalbert I, Cole N. The epidemiology of contact lens related infiltrates. Optom Vis Sci. 2007;84(4):257-72.
39. Cutter GR, Chalmers RL, Roseman M. The clinical presentation, prevalence, and risk factors of focal corneal infiltrates in soft contact lens wearers. CLAO J. 1996;22(1):30-7.
40. Richdale K, Lam DY, Wagner H, et al. Case-control pilot study of soft contact lens wearers with corneal infiltrative events and healthy controls. Invest Ophthalmol Vis Sci. 2016;57(1):47-55.
41. Chalmers RL, Wagner H, Mitchell GL, et al. Age and other risk factors for corneal infiltrative and inflammatory events in young soft contact lens wearers from the Contact Lens Assessment in Youth (CLAY) study. Invest Ophthalmol Vis Sci. 2011;52(9):6690-6.
42. Morgan PB, Efron N, Hill EA, et al. Incidence of keratitis of varying severity among contact lens wearers. Br J Ophthalmol. 2005;89(4):430-6.
43. Wu YT, Willcox MD, Stapleton F. The effect of contact lens hygiene behavior on lens case contamination. Optom Vis Sci. 2015;92(2):167-74.
44. Fleiszig SM, Evans DJ. The pathogenesis of contact lens-associated microbial keratitis. Optom Vis Sci. 2010;87(4):225-32.
45. Fleiszig SM. The pathogenesis of contact lens-related keratitis. Optom Vis Sci. 2006;83(12):866-73.
46. Cornea/External Disease Preferred Practice Pattern Panel. Bacterial Keratitis Preferred Practice Pattern. American Academy of Ophthalmology. 2018. www.aaojournal.org/article/S0161-6420(18)32644-7/pdf.
47. Willcox MD. Which is more important to the initiation of contact lens related microbial keratitis, trauma to the ocular surface or bacterial pathogenic factors? Clin Exp Optom. 2006;89(5):277-9.
48. Musa F, Tailor R, Gao A, et al. Contact lens-related microbial keratitis in deployed British military personnel. Br J Ophthalmol. 2010;94(8):988-93.
49. Giraldez MJ, Resua CG, Lira M, et al. Contact lens hydrophobicity and roughness effects on bacterial adhesion. Optom Vis Sci. 2010;87(6):E426-31.
50. Ahn M, Yoon KC, Ryu SK, et al. Clinical aspects and prognosis of mixed microbial (bacterial and fungal) keratitis. Cornea. 2011;30(4):409-13.
51. Wu YT, Zhu H, Harmis NY, et al. Profile and frequency of microbial contamination of contact lens cases. Optom Vis Sci. 2010;87(3):E152-8.
52. Zantos S. Corneal infiltrates, debris and microcysts. J Am Optom Assoc. 1984;55:196-8.
53. Dart JK, Radford CF, Minassian D, et al. Risk factors for microbial keratitis with contemporary contact lenses: a case-control study. Ophthalmology. 2008;115(10):1647-54.
54. Nilsson SE, Montan PG. The annualized incidence of contact lens induced keratitis in Sweden and its relation to lens type and wear schedule: results of a 3-month prospective study. CLAO J. 1994;20(4):225-30.
55. Stapleton F, Dart JK, Minassian D. Risk factors with contact lens related suppurative keratitis. CLAO J. 1993;19(4):204-10.
56. Dart JK, Stapleton F, Minassian D. Contact lenses and other risk factors in microbial keratitis. Lancet. 1991;338(8768):650-3.
57. Ladage PM, Ren DH, Petroll WM, et al. Effects of eyelid closure and disposable and silicone hydrogel extended contact lens wear on rabbit corneal epithelial proliferation. Invest Ophthalmol Vis Sci. 2003;44(5):1843-9.
58. Ladage PM, Jester JV, Petroll WM, et al. Vertical movement of epithelial basal cells toward the corneal surface during use of extended-wear contact lenses. Invest Ophthalmol Vis Sci. 2003;44(3):1056-63.
59. O'Leary DJ, Madgewick R, Wallace J, Ang J. Size and number of epithelial cells washed from the cornea after contact lens wear. Optom Vis Sci. 1998;75(9):692-6.
60. Ren DH, Petroll WM, Jester JV, et al. The relationship between contact lens oxygen permeability and binding of Pseudomonas aeruginosa to human corneal epithelial cells after overnight and extended wear. CLAO J. 1999;25(2):80-100.
61. Ladage PM, Yamamoto K, Ren DH, et al. Effects of rigid and soft contact lens daily wear on corneal epithelium, tear lactate dehydrogenase, and bacterial binding to exfoliated epithelial cells. Ophthalmology. 2001;108(7):1279-88.
62. Paugh JR, Stapleton F, Keay L, Ho A. Tear exchange under hydrogel contact lenses: methodological considerations. Invest Ophthalmol Vis Sci. 2001;42(12):2813-20.
63. Larkin DF, Leeming JP. Quantitative alterations of the commensal eye bacteria in contact lens wear. Eye (Lond). 1991;5(Pt 1):70-4.
64. Høvding G. The conjunctival and contact lens bacterial flora during lens wear. Acta Ophthalmol. 1981;59(3):387-401.
65. Edwards K, Keay L, Naduvilath T, et al. Characteristics of and risk factors for contact lens-related microbial keratitis in a tertiary referral hospital. Eye (Lond). 2009;23(1):153-60.
66. Jones DB, Liesegang TJ, Robinson NM. Laboratory diagnosis of ocular infections. Am Society for Microbiology, Washington, DC; 1981.
67. Willcox MD, Harmis N, Cowell BA, et al. Bacterial interactions with contact lenses; effects of lens material, lens wear and microbial physiology. Biomaterials. 2001;22(24):3235-47.
68. Szczotka-Flynn L, Lass JH, Sethi A, et al. Risk factors for corneal infiltrative events during continuous wear of silicone hydrogel contact lenses. Invest Ophthalmol Vis Sci. 2010;51(11):5421-30.
69. McLaughlin-Borlace L, Stapleton F, Matheson M, Dart JK. Bacterial biofilm on contact lenses and lens storage cases in wearers with microbial keratitis. J Appl Microbiol. 1998;84(5):827-38.
70. Kodjikian L, Casoli-Bergeron E, Malet F, et al. Bacterial adhesion to conventional hydrogel and new silicone-hydrogel contact lens materials. Graefes Arch Clin Exp Ophthalmol. 2008;246(2):267-73.
71. Szczotka-Flynn LB, Pearlman E, Ghannoum M. Microbial contamination of contact lenses, lens care solutions, and their accessories: a literature review. Eye Contact Lens. 2010;36(2):116-29.
72. Jiang Y, Jacobs M, Bajaksouzian S, et al. Risk factors for microbial bioburden during daily wear of silicone hydrogel contact lenses. Eye Contact Lens. 2014;40(3):148-56.
73. Wu YT, Willcox M, Zhu H, Stapleton F. Contact lens hygiene compliance and lens case contamination: A review. Cont Lens Anterior Eye. 2015;38(5):307-16.
74. Radford CF, Minassian DC, Dart JK. Acanthamoeba keratitis in England and Wales: incidence, outcome, and risk factors. Br J Ophthalmol. 2002;86(5):536-42.
75. Dart JK, Stapleton F, Minassian D. Contact lenses and other risk factors in microbial keratitis. Lancet. 1991;338(8768):650-3.
76. Radford CF, Minassian DC, Dart JK. Disposable contact lens use as a risk factor for microbial keratitis. Br J Ophthalmol. 1998;82(11):1272-5.
77. Stapleton F, Edwards K, Keay L, et al. Risk factors for moderate and severe microbial keratitis in daily wear contact lens users. Ophthalmology. 2012;119(8):1516-21.
78. Morgan PB, Efron N, Brennan NA, et al. Risk factors for the development of corneal infiltrative events associated with contact lens wear. Invest Ophthalmol Vis Sci. 2005;46(9):3136-43.
79. Keay L, Stapleton F. Development and evaluation of evidence-based guidelines on contact lens-related microbial keratitis. Cont Lens Anterior Eye. 2008;31(1):3-12.
80. Cope JR, Collier SA, Nethercut H, et al. Risk behaviors for contact lens-related eye infections among adults and adolescents—United States, 2016. MMWR Morb Mortal Wkly Rep. 2017;66(32):841-45.
81. Radford CF, Lehmann OJ, Dart JK. Acanthamoeba keratitis: multicentre survey in England 1992-6. Br J Ophthalmol. 1998;82(12):1387-92.
82. Seal DV, Kirkness CM, Bennett HG, Peterson M. Acanthamoeba keratitis in Scotland: risk factors for contact lens wearers. Cont Lens Anterior Eye. 1999;22(2):58-68.
83. Schein OD, Glynn RJ, Poggio EC, et al. The relative risk of ulcerative keratitis among users of daily-wear and extended-wear soft contact lenses. A case-control study. N Engl J Med. 1989;321(12):773-8.
84. Keay L, Edwards K, Stapleton F. Signs, symptoms and comorbidities in contact lens-related microbial keratitis. Optom Vis Sci. 2009;86(7):803-9.
85. Phillips SP. Defining and measuring gender: a social determinant of health whose time has come. Int J Equity Health. 2005;4:11.
86. Brett KM, Burt CW. Utilization of ambulatory medical care by women; United States, 1997-1998. Vital Health Stat 13. 2001;(149):1-46.
87. Keijser S, Kurreeman FA, de Keizer RJ, et al. IL-10 promotor haplotypes associated with susceptibility to and severity of bacterial corneal ulcers. Exp Eye Res. 2009;88(6):1124-8.
88. Carnt NA, Willcox MD, Hau S, et al. Association of single nucleotide polymorphisms of interleukins-1 , -6, and -12B with contact lens keratitis susceptibility and severity. Ophthalmology. 2012;119(7):1320-7.
89. Carnt N, Robaei D, Watson SL, et al. The impact of topical corticosteroids used in conjunction with antiamoebic therapy on the outcome of acanthamoeba keratitis. Ophthalmology. 2016;123(5):984-90.
90. Carnt N, Keay L, Willcox M, et al. Higher risk taking propensity of contact lens wearers is associated with less compliance. Cont Lens Anterior Eye. 2011;34(5):202-6.
91. Carnt N, Samarawickrama C, White A, Stapleton F. The diagnosis and management of contact lens-related microbial keratitis. Clin Exp Optom. 2017;100(5):482-93.
92. Willcox MD. Pseudomonas aeruginosa infection and inflammation during contact lens wear: a review. Optom Vis Sci. 2007;84(4):273-8.
93. Oka N, Suzuki T, Ishikawa E, et al. Relationship of virulence factors and clinical features in keratitis caused by Pseudomonas aeruginosa. Invest Ophthalmol Vis Sci. 2015;56(11):6892-8.
94. Dart JK, Saw VP, Kilvington S. Acanthamoeba keratitis: diagnosis and treatment update 2009. Am J Ophthalmol. 2009;148(4):487-499.e2.
95. Robbie SJ, Vega FA, Tint NL, et al. Perineural infiltrates in Pseudomonas keratitis. J Cataract Refract Surg. 2013;39(11):1764-7.
96. Claerhout I, Goegebuer A, Van Den Broecke C, Kestelyn P. Delay in diagnosis and outcome of Acanthamoeba keratitis. Graefes Arch Clin Exp Ophthalmol. 2004;242(8):648-53.
97. Tu EY, Joslin CE, Sugar J, et al. Prognostic factors affecting visual outcome in Acanthamoeba keratitis. Ophthalmology. 2008;115(11):1998-2003.
98. Abad JC, Foster CS. Fungal keratitis. Int Ophthalmol Clin. 1996;36(3):1-15.
99. Thomas PA. Current perspectives on ophthalmic mycoses. Clin Microbiol Rev. 2003;16(4):730-97.
100. Loh RS, Chan CM, Ti SE, et al. Emerging prevalence of microsporidial keratitis in Singapore: epidemiology, clinical features, and management. Ophthalmology. 2009;116(12):2348-53.
101. Robaei D, Carnt N, Minassian DC, Dart JK. The impact of topical corticosteroid use before diagnosis on the outcome of Acanthamoeba keratitis. Ophthalmology. 2014;121(7):1383-8.
102. Chang HY, Chodosh J. Diagnostic and therapeutic considerations in fungal keratitis. Int Ophthalmol Clin. 2011;51(4):33-42.
103. Thomas PA, Leck AK, Myatt M. Characteristic clinical features as an aid to the diagnosis of suppurative keratitis caused by filamentous fungi. Br J Ophthalmol. 2005;89(12):1554-8.
104. Kaufman HE. Adenovirus advances: new diagnostic and therapeutic options. Curr Opin Ophthalmol. 2011;22(4):290-3.
105. Aasuri MK, Venkata N, Kumar VM. Differential diagnosis of microbial keratitis and contact lens-induced peripheral ulcer. Eye Contact Lens. 2003;29(1 Suppl):S60-2; discussion S83-4, S192-4.
106. Garg P. Diagnosis of microbial keratitis. Br J Ophthalmol. 2010;94(8):961-2.
107. Kaiser PK, Friedman NJ, Pineda R, et al. The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology. 4th ed. London: Saunders; 2014.
108. Ehlers JP, Shah CP. The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. Philadelphia: Lippincott Williams & Wilkins; 2008.
109. Shah VM, Tandon R, Satpathy G, et al. Randomized clinical study for comparative evaluation of fourth-generation fluoroquinolones with the combination of fortified antibiotics in the treatment of bacterial corneal ulcers. Cornea. 2010;29(7):751-7.
110. Chawla B, Agarwal P, Tandon R, et al. In vitro susceptibility of bacterial keratitis isolates to fourth-generation fluoroquinolones. Eur J Ophthalmol. 2010;20(2):300-5.
111. Miller D, Flynn PM, Scott IU, et al. In vitro fluoroquinolone resistance in staphylococcal endophthalmitis isolates. Arch Ophthalmol. 2006;124(4):479-83.
112. Adebayo A, Parikh JG, McCormick SA, et al. Shifting trends in in vitro antibiotic susceptibilities for common bacterial conjunctival isolates in the last decade at the New York Eye and Ear Infirmary. Graefes Arch Clin Exp Ophthalmol. 2011;249(1):111-9.
113. Asbell PA, Sanfilippo CM, Pillar CM, et al. Antibiotic resistance among ocular pathogens in the united states: five-year results from the Antibiotic Resistance Monitoring in Ocular Microorganisms (ARMOR) Surveillance study. JAMA Ophthalmol. 2015;133(12):1445-54.
114. Mah FS, Sanfilippo CM. Besifloxacin: Efficacy and safety in treatment and prevention of ocular bacterial infections. Ophthalmol Ther. 2016;5(1):1-20.
115. Katz HR, Morris S, Woolridge R. At Issue: Cycloplegics, fluoroquinolones primary treatment for bacterial keratitis. Primary Care Optometry News. 2001;6(4).
116. Constantinou M, Daniell M, Snibson GR, et al. Clinical efficacy of moxifloxacin in the treatment of bacterial keratitis: a randomized clinical trial. Ophthalmology. 2007;114(9):1622-9.
117. Elder MJ, Kilvington S, Dart JK. A clinicopathologic study of in vitro sensitivity testing and Acanthamoeba keratitis. Invest Ophthalmol Vis Sci. 1994;35(3):1059-64.
118. Dart JK, Saw VP, Kilvington S. Acanthamoeba keratitis: diagnosis and treatment update 2009. Am J Ophthalmol. 2009;148(4):487-499.e2.
119. Ng JK, Fraunfelder FW, Winthrop KL. Review and update on the epidemiology, clinical presentation, diagnosis, and treatment of fungal keratitis. Curr Fungal Infect Rep. 2013;7(4):293-300.
120. Prajna NV, Krishnan T, Mascarenhas J, et al. The mycotic ulcer treatment trial: a randomized trial comparing natamycin vs voriconazole. JAMA Ophthalmol. 2013;131(4):422-9.
121. Prajna NV, Krishnan T, Rajaraman R, et al; Mycotic Ulcer Treatment Trial II Group. Effect of oral voriconazole on fungal keratitis in the mycotic ulcer treatment trial II (MUTT II): a randomized clinical trial. JAMA Ophthalmol. 2016;134(12):1365-72.
122. Zrenner E, Tomaszewski K, Hamlin J, et al. Effects of multiple doses of voriconazole on the vision of healthy volunteers: a double-blind, placebo-controlled study. Ophthalmic Res. 2014;52(1):43-52.
123. Rahman MR, Minassian DC, Srinivasan M, et al. Trial of chlorhexidine gluconate for fungal corneal ulcers. Ophthalmic Epidemiol. 1997;4(3):141-9.
124. Stergiopoulou T, Meletiadis J, Sein T, et al. Isobolographic analysis of pharmacodynamic interactions between antifungal agents and ciprofloxacin against Candida albicans and Aspergillus fumigatus. Antimicrob Agents Chemother. 2008;52(6):2196-204.
125. Stern GA, Buttross M. Use of corticosteroids in combination with antimicrobial drugs in the treatment of infectious corneal disease. Ophthalmology. 1991;98(6):847-53.
126. Cardenas ME, Cruz MC, Del Poeta M, et al. Antifungal activities of

antineoplastic agents: Saccharomyces cerevisiae as a model system to study drug action. Clin Microbiol Rev. 1999;12(4):583-611.
127. Perry HD, Doshi SJ, Donnenfeld ED, Bai GS. Topical cyclosporin A in the management of therapeutic keratoplasty for mycotic keratitis. Cornea. 2002;21(2):161-3.
128. Ansari Z, Miller D, Galor A. Current thoughts in fungal keratitis: diagnosis and treatment. Curr Fungal Infect Rep. 2013;7(3):209-218.
129. Robaei D, Carnt N, Minassian DC, Dart JK. Therapeutic and optical keratoplasty in the management of Acanthamoeba keratitis: risk factors, outcomes, and summary of the literature. Ophthalmology. 2015;122(1):17-24.
130. Yeung KK, Forister JF, Forister EF, et al. Compliance with soft contact lens replacement schedules and associated contact lens-related ocular complications: The UCLA Contact Lens Study. Optometry. 2010;81(11):598-607.
131. Chalmers RL, Keay L, Long B, et al. Risk factors for contact lens complications in US clinical practices. Optom Vis Sci. 2010;87(10):725-35.
132. Huerva V, Sanchez MC. Refractive outcome after severe Pseudomonas aeruginosa keratitis. Optom Vis Sci. 2011;88(4):E548-52.
133. Arshad M, Carnt N, Tan J, et al. Water exposure and the risk of contact lens-related disease. Cornea. 2019;38(6):791-797.

OSC QUIZ

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1. The incidence of CLMK in daily soft contact lens wearers is:
a. 2 to 4 per 1,000.
b. 2 to 4 per 10,000.
c. 20 to 40 per 10,000.
d. 20 to 40 per 100,000.

2. Which of these lenses have the lowest incidence of MK?
a. Daily disposables.
b. Silicone hydrogels.
c. Gas permeable lenses.
d. Cosmetic contact lenses.

3. Which of these is a risk factor for MK?
a. Washing lens cases with contact lens solutions.
b. Use of non-sterile tap water to store contact lenses.
c. Swimming while wearing contact lenses.
d. Both b and c.

4. Which MK type is more rare than others?
a. Bacterial.
b. Fungal.
c. *Acanthamoeba*.
d. Both b and c.

5. A 2006-2007 outbreak of contact lens-related *Fusarium* keratitis was primarily associated with:
a. Water exposure.
b. Disinfecting solution.
c. Non-compliance.
d. Temperate climatic conditions.

6. Contact lens disease-causing pathogens are:
a. Airborne.
b. Waterborne.
c. Both a and b.
d. None of the above.

7. Corneal ulceration begins with:
a. Loss of corneal transparency.
b. Slower epithelial cell proliferation and migration causing inflammatory response.
c. Pathogenic level of bacteria gaining access to the corneal stroma.
d. Penetration of corneal epithelium more severe than punctate staining.

8. All of the following are population-attributable risks for CLMK, except:
a. Smoking.
b. Poor hand hygiene.
c. Omission or infrequent lens disinfection.
d. Diabetes.

9. Which of these pathogens is highly difficult to neutralize?
a. Propionibacterium.
b. *Acanthamoeba*.
c. *Pseudomonas*.
d. Herpes simplex virus.

10. Which microbe produces protease and elastase that digests collagen, contributing to corneal melting and perforation?
a. Propionibacterium.
b. *Acanthamoeba*.
c. *Pseudomonas*.
d. Herpes simplex virus.

11. Which of these is a hallmark sign of *Pseudomonas aeruginosa* keratitis?
a. Ring abscess.
b. Epitheliopathy with dendritic appearance.
c. Perineural infiltrates.
d. Pseudohyphae.

12. Which of the following is a hallmark sign of *Acanthamoeba* keratitis?
a. Hypopyon.
b. Ring abscess.
c. Perineural infiltrates.
d. Pseudohyphae.

13. Which is a hallmark of fungal keratitis?
a. Epitheliopathy, no dendritic appearance.
b. Hyphae.
c. Perineural infiltrates.
d. Ring abscess.

14. About 50% of *Acanthamoeba* keratitis cases are misdiagnosed as keratitis due to:
a. Propionibacterium.
b. *Streptococcus*.
c. *Pseudomonas*.
d. Herpes simplex virus.

15. All of the following are conventional diagnostic techniques to identify causative organisms in MK, except:
a. Corneal biopsy.
b. Histopathological analysis.
c. Filter paper technique.
d. Drug sensitivity testing.

16. All of the following are gold standard treatments for corneal ulceration, except:
a. Cefazoline.
b. Ciprofloxacin.
c. Tobramycin.
d. Amphotericin.

17. Which of these is a concern for using topical corticosteroids in bacterial keratitis?
a. Re-epithelialization.
b. Increased risk of perforation.
c. Recurrent infection.
d. All of the above.

18. Which reason for contact lens complications is highest among teenagers?
a. Smoking.
b. Longer periods of lens wear.
c. Internet purchase of lenses.
d. Non-compliance with manufacturers' recommended frequency of lens replacement.

19. Active dissemination of clear unequivocal guidelines to lens wearers should be through:
a. Contact lens manufactures.
b. Contact lens practitioners.
c. Professional organizations.
d. All of the above.

20. Contact lenses and accompanying lens care solutions are regulated by the FDA as:
a. Medical devices.
b. Drugs.
c. Cosmetics.
d. Biologics.

Examination Answer Sheet

The Dangers and The Diagnosis of CLMK

Valid for credit through August 15, 2022

Online: This exam can be taken online at www.reviewsce.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.

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Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ
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19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

Rate how well the activity supported your achievement of these learning objectives:
1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent

21. Discuss the incidence and types of MK, including bacterial, fungal and *Acanthamoeba* infections. ① ② ③ ④ ⑤

22. Identify the many modifiable and non-modifiable risk factors associated with contact lens-related microbial keratitis. ① ② ③ ④ ⑤

23. Provide prompt and correct diagnosis of MK, followed by effective pharmaceutical therapy or corneal procedure. ① ② ③ ④ ⑤

24. Educate patients about proper contact lens wear, lens replacement, and cleaning and disinfecting lenses and cases. ① ② ③ ④ ⑤

25. Based upon your participation in this activity, do you intend to change your practice behavior? (choose only one of the following options)
Ⓐ I do plan to implement changes in my practice based on the information presented.
Ⓑ My current practice has been reinforced by the information presented.
Ⓒ I need more information before I will change my practice.

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)

ⓐ Apply latest guidelines ⓑ Change in pharmaceutical therapy ⓒ Choice of treatment/management approach ⓓ Change in current practice for referral ⓔ Change in non-pharmaceutical therapy ⓕ Change in differential diagnosis ⓖ Change in diagnostic testing ⓗ Other, please specify: _____

28. How confident are you that you will be able to make your intended changes?

ⓐ Very confident ⓑ Somewhat confident ⓒ Unsure ⓓ Not confident

29. Which of the following do you anticipate will be the primary barrier to implementing these changes?
ⓐ Formulary restrictions
ⓑ Time constraints
ⓒ System constraints
ⓓ Insurance/financial issues
ⓔ Lack of interprofessional team support
ⓕ Treatment related adverse events
ⓖ Patient adherence/compliance
ⓗ Other, please specify:

30. Additional comments on this course:

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

First Name
Last Name
E-Mail

The following is your: ☐ Home Address ☐ Business Address

Business Name
Address
City State
ZIP
Telephone # - -
Fax # - -

Rate the quality of the material provided:
1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

31. The content was evidence-based. ① ② ③ ④ ⑤

32. The content was balanced and free of bias. ① ② ③ ④ ⑤

33. The presentation was clear and effective. ① ② ③ ④ ⑤

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

Signature _____ Date _____

Lesson 118583 RO-OSC-0819

Disinfect the Natural Way

New hypochlorous acid options are making this a viable antimicrobial solution. Here's how to choose—and use—it correctly. **By Paul M. Karpecki, OD**

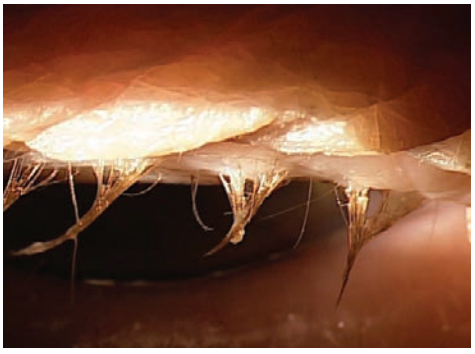
Hypochlorous acid (HOCl) has a multitude of uses in wound care, dermatology, dentistry and eye care. It is the most common disinfectant in medical, industrial and domestic use, and has the same active ingredient of household bleach but with a different chemical structure.[1] Bleach, or sodium hydroxide, is typically found in concentrations that range from 1% to 5%, which would result in chemical burns to the eye upon contact. HOCl, however, is found in much lower concentrations and has no such risks.

HOCl is an appealing disinfectant because it is an all-natural antimicrobial agent. Pure HOCl is produced as an element of the human immune response.[2,3] During the oxidative burst, small, highly reactive molecules such as HOCl are generated as white blood cells respond to pathogens in the body.[2,4] HOCl is released by neutrophils to kill microorganisms and neutralize toxins released from pathogens and inflammatory mediators.[5]

Because it is neutralized quickly, HA is nontoxic to the ocular surface.[5]

Gentle But Effective

Although HOCl is natural, it's surprisingly potent. It has broad-spectrum antimicrobial activity and can kill microorganisms rapidly (*Table 1*).[6] HOCl is highly effective *in vitro* against a wide range of microorganisms, helping to fight infection, reduce inflammation, control the body's response to injury and enhance its natural ability to heal.



Hypochlorous acid is a natural, gentle way to eradicate bacteria on and around the eyelids.

In eye care, HOCl can provide effective relief from dry eyes and hordeola. It's a treatment option for red, itchy eyelids associated with conditions such as blepharitis and meibomian gland dysfunction (MGD). Lid hygiene products containing HOCl are an excellent addition to a patient's daily wellness routine to decrease the microbial load on the lids and lashes.[7]

Surfactant cleaners are often a necessary long-term therapy, but HOCl is also a safe daily use option, as it is unlikely to cause skin irritation associated with other cleaners and scrubs containing preservatives or other additives. HOCl is also an excellent choice following in-office blepharoexfoliation because it can prolong wellness after bacterial load de-bulking.

For patients with MGD, HOCl is well paired with warm moist compress therapy to provide the dual effect of reducing the bacterial burden and promoting secretions.

We can also use HOCl as preoperative antisepsis, to clean instruments such as tonometry tips, and following a short course of a topical antibiotic-corticosteroid to keep the bacterial levels at bay.[8,9]

Protect the Microbiome, Prevent Resistance

Our bodies are home to a diverse microbiome, and eyes are no exception. Some bacteria, fungi and viruses may protect against harmful pathogens, and full eradication is rarely beneficial.[10] Thus, we often aim to simply reduce the overall bacterial load.

Studies show that blepharitis patients, for example, harbor a bacterial load more than 14 times greater than controls, and a HOCl solution may help reduce the number without strengthening harmful strains.[2,11]

Researchers also discovered that HOCl decreased the bacterial load by more than 90% without significantly altering the diversity of the bacterial species.[2] In addition, products containing HOCl generally are not antibiotics and do not contribute to the ever-growing issue of antibiotic resistance.[7,12,13]

Pick the Right Product

Four factors are important when differentiating HOCl solutions: purity, preservation, pH and prescription.

Purity. Many HOCl products contain ingredients, such as bleach byproducts and other chemicals, you may not want patients encountering on a daily basis. For long-term use, look for solution free of additives, fragrance, chemicals or other byproducts. All HOCl formulations are safe

Surfactants and HOCl: A One-Two Punch

While HOCl may be beneficial in severe conditions, many patients still need to use surfactant cleaners. The root cause of anterior blepharitis is the overproduction of oils, which harbor bacteria that cause eyelid inflammation. The key to reducing this bacteria flora is to first remove the excessive oils from the eyelids and then follow up with antimicrobials. Mild surfactants in several eyelid cleansers act to dissolve and remove oil, debris and desquamated skin. HOCl formulas do not contain these surfactants and are largely ineffective in debriding the oil, scales and debris often associated with eyelid irritation. Accordingly, in the most severe cases where HOCl might be most beneficial, clinicians should recommend a combination therapy including both a surfactant cleanser and HOCl to achieve the best outcomes for the patient.

regardless of the purity level. Without the sodium hypochlorite (NaOCl) balance, the equilibrium shifts and the pure products are unstable.

Preservation. The stability of HOCl solutions is generally quite limited. Although HOCl may have a shelf life of years, once a bottle is opened, the product begins to degrade. If your patient uses the solution only occasionally, a brand with a longer shelf life may be important to maximize efficacy and avoid waste.

pH. The properties of HOCl in solution depend strongly on the solution's pH.[14] For patients with pruritis, for example, HOCl can either decrease or promote the condition, depending on the product's pH.[14] Research shows that a pH of 3.5 to 5 is necessary to maintain a stable HOCl solution, maximize its antimicrobial activities and minimize undesirable degrading products.[6]

When pH is less than 3.5, the solution exists as a mixture of chlorine.[6] When pH is higher than 5.5, NaOCl starts to form and becomes the predominant species in the alkaline pH.[6]

Prescription. While in the past optometrists needed to write a prescription for most HOCl solutions marketed for ocular use, several over-the-counter (OTC) options exist today. This is not due to an FDA decision to reclassify HOCl. Instead, the prescription-only availability of specific HOCl solutions is a manufacturer prerogative that generally allows for more specific claims about a product. OTC HOCl solutions can have the same concentration of active ingredient, but marketing statements are typically more general. Several formulations are now available and marketing in eye care, including:

- ***Avenova (NovaBay).*** This prescription spray contains pure HOCl and is designed to remove microorganisms and debris on and around the eyelid margins.
- ***HypoChlor (OcuSoft).*** This 0.02% concentration of HOCl, available without a prescription in both spray and gel formulations, is stable opened or unopened for 18 months.
- ***Bruder Hygienic Eyelid Solution.*** This 0.02% pure HOCl solution is available OTC and may be a beneficial addition to a daily eye care regimen for patients with mild or moderate conditions.
- ***Sterilid Antimicrobial (Akorn).*** This OTC spray is 0.01% HOCl with a 24-hour shelf life open or unopened.
- ***HyClear (Contamac).*** Stable for up to 18 months after opening, this product contains 0.01% HOCl and is available only through ophthalmology or optometry practices.
- ***Zenoptiq Hypochlorous Acid Solution (Focus Laboratories).*** Available without a prescription, this spray maintains stability for 18 months after opening. Ingredients include 99.94% electrolyzed oxygenated water, 0.048% sodium chloride, 0.01% HOCl and 0.002% NaOCl.

Because reducing the bacterial load is a useful tool for blepharitis, MGD and dry eye, more clinicians are turning to HOCl—particularly now that so many choices are available OTC or in doctors' offices.[2] ■

Note: Dr. Karpecki consults for companies with products and services relevant to this topic.

1. Gray MJ, Wholey WY, Jakob U. Bacterial responses to reactive chlorine species. Annu Rev Microbiol. 2013;67:141-60.
2. Stroman DW, Mintun K, Epstein AB, et al. Reduction in bacterial load using hypochlorous acid hygiene solution on ocular skin. Clin Ophthalmol. 2017;11:707-14.
3. Hurst JK. What really happens in the neutrophil phagosome? Free Radic Biol Med. 2012;53(3):508-20.
4. Kumar V, Abbas AK, Fausto N. Acute and chronic inflammation. In: Robbins and Cotran Pathologic Basis of Disease. 7th ed. Philadelphia: Elsevier; 2005:29-73.
5. Sindt C. Wash away your old hygiene strategy. Rev Cornea Contact Lens. 2014;151(4):7.
6. Wang L, Bassiri M, Najafi R, et al. Hypochlorous acid as a potential wound care agent: part I. Stabilized hypochlorous acid: a component of the inorganic armamentarium of innate immunity. J Burns Wounds. 2007;6:e5.
7. Harsch AG, Stout N, Lighthizer N. Beat the blepharitis blues. Rev Cornea Contact Lens. 2016;153(7):12-15.
8. Dang V. Sizing up anti-inflammatories in dry eye disease. Rev Optom. 2018;155(4):62-65.
9. Romanowski E, Stella N, Yates K, et al. In vitro evaluation of a hypochlorous acid hygiene solution on established biofilms. Eye Contact Lens. 2018 Nov;44(Suppl 2):S187-S191.
10. Lina G, Boutite F, Tristan A, et al. Bacterial competition for human nasal cavity colonization: role of Staphylococcal agr alleles. Appl Environ Microbiol. 2003;69(1):18-23.
11. Bezza Benkaouha I, Le Brun C, Pisella PJ, et al. Bacterial flora in blepharitis. J Fr Ophtalmol. 2015;38(8):723-28.
12. Ono T, Yamashita K, Murayama T, Sato T. Microbicidal effect of weak acid hypochlorous solution on various microorganisms. Biocontrol Science. 2012;17(3):129-33.
13. Debabov D, Noorbakhsh C, Wang L, et al. Avenova with Neutrox (pure 0.01% HOCl) compared with OTC product (0.02% HA). NovaBay Pharmaceuticals:1-5.
14. Pelgrift RY, Friedman AJ. Topical hypochlorous acid (HOCl) as a potential treatment of pruritus. Curr Derm Rep. 2013;2:181.

Table 1. Broad Spectrum Activity

HOCl solution can be bactericidal against the following pathogens:[6]

Aspergillus niger
Candida albicans
Corynebacterium amycolatum
Enterobacter aerogenes
Escherichia coli
Haemophilus influenzae
Klebsiella pneumoniae
Methicillin-resistant *staphylococcus aureus*
Micrococcus luteus
Proteus mirabilis
Pseudomonas aeruginosa
Serratia marcescens
Staphylococcus aureus
Staphylococcus epidermidis
Staphylococcus haemolyticus
Staphylococcus hominis
Staphylococcus saprophyticus
Streptococcus pyogenes
Vancomycin-resistant enterococcus faecium

That's Egg on Your Face

When a patient presents with a combination of progressive vision blur and a particular retinal appearance, all signs point to one diagnosis. **By Mark T. Dunbar, OD**

A 50-year-old male presented with slowly progressive blurred vision in his left eye, which he said he'd experienced over the past few years. He reported his right eye has been "bad" for at least 10 years and that he was diagnosed with a retinal condition when he was 27 years old. That primarily affected his right eye, but he believed the left was becoming significantly affected.

On exam, his best-corrected visual acuity was 20/400 OD eccentrically viewing and 20/60 OS. Confrontation visual fields were full-to-careful finger counting OU. His ocular motility testing was normal, and the pupils were equally round and reactive to light without an afferent pupillary defect. The anterior segment was unremarkable. His tensions measured 16mm Hg OU.

On dilated fundus exam, the vitreous was clear, optic nerves appeared heathy with small cups and good rim coloration and perfusion. Obvious retinal changes were seen in the macula of each eye (*Figure 1*). Optical coherence tomography (OCT) was also performed (*Figure 2*).

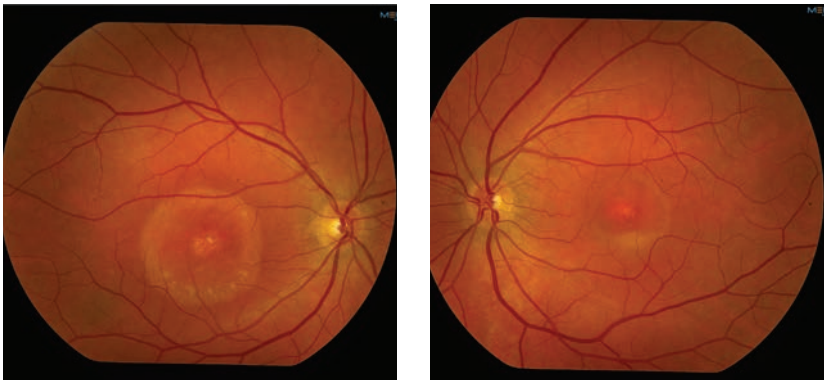


Fig 1 . Our patient's fundus photos reveal changes in both maculae.

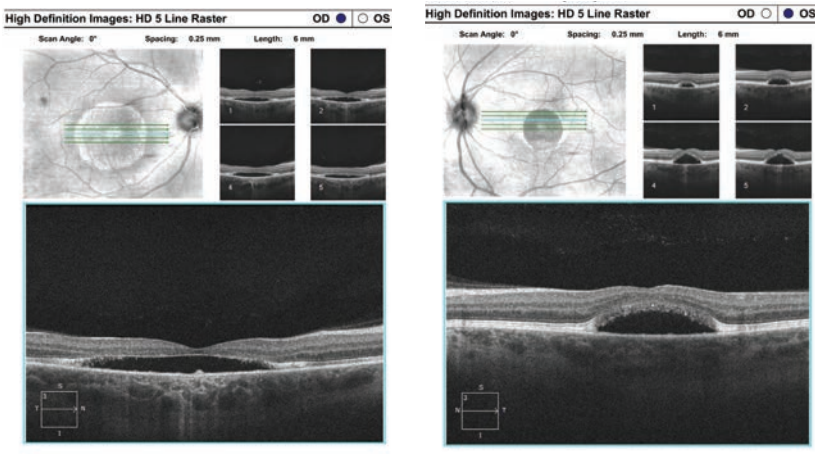


Fig. 2. OCT images of the right and left macula. What are the striking features?

Take the Retina Quiz

1. What is the most likely diagnosis?
a. Central serous chorioretinopathy.
b. Best's vitelliform macular dystrophy.
c. Adult-vitelliform macular dystrophy.
d. Cone dystrophy.

2. What are the essential findings on the OCT?
a. Neurosensory retinal detachment and loss ellipsoid zone.
b. Retinal pigment epithelial detachment.
c. Choroidal neovascularization.
d. Macular schisis.

3. What is the likelihood that any siblings he has might have the same condition?
a. Almost no chance. It's not hereditary.
b. About a one in 10 chance his siblings would have it.
c. 50% chance.
d. Greater than 90% chance.

4. What is the prognosis?
a. Stability with no effect on visual function.
b. Slow, steady progression and loss of central vision in his left eye.
c. Return to normal vision following treatment.
d. Complete blindness.

Diagnosis

The macular appearance in both eyes was quite striking, especially the right eye where central retinal pigment epithelial (RPE) depigmentation and atrophy were clearly visible. Surrounding the macula we noted a circumferential ring that had a bull's eye pattern. The left eye also had central RPE atrophic changes, but clearly not to the extent of the right eye. The patient appeared to have a neurosensory detachment that was visible on both the clinical exam and OCT. Inferior to the macula the almost hypopyon-like appearance helped make the diagnosis.

The family history also provided a clue to the diagnosis—his sister reportedly has the same condition, and his mother is a carrier. Our patient was diagnosed when he was 28 years old. So what does this all add up to? Our patient has Best's vitelliform macular dystrophy (BVMD), an autosomal dominant hereditary retinal dystrophy that affects the retinal pigment epithelium. Vision is usually not affected until childhood or early adulthood and generally has a good prognosis for maintaining good central vision in at least one eye.

The classic description for BVMD is the bilateral yellow egg-yolk appearance of the macula. At one point our patient had that appearance, but as the disease evolves the appearance can change. BVMD is classified by five stages. (*see BVMD staging*).

Our patient's right eye has progressed from Stage IV to Stage V because of the atrophic changes in the fovea, but you can still see remnants of the tale tell sign of Stage IV BVMD, which is more like a "scrambled egg" appearance around the macular than Stage II, which resembles an "egg yolk." His left eye is in stage III, as the pseudohyopyon is clearly apparent with layering of the lipofuscin material. It is likely that his left eye will experience a slow progression. About 20% of patients will develop choroidal neovascularization (CNV) following the atrophic stage which can further have an effect on central acuity.[1,2]

Research links BVMD to a genetic mutation on the BEST1 gene, which is located on chromosome 11 (11q12.3).[3] It encodes for a transmembrane protein bestophin 1 which is believed to affect the conductance of chloride which negatively affects the transport of fluid across the RPE. This in turn results in the accumulation of debris between the RPE/photoreceptor complex and Bruch's membrane.

The diagnosis can usually be made based on clinical presentation. When in doubt, an electrooculogram is diagnostic and will be positive even in the previtelliform stage when the retina appears unaffected and the vision is normal.

Best's Vitelliform Macular Dystrophy Staging

- Stage I (Previtelliform): may appear normal or have only minimal retinal changes.
- Stage II (Vitelliform): classic "egg-yolk" lesion.
- Stage III (Pseudohypopyon): layering of lipofuscin.
- Stage IV (Vitelleruptive): breakup/clumping of the material gives "scrambled" egg appearance.
- Stage V (Atrophic): Central RPE and retinal atrophy.

Monitoring

Mutimodal imaging, including fundus photography, OCT and fundus autofluoresence (FAF), may be helpful in characterizing BVMD. OCT can show classic structural changes within the retina. Even in the previtelliform stage, thickening and hyper-reflectivity of the RPE/ellipsoid zone (IS/OS junction) can be seen. In the vitelliform stage, a homogenous hyper-reflective band will be present, which represents the vitelliform material that is deposited in the outer retinal layers. This is thought to be the accumulation of the photoreceptor outer segments containing lipofuscin.[4] As the disease progresses and atrophy develops, loss of the ellipsoid junction will occur in the macula; outside the macula, the hyper-reflected band can still be present as we see in our patient. The hyper-reflective area in the right eye represents the white band that we described as a bull's eye in appearance.

It is unknown if the serous detachment seen in both eyes truly represents a detachment of the sensory retina from the RPE, or more likely a separation of the RPE from its underlying attachment to Bruch's membrane leaving a central subfoveal hypolucent space.

No genetic treatments available yet for BVMD. Our patient was able to maintain good central acuity in the left eye for many years. Five years earlier, at 45 years old, his acuity was still 20/25 and he was able to drive and was functioning without difficulty. Over the following five years, he has experienced a slow decline in acuity that affected his quality of life. We referred him to low vision services and issued him stronger reading glasses over his contact lenses. ■

1. Ryan S. Retina, 4th ed. In: Schachat AP, ed. Volume II; Medical Retinal. Philadelphia. Elsevier;2006.
2. Gass J. Stereoscopic atlas of macular diseases. Diagnosis and Treatment. 4th ed, Mosby, St. Louis;1997:304-11.
3. Stone E, Nichols B, Streb L, et al. Genetic linkage of vitelliform macular degeneration (Best's disease) to chromosome 11q13. Nat Genet. 1992;1(4):246-50.
4. Battaglia Parodi M, Iacono P, Romano F, Bandello F. Spectral domain optical coherence tomography features in different stages of Best vitelliform macular dystrophy. Retina. 2018;38(5):1041-6.

When They Go Low, ODs Go High

Bad-mouthing other doctors doesn't serve your patients. **By James L. Fanelli, OD**

A 64-year-old Caucasian female presented as a new patient after her previous eye care provider retired. She complained of long-standing blurred vision, relatively equal between the two eyes. She also reported chronic blepharitis and dry eye diagnoses, with blepharitis flare-ups on the rise. She had been given a long term prescription for neo-poly-dex drops to use when the blepharitis flared and has been using them about once a month, for approximately a week, twice a day. She had undergone LASIK surgery approximately 18 years prior. She was also taking simvastatin 80mg QD, multivitamins, calcium glucosamine and magnesium daily. She reported that her son had been diagnosed with glaucoma a few years earlier.

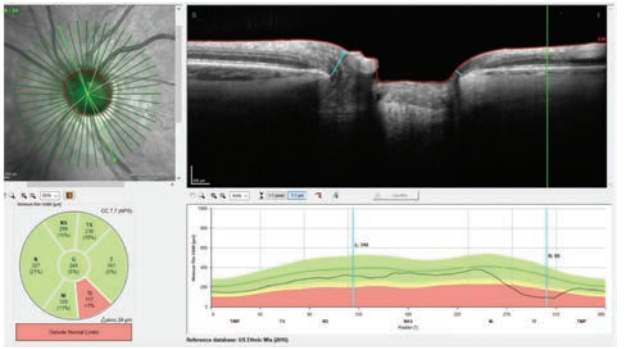


Fig. 1. Bruch's membrane opening display of the patient's left eye demonstrating erosion of the left inferotemporal neuroretinal rim. Note at the marked area the neuroretinal rim is only 95µm thick at that point.

Her last visit to her previous provider was a year earlier, at which time she was told the blurry vision was related to her dry eyes as well as, apparently, some 'changes' to her corneas following the LASIK surgery.

At the initial visit, best-corrected visual acuities (BCVA) were 20/25-2 OD and 20/30-2 OS. Confrontation fields were slightly restricted superiorly, which I initially attributed to her dermatochalasis.

Evaluation

A slit lamp examination of her anterior segments was remarkable for clear post LASIK flaps, with no evidence of striae, epithelial ingrowth or other physical aberration to both corneas. The anterior chamber angles were open and the anterior chambers were quiet. Applanation tensions were 12mm Hg OD and OS at 2:42pm. Through dilated pupils her crystalline lenses were characterized by incipient nuclear sclerosis, but not to the level to account for the 20/30-2 BCVA. Pupils were ERRLA with no afferent pupillary defect.

Stereoscopic examination of her optic nerves demonstrated eroded neuroretinal rims in both eyes, with the right showing more thinning than the left. The remaining, thinned neuroretinal rims were plush and perfused. The cup-to-disc ratios were 0.70 x 0.85 OD with an exceedingly thin neuroretinal rim from 6 o'clock to 9 o'clock, and 0.60 x 0.75 OS with an eroded rim from 3 o'clock to 6 o'clock. Peripapillary atrophy was evident in both eyes.

Her macular evaluations were essentially unremarkable. Her vascular appearance was consistent with mild arteriolarsclerotic retinopathy in both eyes, and her peripheral retinal evaluations were remarkable for 360 degrees of cystoid and scattered areas of pavingstone degeneration.

During the fundus examination, it was clear that her optic nerves were not healthy, and with the erosion of the neuroretinal rims to the extent they were, my initial impression was that she most likely had visual field loss involving fixation. Given her relatively low IOP, the differential of normal tension glaucoma arises, which has connotations of possible neuro-ophthalmic etiologies. This instigated a closer re-evaluation of the optic nerves, which, on second view, demonstrated normal size optic nerves, no evidence of edema or elevation, and in particular no evidence of pallor.

The previous LASIK is also playing a role in the low IOP readings, as contrasted to the optic nerve appearance. Pachymetry measurements were obtained, and the central corneal readings were 487µm OD and 470µm OS. When asked, she reported that she was fairly myopic prior to the LASIK,

and when pressed for an estimate of her contact lens powers, she reported that they were in the -9.00 range OU.

Diagnosis

At this point, the case seems to be pretty straightforward of undiagnosed glaucoma. Exceedingly straightforward in fact. A high myope undergoes LASIK surgery OU, with resultant thin corneas and resultant low IOP readings by applanation tonometry. The extent of her optic nerve damage indicated that the damage did not occur in the last six to 12 months. Even though the patient was using a drop containing dexamethasone off and on for an extended period of time, IOP readings at the initial visit were not indicative of a significant steroid response.

Could IOPs have been significantly high for a long enough period of time to cause the neuroretinal rim damage? While it's possible, she only used the steroid infrequently. Even though one can argue that a corrected IOP would be higher than that seen in clinic, the evidence is clear that the neuroretinal damage occurred over time. And certainly it would have been present at the time of her last visit to her previous eye care provider.

Managing the Mismanaged

We have all been in a scenario where a patient appears to have been mismanaged. While we often we give another provider the benefit of the doubt, in this case the prior provider was a militant anti-optometry crusader who, as a result, received few OD referrals over their career and, partly as a result, their surgical outcomes suffered. Do you handle this any differently than any other case where we suspect someone dropped the ball? Do we undermine the other doctor's reputation? No. Though it's tempting to react, it serves no purpose, especially to the patient. Treat this patient like any other. Explain your findings, make no excuses for the disease and explain your management plan. In this case, I asked the patient to return to clinic in a couple of weeks for threshold fields, optical coherence tomography and Heidelberg retina tomograph optic nerve imaging, as well as gonioscopy.

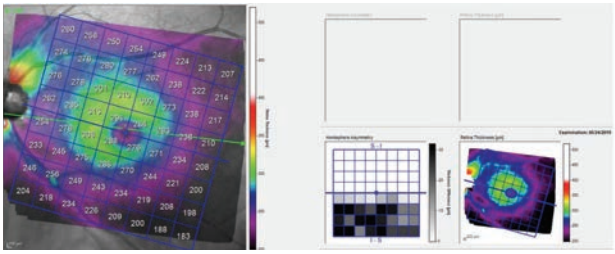


Fig. 2. Total macular thickness of the left eye, demonstrating thinning of global retinal indices inferotemporally. Note the evident difference on the asymmetry map between the superior and inferior macular hemispheres.

When the patient asks why their previous doctor didn't catch the disease, I would encourage you not to trash the other provider, as that may only serve the purpose of making the new-to-you patient feel uncomfortable with your bedside manner. For those providers whom I respect, and for whom I understand will occasionally have a case head south, I usually reply something like: "Well, Dr. X is a competent doctor and your condition is difficult to diagnose. It's probable your condition was not manifest when you last saw them." For others, where I may find it hard to be complimentary, I may reply similarly to this: "Well, I'm not sure exactly what Dr. X was seeing at that time, but this is what you have now and this is how I'm going to care for you."

Did I throw the previous doctor under the bus? No, I redirected, took control of the discussion and told the patient what I would do for them. It's actually quite an effective tactic, especially when a new-to-you patient has been seeing a different provider and you're delivering news they have not heard previously.

Remember, our duty is to the patient and the profession. Treat each patient as you would want to be treated. Is it in a patient's best interest to be seen by a provider who is very anti-optometry? No, but neither is it in their interest to hear you grouse about organized medicine.

The provider in this patient's earlier care was someone who did not respect optometry, did not work with optometry and who at every occasion took the opportunity to disparage optometry. When the shoe is on the other foot, don't be like them! We can work around providers like that and still care for the patient. ■

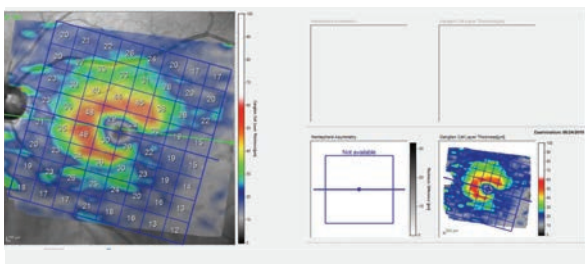


Fig. 3. This macular image demonstrates significant thinning of the ganglion cell layer contiguous with the eroded inferotemporal neuroretinal rim as seen in Figure 1.

Edited By Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

A Two-for-one Deal

Here's when to consider referring your patient for phaco-ECP, which addresses cataracts and glaucoma in one operation. By Christina Tran, BS, and Leonid Skorin, Jr., DO, OD, MS

Combining cataract phacoemulsification with endoscopic cyclophotocoagulation (ECP) of the ciliary processes can help lower a patient's intraocular pressure (IOP) by decreasing the production of aqueous humor. Studies show the combination procedure can achieve an average IOP decrease between 2.6mm Hg and 3.3mm Hg.[1,2] Several studies found the average number of post-op glaucoma therapies decreased by one medication two years post-op.[2-4]

The endoscope is inserted through the phacoemulsification incision, and the second procedure only adds a few minutes to the operating time.

The IOP-lowering effect is equivalent to procedures such as trabeculectomy and insertion of drainage devices but comes with fewer complications of hypotony or phthisis.[5] Other advantages over MIGS include no need to implant a device in the eye and decreasing IOP (aqueous) production versus working on IOP outflow.

Ideal Candidates

Patients need to qualify for both cataract surgery and ECP to qualify for the combined procedure. Most types of glaucoma can be treated with ECP with the exception of active uveitic glaucoma and patients with IOPs greater than 40mm Hg.[6] ECP is a viable option for patients struggling with medication compliance and want to reduce their dependence on multiple topical medications.

Using a video monitor, the surgeon locates and treats the ciliary processes, which become white and shrunken.

Step-by-step

After phacoemulsification of the cataract and insertion of the intraocular lens (IOL), the surgeon injects non-preserved lidocaine intracamerally for additional anesthesia. Viscoelastic injected over the capsular bag and under the iris creates more space for the endoscope, reduces the risk of damaging the iris or the IOL and allows for a clear view of the ciliary processes.[7] The surgeon inserts endoscope through the incision and applies treatment to at least 270 degrees of the ciliary processes with 0.25 watts on a continuous mode.[7,8] Ciliary processes become white and shrunken after a few seconds of treatment. After treatment, the endoscope is removed and viscoelastic is aspirated.

Post-op Considerations

The patient is seen at one day, one week and one month after the procedure. Their instructions are almost identical to those after cataract surgery. The patient should avoid rubbing their eyes for one week and will need to wear a plastic eye shield at bedtime. The patient is prescribed topical moxifloxacin and ketorolac QID. After phaco-ECP, patients will have more postoperative iritis and will need to use topical prednisolone every two hours for one week, which will then be tapered over the next month. The patient should continue using their glaucoma medications, as it may take four to six weeks for the procedure to take maximal effect.[7,8] Glaucoma medications may be tapered off accordingly as IOPs begin to drop. ■

Ms. Tran is a fourth-year student at Pacific University College of Optometry.

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

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

1. Roberts SJ, Mulvahill M, SooHoo JR, et al. Efficacy of combined cataract extraction and endoscopic cyclophotocoagulation for the reduction of intraocular pressure and medication burden. Int J Ophthalmol. 2016;9(5):693-98.
2. Siegel MJ, Boling WS, Faridi OS, et al. Combined endoscopic cyclophotocoagulation and phacoemulsification versus phacoemulsification alone in the treatment of mild to moderate glaucoma. Clin Exp Ophthalmol. 2015;43(6):531-39.
3. Francis BA, Berke SJ, Dustin L, Noecker R. Endoscopic cyclophotocoagulation combined with phacoemulsification versus phacoemulsification alone in medically controlled glaucoma. J Cataract Refract Surg. 2014;40(8):1313-21.
4. Clement CI, Kampougeris G, Ahmed F, et al. Combining phacoemulsification with endoscopic cyclophotocoagulation to manage cataract and glaucoma. Clin Exp Ophthalmol. 2013;41(6):546-51.
5. Sun W, Yu CY, Tong JP. A review of combined phacoemulsification and endoscopic cyclophotocoagulation: efficacy and safety. Int J Ophthalmol. 2018;11(8):1396-1402.
6. Kahook MY, Noecker RJ. Endoscopic cyclophotocoagulation. Glaucoma Today. 2006;4(6):24-9.
7. Berke SJ. Endophotocoagulation. In: Glaucoma. 2nd ed. Philadelphia: Elsevier; 2015:1160-66.
8. Fallano KA, Conner IP, Noecker RJ, Schuman JS. Cyclodestructive procedures in glaucoma. In: Ophthalmology. 5th ed. Philadelphia: Elsevier; 2019:1131-34.

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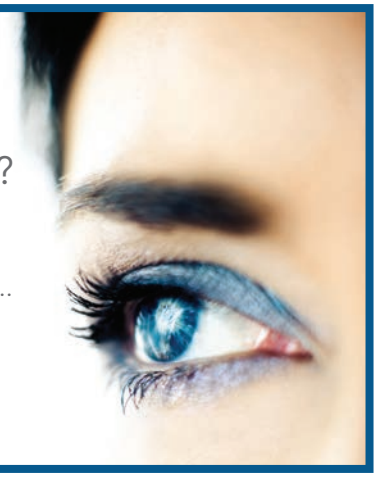

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High Risk, Limited Options

Treatment for limbal stem cell deficient eyes can take one of two routes, neither of which guarantees a successful outcome. **Edited by Joseph P. Shovlin, OD**

Q I have a patient with severe limbal stem cell deficiency (LSCD) who was told by her cornea specialist that she is at high risk for corneal transplantation and her only option is a keratoprosthesis. What is her prognosis with this procedure? What are the risks associated with it? Does she have any other options?

A "There are multiple facets to this question," according to Scott G. Hauswirth, OD, who practices and teaches in Colorado, "but it is important to understand the basic premise behind this dilemma." He says a high-risk transplant is usually defined as one that has a vascularized cornea or a history of multiple grafts. Immunologic rejection can occur in up to 70% of these grafts, even with aggressive, local immunosuppressive therapy.[1]

Limbal Territory

The limbus is the border between the cornea and the sclera and is typically 1mm to 2mm wide. It contains a variety of cells with various functions, including the limbal stem cell niche, which is home to the progenitor cells that eventually differentiate and migrate across the cornea to form the layers of the corneal epithelium. Dr. Hauswirth notes that insult to this area decreases its ability to regenerate a healthy corneal epithelium and disrupts the barrier function of the limbus, without which the cornea would become repopulated by conjunctiva, leading to stromal haze, vascularization, opacification and scarring.

Unfortunately, corneal transplantation in patients with LSCD is often destined to fail and represents a significant challenge to practitioners and surgeons.[2-4]

Choose Wisely

According to Dr. Hauswirth, there are two methods to address this challenging case—keratoprosthesis or limbal stem cell transplantation, followed by penetrating keratoplasty. While both are viable choices, he says the decision to choose one over the other depends on the number of previous corneal transplantation attempts and the degree of scarring and loss of viable limbus. Surgeon comfort and experience also play roles, he adds. It's worth noting that the limbal stem cell transplantation option typically only works well if the patient does not have severe dry eye.[3]

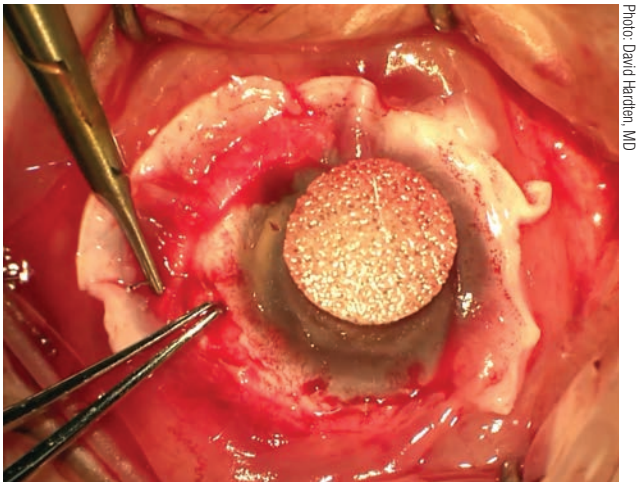

Photo: David Hardten, MD

This patient is undergoing limbal stem cell transplantation.

Multiple studies show good results for keratoprosthesis in eyes with LSCD.[5-7] One large literature review noted that 64.1% of eyes reached visual acuities better than 20/200 with a device retention rate of 88.9% over an average follow-up period of 25 months.[8] Similarly, a study comparing the results of Boston Type I keratoprosthesis implantation in patients with or without LSCD revealed a device retention rate of 75% and visual acuities of at least 20/200 in 77% of patients in the LSCD cohort.[5] Outcomes of patients who underwent limbal stem cell transplantation and penetrating keratoplasty were demonstrated in a study of 48 eyes with LSCD—90% of which were considered high-risk—that achieved a three-year graft survival rate of 62.5%.[4]

Dr. Hauswirth says keratoprosthesis has evolved to solve the problems presented by multiple graft failures, high-risk grafts and LSCD. He notes, however, that creating a device with biologically inert material that can be incorporated into the ocular tissues to replace the cornea is a novel approach that has taken many different revisions to even come close to perfecting. Dr. Hauswirth adds that while Boston type I keratoprosthesis is the

most common in the United States, other methods, such as tibial bone keratoprosthesis and osteo-odonto-keratoprosthesis, have performed well in clinical trials, and some, including AlphaCor keratoprosthesis, are available but have had a less robust uptake in the United States.[9]

According to Dr. Hauswirth, limbal stem cell transplantation techniques differ based on the origin of the transplanted tissues and their placement location on the eye but usually involve the use of systemic immunomodulatory medications that all have their own set of risks.[10] He notes that these stronger local and systemic immunomodulators keep the host immune system from attacking the new limbal cells, ensuring corneal graft survival.

Dr. Hauswirth says the limbal cells can be harvested through autologous cultivation (from a presumably healthy second eye), living donor cultivation or allogenic donor cultivation. In the case of allogenic harvesting, ABO/HLA tissue matching is preferred.[11] He notes that while the cells can also be harvested from a cadaver with or without ABO/HLA matching, these patients would likely then be on long-term immunosuppression. After harvesting a 1mm to 2mm section of the limbus, Dr. Hauswirth says the cells are then cultivated on organic media until they reach a size where they can be directly transplanted to the host.

On the horizon are methods that involve transplanting stem cells derived from other areas, such as the oral mucosa, and *ex vivo* methods of stem cell cultivation, including mesenchymal stem cell harvesting.[12,13] However, these methods are rather new and do not have a long track record of success like the two discussed earlier, both of which are worth looking into. ■

1. Jabbehdari S, Baradaran Rafii A, Yazdanpanah G, et al. Update on the management of high-risk penetrating keratoplasty. Curr Ophthalmol Rep. 2017;5(1):38-48.
2. Dua HS, Saini JS, Azuara-Blanco A, et al. Limbal stem cell deficiency: concept, aetiology, clinical presentation, diagnosis and management. Indian J Ophthalmol. 2000;48(2):83-92.
3. Sacchetti M, Rama P, Bruscolini A, et al. Limbal stem cell transplantation: clinical results, limits and perspectives. Stem Cells Int. 2018.
4. Borderie VM, Levy O, Georgeon C, et al. Simultaneous penetrating keratoplasty and amniotic membrane transplantation in eyes with a history of limbal stem cell deficiency. J Fr Ophthalmol. 2018;41(7):583-91.
5. Aravena C, Bozkurt TK, Yu F, et al. Long-term outcomes of the Boston type I keratoprosthesis in the management of corneal limbal stem cell deficiency. Cornea. 2016;35(9):1156-64.
6. Basu S, Taneja M, Narayanan R, et al. Short-term outcome of Boston type 1 keratoprosthesis for bilateral limbal stem cell deficiency. Indian J Ophthalmol. 2012;60(2):151-3.
7. Sejpal K, Yu F, Aldave AJ. The Boston keratoprosthesis in the management of corneal limbal stem cell deficiency. Cornea. 2011;30(11):1187-94.
8. Shanbhag SS, Saeed HN, Paschalis EI, et al. Boston keratoprosthesis type 1 for limbal stem cell deficiency after severe chemical injury: a systematic review. Ocul Surf. 2018;16(3):272-81.
9. De la Paz MF, Salvador-Culla B, Charoenrook V, et al. Osteo-odonto-, Tibial bone and Boston keratoprosthesis in clinically comparable causes of chemical injury and autoimmune disease. Ocul Surf. April 12, 2019. (Epub ahead of print).
10. Ballios BG, Weisbrod M, Chan CC, et al. Systemic immunosuppression in limbal stem cell transplantation: best practices and future challenges. Can J Ophthalmol. 2018;53(4):314-23.
11. Cheung AY, Sarnicola E, Kurji KH, et al. Cincinnati protocol for preoperative screening and donor selection for ocular surface stem cell transplantation. Cornea. 2018;37(9):1192-7.
12. Choe HR, Yoon CH, Kim MK. Ocular surface reconstruction using circumferentially-trephined autologous oral mucosal graft transplantation in limbal stem cell deficiency. Korean J Ophthalmol. 2019;33(1):16-25.
13. Yazdanpanah G, Jabbehdari S, Djalilian AR. Emerging approaches for ocular surface regeneration. Curr Ophthalmol Rep. 2019;7(1):1-10.

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Diagnostic **Quiz**

Painless But Suffering

By Andrew S. Gurwood, OD

History

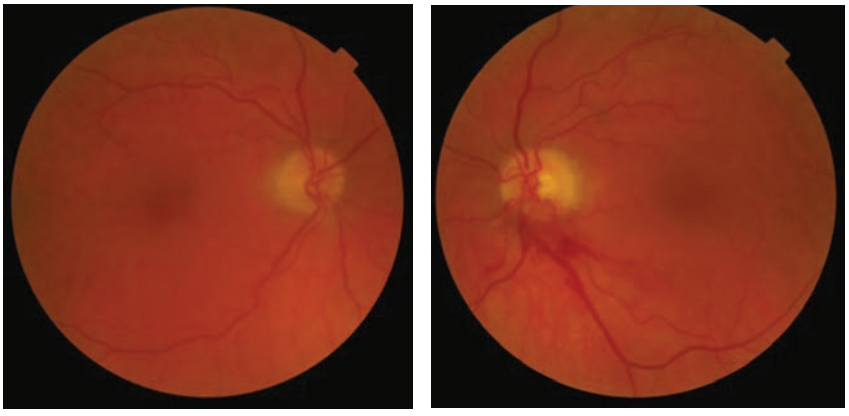
A 77-year-old Caucasian male presented to the office with a chief complaint of poor vision in his left eye for five days. He explained that, upon waking, he noticed his vision was bad.

He recounted having been treated by a retinal specialist the last time this happened, that time to his right eye. He reported no pain. His history included medically controlled diabetes and hypertension. He denied allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OD and 20/200 OS at distance and near. His external examination uncovered a central scotoma in the left eye upon facial Amsler testing.

All other external findings were normal, and there was no evidence of afferent pupillary defect. Biomicroscopic examination of the anterior segment was normal in the right eye, but found suspended red cells in the anterior chamber of the left. Goldmann applanation tonometry measured 15mm Hg OU.



This 77-year-old patient has had poor vision in his left eye for nearly a week. Can the details of his exam and these fundus images explain the cause of his disturbance?

Your Diagnosis

Does the case presented require any additional tests, history or information? What steps would you take to manage this patient? Based on the information provided, what would be your diagnosis? What is the patient's most likely prognosis? To find out, please visit www.reviewofoptometry.com. ■

Retina Quiz Answers

(from page 80): 1) b; 2) a; 3) c; 4) b.

Next Month in the Mag

Coming in September, *Review of Optometry* will present its Annual Technology Issue. Topics include:

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