Urgent Care: Anaphylaxis in Your Exam Lane, *p. 71*

REVIEW® OF OPTOMETRY

January 15, 2019

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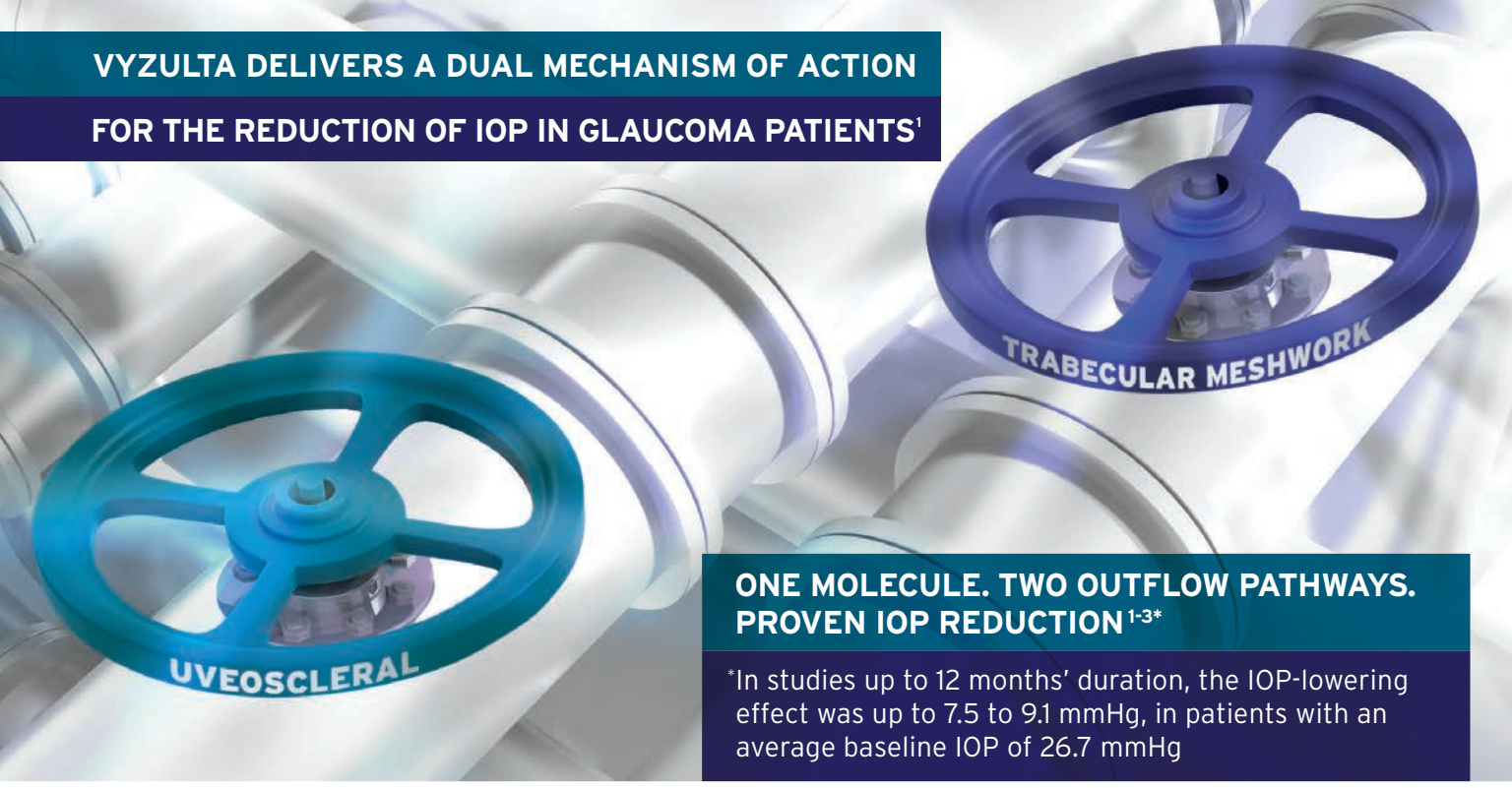
MINIMIZING MYOPIA

We may never truly eradicate it. But optometrists can make some of its most pernicious effects disappear.

VYZULTA DELIVERS A DUAL MECHANISM OF ACTION FOR THE REDUCTION OF IOP IN GLAUCOMA PATIENTS[1]



ONE MOLECULE. TWO OUTFLOW PATHWAYS. PROVEN IOP REDUCTION[1-3*]

*In studies up to 12 months' duration, the IOP-lowering effect was up to 7.5 to 9.1 mmHg, in patients with an average baseline IOP of 26.7 mmHg

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 (CONTINUED)

- 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. 2017.
2. Weinreb RN, Sforzolini BS, Vittitow J, Liebmann J. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: the APOLLO study. *Ophthalmology*. 2016;123(5):965-973.
3. Medeiros FA, Martin KR, Peace J, Sforzolini BS, Vittitow JL, Weinreb RN. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. *Am J Ophthalmol*. 2016;168:250-259.

For more information about VYZULTA and how it works, visit vyzultanow.com

VYZULTA™
(latanoprostene bunod ophthalmic solution), 0.024%

BAUSCH + LOMB

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.

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IN THE NEWS

The **Council on Optometric Practitioner Education (COPE) recently restructured to include the American Academy of Optometry (AAO) and the Association of Schools and Colleges of Optometry (ASCO)** to further grow optometry through regulated oversight of the discipline's education. The new governing committee will be comprised of representatives of the AAO, ASCO and the Association of Regulatory Boards of Optometry (ARBO).

A nationwide study in Denmark demonstrated an **increased incidence of cancer in patients with retinal vein occlusion (RVO)** compared with age- and gender-matched controls. Researchers assessed 7,963 patients without cancer at the time of the first RVO diagnosis, and a control cohort of 39,815. The analysis revealed an RVO diagnosis was associated with a 22% increased risk of being diagnosed with cancer. The researchers believe their study suggests the association is mainly due to shared risk factors.

Toft-Petersen AP, Muttuvelu DV, Heegaard S, Torp-Pedersen C. Correlation between retinal vein occlusion and cancer-a nationwide Danish cohort study. Acta Ophthalmol. 2018;96:800-3.

Researchers analyzed a subset of the Twins Early Development Study and found **factors significantly associated with myopia included level of maternal education, fertility treatment, summer birth and hours spent playing computer games**. The researchers also noted associations with socioeconomic status, educational attainment, reading enjoyment and certain cognitive variables.

Williams KM, Kraphol E, Yonova-Doing E, et al. Early life factors for myopia in the British Twins Early Development Study. British Journal of Ophthalmology. Published Online First: 06 November 2018.

Antivirals Plus Steroids Effective in HZO

Majority believe treatment combo works for both recent-onset and chronic HZO.

By Jane Cole, Contributing Editor

Slightly more than half of the investigators from the Zoster Eye Disease Study (ZEDS) treat herpes zoster ophthalmicus (HZO) with prolonged oral antivirals along with topical steroids, and two-thirds believe this regimen is effective, a recent study says.

In an effort to determine practices and opinions among study investigators in ZEDS regarding suppressive valacyclovir treatment for recent-onset and chronic HZO, a team of investigators collected data from an internet-based survey sent to 170 ZEDS investigators. The study polled the ZEDS researchers on treatment practices for stromal keratitis in HZO and their opinions regarding the efficacy of prolonged antiviral prophylaxis. The response rate was 72.4% (123 out of 170).

The study found topical steroids and oral antivirals were used by the majority of respondents for stromal keratitis in both recent-onset (69.1%, or 85 out of 123) and chronic HZO (63.4%, or 78 out of 123). Additionally, researchers reported the duration of treatment was similar in both recent-onset and chronic HZO with 50.4% (124 out of 246) of ZEDS investigators using prolonged treatment for stromal keratitis due to recent-onset or chronic HZO.

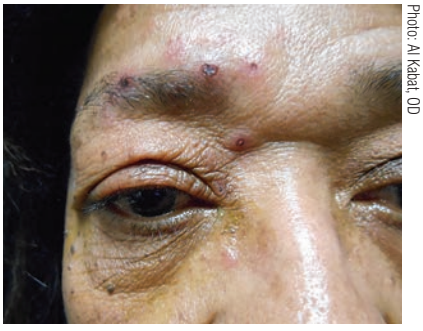

Photo: Al Kabat, OD

Prompt intervention is required for patients with active HZO.

The study also reported 70.7% (87 out of 123) ZEDS respondents believed oral antivirals were effective during treatment.

"The ZEDS randomized placebo controlled clinical trial of prolonged suppressive valacyclovir treatment is a unique opportunity to obtain evidence to determine standard of care in treatment of HZO to reduce complications and improve outcomes," the study authors wrote.

Completion of ZEDS, they added, was vital to determine whether or not these practices are effective. "Participation in this study is necessary to obtain evidence to support treatment" options commonly used by clinicians and believed to be appropriate.

Lo DM, Jeng BH, Gillespie C, et al. Current practice patterns and opinions on the management of recent-onset or chronic herpes zoster ophthalmicus of zoster eye disease study investigators. Cornea. 2019 Jan;38(1):13-17.

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Ohio Vision Bill Provides More Leeway

Ohio's optometrists are celebrating the passage of an over-arching bill designed to give ODs more autonomy in dealings with insurers. After a near-unanimous vote—91-2 in the House and 31-0 in the Senate—the new legislation was signed into law by Ohio's Governor John Kasich in late December. In a joint announcement, the Ohio Optometric Association and the National Association of Vision Care Plans (NAVCP) applauded the measure, saying it "will provide consumers with an enhanced ability to make informed choices about their vision care." The two groups worked closely with legislators to develop the bill.

The new guidelines essentially feature three components, explains Michael Earley, OD, Ohio Optometric Association president and associate dean of academic affairs at Ohio State University's College of Optometry.

First, it allows ODs to use any lab—previously they had to use the insurance company's lab. The insurer can still adjust its level of coverage, but can't deny coverage to any lab completely. Additionally, both the doctor and the insurer must be transparent to the patient about the lab they use.

Second, it stymies any attempt by insurance companies to force ODs to accept a vision discount plan. In the past, insurers mandated optometrists accept these if they want to be on a medical plan, too. Some optometrists are losing money on this, causing them to drop the medical plan and lowering the number of ODs a patient could see in the area, said Dr. Earley. Ophthalmologists aren't treated like this—and now, optometrists won't be either.

Third, it restructures what Dr. Earley calls "uncovered benefits," in a way that gives ODs dominion over what discounts they accept.

"Many vision plans negotiate discounts on behalf of their members so that they can get lower-priced eyewear with their providers once their benefit runs out, for additional pairs, sunglasses" and such, an NAVCP spokesman explained to *Vision Monday*. ODs were not reimbursed by the discounts. "This bill still allows vision care providers to participate in these discount plans if they want to, but assures that they aren't mandatory," the spokesman said.

Additionally, it allows doctors to offer their own benefits such as "faithful patient discounts."

NAVCP. Ohio Optometric Association and NAVCP Applaud Governor Kasich for Signing "Consensus-driven" Vision Health Legislation. netforum.avectra.com/eWeb/DynamicPage.aspx?Site=NAVCP&WebCode=ArticleDetail&faq_key=6471f946-c7b0-4ad4-a55a-39b1045c8b4d. December 20, 2018. Accessed December 21, 2018.

IIH on the Rise Abroad

An observational study of hospitals in England over a 14-year period has revealed the incidence of idiopathic intracranial hypertension (IIH) has increased by 108% and that the hospital economic burden has risen from £9.2 million in 2002 to £49.9 million in 2014, with a cost forecast of £462 million per year by 2030.

The study analyzed a total of 23,182 new IIH cases, of which 52% resided in the most socially deprived areas. Peak incidence occurred in females aged 25, with the incidence in women being four times higher than in males. Elective Caesarean section rates were significantly higher in IIH (16%) compared with the general population (9%). Admission rates rose by 442% within the 14 years, with 38% having repeated admissions in the year following diagnosis.

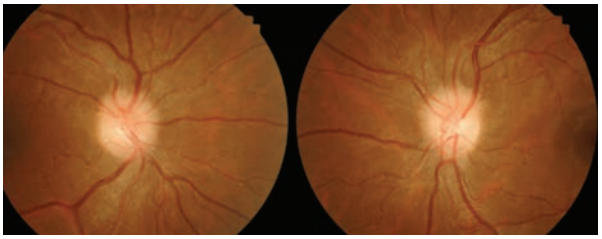

Photo: Joseph W. Sowka, OD

This patient with IIH has mildly elevated and hyperemic optic nerve heads in both eyes, with indistinct margins and a cup-to-disc ratio of approximately 0.2/0.2.

Adult IIH has not been previously associated with social deprivation and adverse obstetric outcomes. Researchers believe these factors are new signals reinforcing that this disease should not be assumed "benign." The increased incidences, hospital visits and the resultant economic burden could have wide reaching implications. They conclude that developing novel therapeutic options may help to reduce the burden.

Mollan SP, Aguiar M, Evison F, et al. The expanding burden of idiopathic intracranial hypertension. Eye. October 24, 2018. [Epub ahead of print].

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New Data Suggests Cerebrospinal Outflow Slows in Glaucoma

At optometric conferences and in the academic literature, speculation about the connection between intraocular pressure (IOP) and cerebrospinal fluid (CSF) has been building in recent years. Researchers, such as John Berdahl, MD, suspect that the CSF holds the key to understanding glaucoma.[1]

Investigators in that camp are now emboldened by new evidence that shows, for the first time, that CSF's entry into the optic nerve subarachnoid space is impeded in glaucoma.[2]

The mouse-model research included eight subjects with glaucoma for 10 months and another nine with glaucoma for only two months.[2] The investigators discovered the CSF flowed more into the areas around the optic nerve of the two-month-old glaucoma cohort than the 10-month-old cohort.[2] The researchers used a CSF-injected tracer dye to track the flow.[2] The tracer was found in the vessels around the eye in six out of nine of the two-month cohort.[2] However, when the same test was run on the 10-month-cohort, only three of the eight mice showed the tracer anywhere in the subarachnoid space.[2] For comparison, eight age-controlled mice without glaucoma all showed the tracer.[2] Simply put, glaucoma is gumming up the cerebrospinal works, especially as it progresses.

"This finding suggests an association between CSF flow obstruction and axon pathology in this glaucoma model, although a causative relationship cannot be concluded," the authors noted.[2]

1. Berdahl J, Allingham R, Johnson D. Cerebrospinal fluid pressure is decreased in primary open-angle glaucoma. Ophthalmol. 2008;115(5):763-8.
2. Mathieu E, Gupta N, Paczka-Giorgi L, et al. Reduced cerebrospinal fluid inflow to the optic nerve in glaucoma. IOVS. 59(15):5876–5884

Another Advance for Glaucoma Diagnosis

Characterized by a progressive loss of ganglion cells and the retinal nerve fiber layer and related to a higher intraocular pressure (IOP) than normal, glaucoma can be divided into open-angle and angle-closure, which is more aggressive and known for reaching extremely high IOP levels.

At the time the technique known as a prone position provocative test for angle-closure glaucoma was created, optical coherence tomography (OCT) did not exist, so angle-closure glaucoma was determined by comparing IOP measurements before and after patients lie in the prone position.

A team of researchers improved this technique by incorporating OCT to help identify the camerular sinus type and improve the diagnosis of angle-closure glaucoma by performing OCT of the anterior segment in the lying position.

Silva HRR. OCT in prone position-a new approach to glaucoma. Clin Exp Ophthalmol. December 2018. [Epub ahead of print].

Better Lid Hygiene Reduces Dry Eye

Researchers in Russia have found that improved eyelid hygiene before refractive surgery in patients with dry eye and meibomian gland dysfunction (MGD) leads to a more significant correction of the eye surface condition compared to isolated bioprotective and reparative therapy.

The study separated 48 women with myopia, dry eye and MGD of noninfectious etiology into two groups. The first group received presurgical treatment for dry eye symptoms—eyelid cleaning, applying warm compresses on the area and massage of eyelid margin—alongside bioprotective and reparative therapy twice a day for two months. The second group only received bioprotective and reparative therapy for presurgical time period.

The group who received eyelid hygiene had a greater reduction on the Ocular Surface Disease Index and xerosis index, as well as greater tear break-up time. The significant decease in MGD severity and a decrease in lipid deficiency, attributed to the eyelid hygiene, caused these changes. The results provide an opportunity to explore more favorable conditions for surgical correction of ametropia, the researchers concluded.

Sakhnov SN, Yanchenko SV, Malyshev AV, et al. Dry eye treatment optimization in patients prior to refractive surgery. Russ Ophthalmol J. 2018;11(4):87-95.

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*HA is an inactive ingredient.

Sunlight Exposure, AMD Not Linked

Researchers in China have found that sunlight exposure may have no bearing on risk of age-related macular degeneration (AMD) based on current published data. The team analyzed 14 eligible studies that included 43,934 individuals. In subgroup analyses, the study found only one mildly significant association in the case-control studies but not in the cross-sectional studies. When they stratified the data based on the stage of AMD, method of exposure assessment and study latitude, the researchers observed similar insignificant results. When observing sun-avoidance behaviors, the study found that subjects who used sunglasses or hats regularly didn't have a decreased risk of AMD.

The researchers did note that it was difficult to quantify sunlight exposure objectively. Assessments of total sunlight exposure were based on questionnaires, and the accuracy of the data obtained depended heavily on question quality and respondents' memory. While some studies used sunlight-related factors to assess exposure, the relationship between proxy measures for sunlight exposure and AMD was not conclusive. The common limitation in the studies was that the correlation between such proxies and true sunlight exposure was overall unknown.

Zhou H, Zhang H, Yu A, Xie J. Association between sunlight exposure and risk of age-related macular degeneration: a meta-analysis. BMC Ophthalmol. December 20, 2018. [Epub ahead of print].

Assessing the Choroid in Diabetes

Pediatric patients with diabetes might benefit from regular screenings with spectral-domain optical coherence tomography (SD-OCT) to catch early choroidal thickening, according to new research. An international team recently discovered that changes in the choroid occur in children with diabetes mellitus and seem to progress with disease duration.

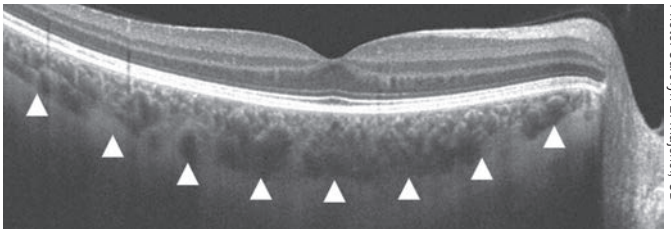

Photo: Carolyn E. Majcher, OD

Analyzing the choroid with OCT could be a beneficial screening tool, even for children. This OCT shows a healthy eye, and the white arrows correspond to the choroid/scleral junction.

The new study also found choroidal thickness increased despite the lack of other signs of diabetic retinopathy (DR) and without retinal thickening. While other studies have already documented similar findings in adults, this is the first to look closely at the pediatric population.[1]

The researchers studied 121 children with diabetes—ages six through 18—and 32 age-matched controls. The participants were divided into three groups: those with the condition for less than five years (group 1), five to 10 years (group 2) and more than 10 years (group 3). Using SD-OCT, researchers found group 3 had significantly thicker choroids, an average of 367.4µm, than the controls (305.5µm) and both groups 1 and 2 (309.2µm and 315.2µm, respectively).[1]

The researchers note the data contradicts other studies that found no significant difference from controls in choroidal thickness between children with diabetes without DR.[2,3] However, the earlier studies had small study groups that were hampered by the significant variability of the disease. The current, larger study group also includes a significantly longer duration of diabetes, with a maximum of 15.9 years, compared with zero to 10 years for the earlier studies.[1]

The findings could lead to a new screening opportunity, the authors wrote—an important need, considering "children are at high risk of developing DR in adulthood because of long diabetes mellitus duration," the study says. Identifying and tracking choroidal thickening early could help clinicians better counsel patients about the importance of good glycemic control to reduce the risk of possible ocular sequelae such as diabetic choroidopathy and DR. ■

1. Niestrata-Ortiz M, Fichna P, Stankiewicz W, Stopa M. Determining the effect of diabetes duration on retinal and choroidal thicknesses in children with type 1 diabetes mellitus. Retina. December 18, 2018. [Epub ahead of print]
2. Sayin N, Kara N, Pirhan D, et al. Evaluation of subfoveal choroidal thickness in children with type 1 diabetes mellitus: an EDI-OCT study. Semin Ophthalmol. 2014;29:27-31.
3. Elhabashy S, Elbarbary N, Nageb K, Mohammed M. Can optical coherence tomography predict early retinal microvascular pathology in type 1 diabetic adolescents without minimal diabetic retinopathy? A single-centre study. J Pediatr Endocrinol Metab. 2015;28:139-146.

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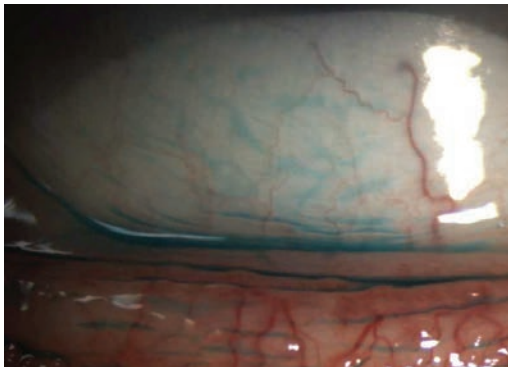

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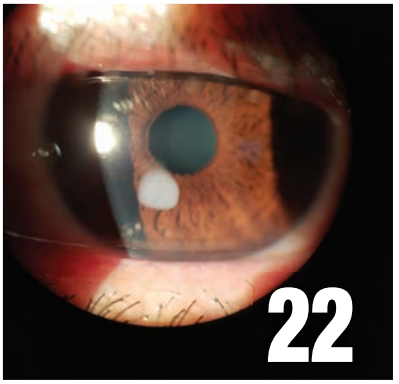

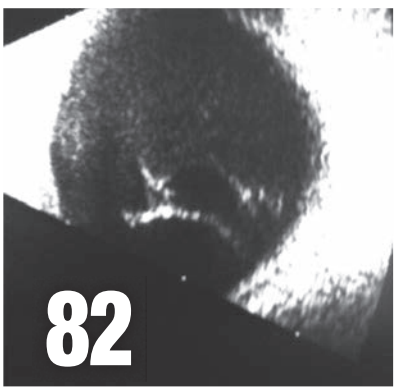

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Outlook

By Jack Persico, Editor-in-Chief

Be Farsighted About Myopia

Consider it a disease, not a trait. And then treat it like any other pathology.

It's hardly breaking news that myopia is exceedingly common. After all, a condition that affects such a wide swathe of the population doesn't exactly escape notice. Myopia is also optometry's oldest adversary. Measuring refractive error was literally built into the earliest definition of the profession, and correcting it sustained practitioners for decades before the rise of medical optometry.

It's tempting to think of refractive errors as simple traits dictated by genetic factors, like hair or eye color. Just a fact of life. And we sometimes bring that same passive acceptance to the modern-day environmental factors that are accelerating its prevalence, like increased near-vision tasks in a digital device–centric world and reduced time spent outdoors in developed, industrialized societies.

So, why is myopia suddenly being discussed with an urgency and alarm usually reserved for a disease epidemic—think Spanish flu or AIDS—that comes out of nowhere and strikes without warning? At the risk of oversimplifying, I think we can thank one man: the late Brien Holden, who passed away in the summer of 2015.

Professor Holden is of course renown for a career's worth of breakthroughs and insights. Soft contact lenses as we know them today wouldn't exist without him, for one.

But his parting gift, to optometry and the world, was calling out myopia for what it is: a disease, one that can and should be influenced by doctors for the good of the populace, or else we should expect dire consequences.

"I don't want to be an alarmist," Brien told the Australia Broadcasting Corporation mere weeks before his death, "but the fact of the matter is if we ignore our children becoming short-sighted, and increasing rapidly, they are at risk for future life." In that interview, he was getting the word out about his then-forthcoming study of myopia prevalence that predicted over half the global population becoming myopic by the year 2050, including one billion high myopes. "That of course makes it very difficult for children to see, to learn, people to work, older people to survive," he told the news service at the time.

This landmark study—Brien's last major work—was a wake-up call to the eye care community and other stakeholders in health care: insurers, politicians, industry and mom and dad. Hopefully, we're entering the next phase of myopia, where all the aforementioned are working to implement new ideas and policies aimed at stemming the tide.

Changes in thinking and behavior are never easy. But everyone can, and should, play a part. Manufacturers are developing targeted products. The researchers at the Brien Holden Vision Institute are working hard to continue the legacy of their founder. We at *Review* have devoted this issue to myopia management to help give you a roadmap forward. And as the parent—a myopic one, at that—of a two-year-old, I'll be sure to take my son outside to play more often (knowing the protective effect it confers) and will keep my iPhone out of his hands as much as possible. As is often the case, it takes a village. ■

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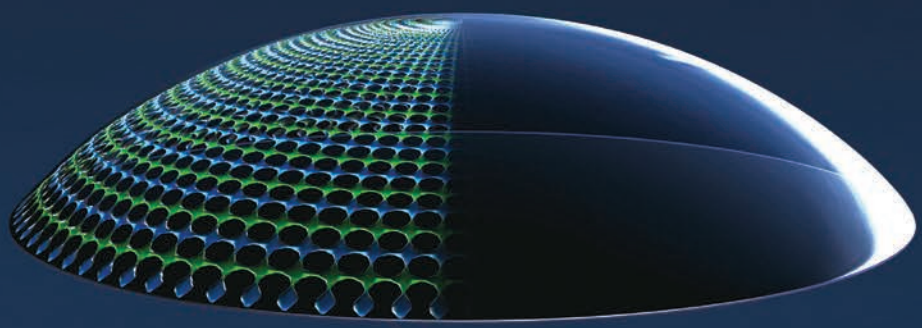

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New Year, New Toys

The kids aren't the only ones with new gadgets this year—2019 promises many new technologies for your practice, too. **By Paul M. Karpecki, OD, Chief Clinical Editor**

At the beginning of a new year, we have an incredible opportunity to take stock of the previous year's successes and failures. We also have a chance to reposition our practices and try to anticipate where things are going. The following new technologies may be instrumental in shaping the future of clinical care in 2019 and beyond.

Glaucoma

Optometry plays a key role in glaucoma management, and we now have new drugs to improve our efforts. Vyzulta (Bausch + Lomb) can lower IOP by as much as 9.1mm Hg with Qhs dosing. Another new option is Rhopressa (Aerie Pharmaceuticals), a rho-kinase inhibitor designed to expand the trabecular meshwork. In 2019 we might see Roclatan (Aerie Pharmaceuticals), a combination of Rhopressa with latanoprost.

We also have new surgical options to recommend, such as additions to the slate of minimally invasive glaucoma surgery (MIGS) devices The recently approved Hydrus microstent (Ivantis) has shown one of the greatest IOP lowering of any MIGS device in FDA clinical studies to date in patients undergoing phacoemulsification with Hydrus vs. those undergoing phaco alone. Most notably, the delta increased from one to two years post-procedure.

As far as diagnostics, hysteresis data (Ocular Response Analyzer, Reichert) has been validated to predict field loss progression.

Genetics

Many diseases we diagnose arise from gene mutations. Conditions such as Leber's and Stargardt's disease may see treatment options in the near future (Ophthotech), and even common conditions such as age-related macular degeneration (AMD) and glaucoma have genetic origins. Genetic testing is expanding, and some are now fully covered by insurance, including testing for corneal dystrophies. In 2019, a test that can help diagnose keratoconus at an early stage may hit the market (Avellino Labs). Other companies continue to work on vast retinal genetic testing akin to 23andMe but for ocular conditions (EyeCheck).

Contact Lenses

This year, we can expect a long-awaited roll-out of a toric presbyopic soft contact lens from Bausch + Lomb. In time, we may also see drug-eluting contact lenses (Johnson & Johnson Vision and OcuMedic) and lenses to measure IOP (Sensimed) that could help us predict fast-progressing glaucoma patients.

Myopia Control

With greater awareness, it seems inevitable that myopia control will be a major opportunity for optometry. Clinicians who still need to invest in ultrasound for A-scan measurements might consider DGH's new option that is cheaper than a traditional ultrasound unit and uses tablet technology. Better access to specialty contact lenses and atropine drops will help clinicians better care for this patient population.

Ocular Surface Disease

This is on the list of opportunities each year, and in-office treatments with technologies such as LipiFlow (TearScience), Blephex and iLux (Alcon) are leading the way. Another device to expect in 2019 is Sight Sciences' TearCare.

Intense pulsed light is becoming a useful technology, as new entrants such as Lombard Medical have found ways to make the treatment more efficient and convenient. Blephex hopes to further expand its in-office treatments for biofilm with a product called Aurora.

Companies such as VitalTears now make autologous serum available nationwide, which is essential for patients with severe keratoconjunctivitis sicca. Punctal plugs that dissolve in 180 days (Oasis Medical, Beaver-Visitec International, OcuSoft, Paragon Biotech) are gaining traction as the optimal balance between duration and side effects.

Amniotic membrane and amnion-based eye drops (BioTissue, Ocular Sciences), expected in 2019, can help treat superficial punctate keratitis, persistent epithelial defects, neurotrophic keratitis and even neuropathic corneal pain. We may also see an ocular bandage lens made from cross-linked hyaluronic acid for the treatment of abrasions and epithelial defects (Eyegate).

In diagnosis, meibography (Oculus, Topcon, Johnson & Johnson,

Meibox, TelScreen) will become essential, as will point-of-care testing—especially with devices that can measure osmolarity and MMP-9 in one unit (TearLab). From a drug perspective, a new version of cyclosporine in a 0.09% concentration (Cequa, Sun Pharmaceuticals) was approved at the end of 2018.

Both Bausch + Lomb and Kala have or will advance the efficacy of loteprednol. Kala recently received FDA-approval for Inveltys, a 1% loteprednol that uses mucus-penetrating particles (MPPs) to reach the target tissue. Kala may also receive approval for a version of loteprednol for dry eye flare-ups.

Bausch + Lomb will soon launch a 0.38% loteprednol that appears to match or increase the efficacy of the current 0.5% by using sub-micron particles sizes, additional moisturizers and a better pH. It does not require shaking, adding one more improvement to the drug profile.

AMD Management

Today, clinicians could add an AMD management focus involving dark adaptometry (Maculogix), wet AMD monitoring (Notal Vision), blue light-blocking technology (e.g., BlueTech, Prevencia) and nutritional supplements. Low vision technologies are also crucial, and inter-professional referrals, such as to an OD colleague who specializes in low vision, should be on your mind for patients with advanced AMD.

Cataract Patient Management

The future of cataract surgery lies in technologies that give patients more options such as light-adjustable lenses (RxSight), femtosecond lasers applied to the IOL to correct post-cataract refractive error (PerfectVision) and products that allow for future IOL upgrades or the addition of augmented reality or monitoring technologies. Diagnostic technologies now combine Scheimpflug imaging with back corneal surface elevation for optimal calculations (Visonix, Oculus), and ocular scatter off the lens can help determine the exact level of cataract your dealing with or if the issue is on the ocular surface (Visiometrics).

All of these advances keep our profession moving forward, but we must not get caught up in the excitement. Remember to carefully evaluate how they can best serve our patients and enhance our practices. ■

Note: Dr. Karpecki consults for a number of manufacturers with products relevant to this topic.

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Attendees on-site can pick up the SECO Daily each morning for the latest news and highlights. Those at home can stay in touch, too—a digital edition of the SECO Daily will be posted online, plus an e-newsletter will be sent out each morning with the day's top stories.

Show copies will also be available at the *Review of Optometry* booth.

I'm in a Cleveland State of Mind

After speaking at a conference, I wondered, "Why, oh why, oh why, oh, would I ever leave Ohio?" **By Montgomery Vickers, OD**

It's a beautiful place. A location steeped in history. A calming, complete destination that, somehow, reveals its vibrancy around every corner. Lahaina? Maui? Nassau? No, I'm talking about Cleveland. Yes, THAT Cleveland. The one in Ohio.

I was asked to deliver the Keynote Address at the EastWest Eye Conference there. Since I hadn't heard back from The Hawaii Optometric Association—in fact, I actually have never heard from them, at all—I accepted the opportunity in Cleveland. (Hawaii, if you're listening, I am a very busy man so, if you want me there, I cannot possibly make it until later today.)

Now, this was the first time I had taken my show on the road in maybe 10 years. I had my reasons for the speaking sabbatical. I sold my practice, moved to Texas, spent time taking care of family, especially, as many of you know, my granddaughter, Grace, who has had three major heart surgeries. Thank you, my fellow doctors for your prayers and your financial gifts for Grace. She is five years old now; nobody expected her to survive, even a day. Grace's challenges will never go away but, man, is she the most wonderful person I have ever known!

Back to Ohio

Folks, I have had a longtime love for Ohio. I had the opportunity to lecture a few times at Ohio State University School of Optometry when I lived in West Virginia and I constantly say I have never met a stupid optometrist from Ohio State. Don't let me down, Buckeyes! Also, Grace has had all of her surgeries at Cincinnati Children's Hospital Medical Center. It's an amazing place full of amazing healers. So, when the Ohio Optometric Association called, I was immediately in!

I used to think Cleveland was cold, dreary, dirty, maybe even dying due to loss of industry and the city's unfortunate northern, blustery location. Any place can be dreary and cold I have since learned, as typically sunny Dallas has had a record cold rain that has lasted more than a month. Maybe I shouldn't have bought that giant SUV—my car apparently caused global warming, which has made it cold. OK, I haven't quite figured that part out yet, or how to get six grandkids into a smart car.

But Cleveland reminded me that I truly am, as many people remind me daily, an idiot. Cleveland is AWESOME! It's clean, gorgeous and growing. And the food scene? Crazy good, from Michael Symon's to Slyman's Deli, I dare you to beat Cleveland's choices and quality. Yes, Cleveland is a great weekend escape. Just not in January.

A better time to go is during the EastWest Eye Conference. Jordan Quickel and the Ohio Optometric Association folks know how to do it. The education is great, the venue is easily accessible and huge, and the extracurricular events are well done.

I even got to high five my old music buddies, Bad Habits: The Eye Docs of Rock. They always put on a great show and I still cannot figure out how that many rocking optometrists ended up in one place. They should have been something way cooler, like morticians. Maybe they made a wrong turn.

Anyway, I entertained the doctors and left them with smiles and hope and plenty of quizzical looks. At least they got an hour of CE. Now, they can go back to their staff members and patients and tell them even more dumb optometry jokes. ■

COMING 2019

Open your eyes to what's on the horizon in dry eye.

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TearCare is indicated for the application of localized heat when the current medical community recommends the application of a warm compress to the eyelids. Such applications would include Meibomian Gland Dysfunction (MGD), Dry Eye, or Blepharitis.

Caramel Eyes

Corneal burns aren't that common, but be prepared to identify and treat them.

Edited by Paul C. Ajamian, OD

Q I have an emergency walk-in patient who came into our practice complaining of a foreign body sensation after cooking. Upon slit-lamp examination, I found no foreign body but, instead, a white spot on the cornea. What could it be?

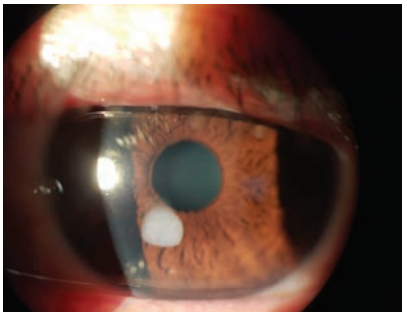
A As with all emergency patients, the most essential part of your exam is a careful case history. "Asking detailed questions is paramount," says Kirsten Weitzel, a fourth year optometry extern working with Dr. Ajamian at Omni Eye Services of Atlanta. Her patient that day, a 42-year-old bakery worker, stated that he was cooking hot caramel in a saucepan under medium heat when some of it splattered into his eye. Irrigation with water helped, but he still noticed a white spot on his eye when looking in the mirror (*Figure 1*). The patient stated the eye was mildly irritated and sensitive to light. His unaided vision was 20/20 OD and OS.

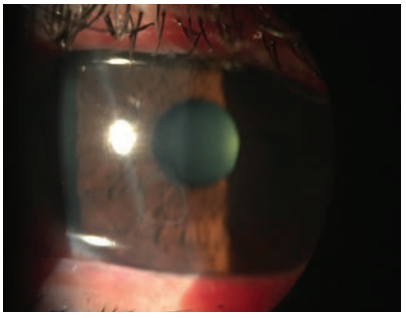
First-degree Examination

Ms. Weitzel advises, in any case of a suspected foreign body, to get the big picture by having the patient look up, down, left and right, and then evert the lids and make sure nothing else is hiding. Next, perform a complete corneal and anterior chamber evaluation.

After a thorough examination, the only noteworthy finding was the spot on the epithelium. The list of differentials included subepithelial infiltrate, corneal ulcer, corneal foreign body and thermal burn.

"While it is the clinician's job to consider every potential diagnosis, typically the simplest answer is most often the correct one," says Ms. Weitzel. Given the history and the exam, the team diagnosed their patient with a minor corneal thermal burn.





Figs. 1 and 2. Focal caramel burn and eye post-debridement.

These burns have many causes, but each culprit will have a slightly different presentation. The most common causes of thermal burns include: firework explosions, curling irons, cooking oil, boiling water/steam, molten metals/plastics and, of course, hot caramel!

Corneal thermal burns do not look like a corneal abrasion or foreign body, as the epithelium is typically not missing. Epithelial burns tend to appear as white, flat lesions, where the affected tissue is necrotic. The patient's symptoms will vary depending on the amount of corneal involvement and the extent of the burn.

Second-degree Management

Determine the depth of the burn, Ms. Weitzel advises. This indicates the amount of scarring, if any, that will occur after the cornea is fully healed.

The first step in thermal burn treatment is to debride the necrotic epithelium. Anesthetize the eye and run a lubricated cotton swab over the necrotic tissue; the involved epithelium should come off easily, with well-defined borders leaving a clean defect. A foreign body spud can be used but could be more time consuming; however, in the case of a small focal burn, the spud is too large for the targeted tissue.

During initial management, the main goal is to support epithelial healing, which involves heavy lubrication and prophylactic antibiotic therapy. If the patient is uncomfortable or light sensitive, a cycloplegic or oral NSAID can be prescribed. If the burn is more involved (i.e., more corneal layers affected) or if the burn is located on the visual axis, an amniotic membrane may be considered.

"This patient's burn was not located in the visual axis and the affected area was superficial," Ms. Weitzel pointed out. After debridement, the patient was left with a small epithelial defect (*Figure 2*) that healed within a day. ■

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Moving in Stereo

We rely on it and rarely give it a second thought—until it's jeopardized.

By Bisant A. Labib, OD

The way humans acquire and perceive visual input is a complex and fascinating subject. Having two forward-facing eyes separated by a small distance allows each eye to have a slightly different view of the world. Known as binocular disparity, it is the basis of stereoscopic viewing, or depth perception.[1,2]

Stereopsis is the result of higher-order visual processing that allows three-dimensional data to be extracted through the comparison of these slightly dissimilar retinal images observed from different vantage points.[1,3,4] It allows humans to gauge spatial relationships and is crucial in many task that require depth perception, such as driving, sports, visually guided hand movement, motor control and viewing three-dimensional movies.[5] Amblyopia is the most common condition that may affect stereoscopic depth perception under ordinary binocular viewing conditions.[5]

Amblyopia, a neuro-developmental disorder that manifests within the first three years of life (i.e., during neural plasticity), results from early abnormal visual experiences such as strabismus and refractive errors.[5] This condition requires early diagnosis and intervention to preserve the quality of vision.

Recent studies have evaluated the possibility of treatment past the critical period to recover vision and stereoacuity. While researchers previously thought that patients over the age of seven will not benefit from treatment, recent studies show some promise in the induction of plasticity even beyond this critical timing.[5]

Eye care practitioner must be able to accurately diagnose patients with amblyopia as early as possible to ensure effective and time-sensitive treatment. Since amblyopia is an abnormality of neurons on a cortical level, the clinician must rely on identifying the presence and timing of amblyogenic factors as well as ruling out contributory ocular pathology. Stereopsis testing is a helpful tool in these instances.

Greater attention to stereo vision testing can elicit pathology and help guide the course of corrective lens wear.

Table 1. Stereoacuity Testing Methods

Test	Method	Disparity Range
Lang I	Random dot technique	1200 – 550 seconds of arc
Lang II	Random dot technique	600 – 200 seconds of arc
Titmus	Cross-polarized filters	800 – 100 seconds of arc and 3000 seconds of arc
TNO	Red/Green filters	480 – 15 seconds of arc
Randot	Polarized vectographs	500 – 20 seconds of arc

Sizing up the Options

Stereopsis in amblyopes correlates with the degree of reduction in visual acuity. It is also more diminished in patients with monocular rather than binocular blur, and in patients whose amblyopia is strabismic instead of anisometropic in origin.[2,5] Many commercial in-office tests for stereopsis are available.

Lang I: This test card contains illustrations of a cat, star and car, each with

varying levels of disparity. The test uses random dot and cylindrical gratings without the use of filters. The disparities range from 1,200 to 550 seconds of arc.[6]

Lang II: This test is similar to Lang I but contains illustrations of an elephant, car and moon. The range of disparity falls between 600 and 200 seconds of arc.[6]

Titmus: Unlike the aforementioned tests, the Titmus stereo test requires the use of cross-polarized filters and consists of identifying the elevated circle or animal in a set. The disparities range from 800 to 100 seconds of arc.[6] Part of the test includes the Wirt fly, which represents the largest level of disparity available in a commercial test at 3,000.[7]

TNO: Patients use red/green filters for dissociation and are required to identify hidden objects in a series of plates. This also employs random dot techniques.[6,7]

Randot: Polarized vectographs are used here to present different images to each eye. The difference is not noticeable to the test taker since humans are not able to appreciate light polarization. As a result, both images appear identical, except for the disparity.[5,8]

The use of monocular cues is an important consideration in the administration and interpretation of these tests, and can result in false positive stereo results. Monocular patients without stereovision can elicit information on the depth of an object using methods such as recognizing the smaller visual angle an object would subtend, the further away it is in space.[1] When implementing the use of chairside stereo tests, clinicians should remember that some shifts in the contours of Randot circles, for example, are visible under monocular viewing conditions.[8]

Overall, stereo testing is a readily available, quick and cost-effective method to identify and quantify the level of binocularity in a patient. These tests offer valuable information for the diagnosis of amblyopia, as well as the progress of treatment, given that visual acuity and stereovision are directly correlated. Early identification is crucial in the preservation of vision in these patients, and this is a quick and helpful screening tool that we should often consider. ■

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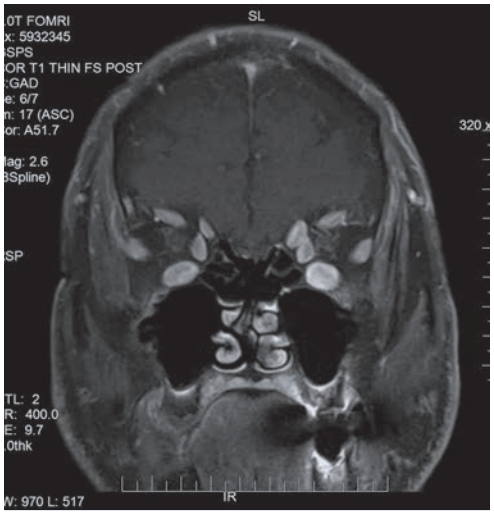
Finding Time to Decompress

Consider all possible treatments when dealing with thyroid eye disease.

By Michael Trottini, OD, and Michael DelGiodice, OD

A 70-year-old Caucasian male presented with an acute onset of diplopia, which he noted for the past two weeks. He described the diplopia as constant, vertical and worse when looking to the left. His medical history was remarkable for hypertension, high cholesterol, GERD and a history of prostate cancer, which was in remission for a number of years. Other than the new onset of double vision, he felt to be in very good heath with good control of his vascular issues and no other systemic symptoms.



MRI of the orbits showed significant enlargement of the extraocular muscles.

Evaluation

On examination, his best-corrected visual acuity was 20/25 in the right and left eyes. His extraocular muscle movements appeared full with no restriction. There was no afferent pupillary defect noted. His intraocular pressures were normal at 12mm Hg OD and 14mm Hg OS. Cover testing showed a right hypertropia worse on left gaze and right head tilt. His retinal exam was remarkable only for two small choroidal nevi, which had been noted on prior exams and were stable.

The pattern of his cover test was consistent with a right fourth nerve palsy. Given his age, the most likely cause of a fourth nerve palsy is microvascular; however, due to his prior history of prostate cancer, an MRI of his brain was obtained, which was normal. Although the MRI was of his brain, his orbits and extraocular muscles were also well visualized and looked normal. Over the next two months, his diplopia continued to worsen, and his right hypertropia worsened significantly.

Although the cover test findings were consistent with a fourth nerve palsy, the progression was not typical for a microvascular nerve palsy. Subsequent testing included brain MRA, myasthenia labs, ESR and CRP, all of which were normal. A neuro-ophthalmology second opinion agreed with the fourth nerve palsy diagnosis, but with no definitive etiology. The neuro-ophthalmologist was considering seronegative myasthenia gravis mimicking a fourth. As the patient's presentation didn't seem consistent with myasthenia, a brain MRI was reordered, as well as an orbital MRI, given his continued progression of the hypertropia.

The Path to Orbital Radiation

The follow-up MRI of his brain was still normal, but the orbit MRI now showed significant enlargement of his extraocular muscles with sparring of the tendinous insertions. These findings were highly suspicious for thyroid associated orbitopathy, which was micmicking a fourth nerve palsy, and he was sent for thyroid labs, which showed an extremely low TSH and elevated TSI, thyroglobulin and thyroperoxidase.

Endocrinology diagnosed him with Grave's disease and started the patient on prednisone and methimazole. His thyroid levels began to normalize; however, in spite of being on oral prednisone, his hypertropia and diplopia continued to worsen. He now showed moderate muscle restriction.

His orbitopathy was unresponsive to oral prednisone, so he was referred to oculoplastiscs for further management. The patient was treated with intraorbital steroid injections that didn't have any effect so it was recommended that he have orbital radiation. He then received a total of 20Gy distributed over a two-week period, at which point seemed to stop the progression of his hypertropia and restriction.

It wasn't until approximately one year after his diagnosis that his findings had stabilized, and he

underwent muscle surgery to correct the residual ocular deviation. Fortunately, the surgery had completely resolved his double vision, and he has done well since.

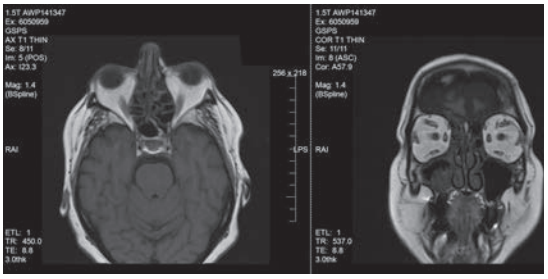
Discussion

Thyroid associated orbitopathy (TAO) is an immune-mediated inflammatory disorder that produces expansion of the extraocular muscles and fat within the orbit.[1] Generally, you'll see one of two types of TOA presentations. The first is fat-centric thyroid eye disease, where patients develop fat expansion, eyelid retraction and proptosis.[2] This occurs in two-thirds of patients with TAO and typically progresses slowly and occurs in a younger, female population.[2] The second type is muscle-centric thyroid eye disease, which was seen in our patient and causes enlargement of the extraocular muscles.[2] This can lead to restricted ocular motilities and compression of the optic nerve at the orbital apex, which occurs in one-third of patients with TAO, typically progresses rapidly and occurs in a more balanced gender distribution.[2]

We will focus on management of muscle-centric TAO. This presentation will often follow an active phase that generally lasts anywhere from six to 18 months, followed by an inactive phase.[2] During this active phase, treatment is initiated to help minimize the ocular complications of the inflammatory cascade. The major complications of muscle-centric TAO are diplopia as well as the possibility of vision loss due to optic nerve compression. There are various treatments one can initiate; however, depending on the severity, treatment doesn't always reverse the orbitopathy but helps to achieve stabilization until it reaches an inactive phase, as seen in our patient.

Treatment

Corticosteroids are usually the first-line treatment; however, there is no general consensus on the optimum dose, interval, route of administration or duration of treatment.[3] Oral, IV and local/intraorbital steroids are all used for treatment of TAO.[3] Additionally, steroid-sparing agents, such as cyclosporine and rituximab, are being used either alone or in conjunction with corticosteroids.[3]



Patient with fat-centric TAO. Note the fat expansion and proptosis.

In patients who are either intolerant of steroid therapy, have reached a cumulative dose of more than eight grams of steroids or are inadequately controlled by steroid therapy, orbital radiation is another option.[4] Generally, a total of 20Gy in 10 fractions over two weeks is recommended.[3-5] Lastly, in patients who develop optic nerve compression, orbital decompression is used as an urgent intervention.

The goal of orbital decompression is expand the orbital volume to decrease the pressure within the orbit. The orbital expansion will relieve pressure on the optic nerve and restore function in cases of compressive optic neuropathy. Orbital decompression in the setting of compressive optic neuropathy must concentrate on bony removal of the orbital apex, where the optic nerve is most susceptible to compression.[3,6]

Except in the case of compressive optic neuropathy, surgical intervention for strabismus or eyelid retraction should be delayed until the inflammatory component of the orbitopathy has resolved and been stable for at least six months.

Our patient was challenging for various reasons, including his elusive presentation and difficulty in diagnosis TAO as well as his poor response to treatment. TAO can mimic other ocular motility disorders, so the clinician should be aware and consider this in atypical presentations. As mentioned previously, more severe cases of TAO can be unresponsive to certain treatments, and the goal at times is to try and stabilize the orbitopathy until it reaches an inactive phase.

Because diplopia from TAO can significantly impact a patient's quality of life, all possible treatments should be considered to minimize this, as well as to help prevent further complications of compressive optic neuropathy. If stable and there is still double vision and muscle restriction present, patients can elect to undergo muscle surgery to help correct the deviation. Recurrences of TAO are not common (around 10%), yet patients should be continuously monitored for changes.[2] ■

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As Soon as You've Got It, Things Change

The good news: 2019 will bring policy changes that will benefit both you and your patients. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

With the New Year, changes come to Evaluation & Management (E&M) coding this year and beyond. With simplification to Medicare policies regarding E&M service, we will see a few of these implemented beginning January 1, 2019.

Coding E&M services has always been confusing, and the amount of documentation required to support it degrades the patient experience. With that in mind, the 2019 Medicare Physician Fee Schedule Final Rule cemented some very material and important changes are going to be happening between January 1, 2019 and January 1, 2021.[1]

What to Know Today

• Elimination of the requirement to document the medical necessity of a home visit in lieu of an office visit;

• For established patient office/outpatient visits, when relevant information is already contained in the medical record, practitioners may choose to focus their documentation on what has changed since the last visit, or on pertinent items that have not changed, and need not re-record the defined list of required elements if there is evidence that the practitioner reviewed the previous information and updated it as needed. Practitioners should still review prior data, update as necessary and indicate in the medical record that they have done so;

• For E&M office/outpatient visits, for new and established patients for visits, practitioners need not re-enter in the medical record information on the patient's chief complaint and history that has already been entered by ancillary staff or the beneficiary. The practitioner may simply indicate in the medical record that he or she reviewed and verified this information; and

• Removal of potentially duplicative requirements for notations in medical records that may have previously been included in the medical records by residents or other members of the medical team for E&M visits furnished by teaching physicians.

These changes reduce some of the minutia that did not enhance patient outcomes. Removing the obligation to re-record duplicative information should improve efficiencies and enhance compliance.

More Coming Up

The Centers for Medicare & Medicaid Services will continue to reduce burden by implementing of payment, coding and other documentation changes. Payment for E&M office/outpatient visits will be simplified, and payment will vary, primarily based on attributes that do not require separate, complex documentation.

Here are the policies that CMS is currently finalizing to be implemented in January 2021:

• Reduction in the payment variation for E&M office/outpatient visit levels by paying a single rate for E&M office/outpatient visit Levels 2 through 4 for established and new patients while maintaining the payment rate for E&M office/outpatient visit Level 5 in order to better account for the care and needs of complex patients;

• Permitting practitioners to choose to document E&M office/outpatient Level 2 through 5 visits using medical decision-making or time instead of applying the current 1995 or 1997 E&M documentation guidelines, or alternatively practitioners could continue using the current framework;

• For E&M office/outpatient visits, for new and established patients for visits, practitioners need not re-enter in the medical record information on the patient's chief complaint and history that has already been entered by ancillary staff or the beneficiary. The practitioner may simply indicate in the medical record that he or she reviewed and verified this information; and

• When time is used to document, practitioners will document the medical necessity of the visit and that the billing practitioner personally spent the required amount of time face-to-face with the beneficiary.

All of these changes allow for a better patient experience and reduce your documentation burden. It is critical that you and your staff keep up with these changes as they ensure that your practice is in step with federal guidelines, oh and did I say Happy New Year? ■

Send questions and comments to rocodingconnection@gmail.com.

1. Centers for Medicare & Medicaid Services. Final policy, payment and quality provisions changes to the Medicare Physician Fee Schedule for calendar year 2019. www.cms.gov/newsroom/fact-sheets/final-policy-payment-and-quality-provisions-changes-medicare-physician-fee-schedule-calendar-year. Published November 1, 2018. Accessed December 11, 2018.

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Bring Myopia Management to the Foreground

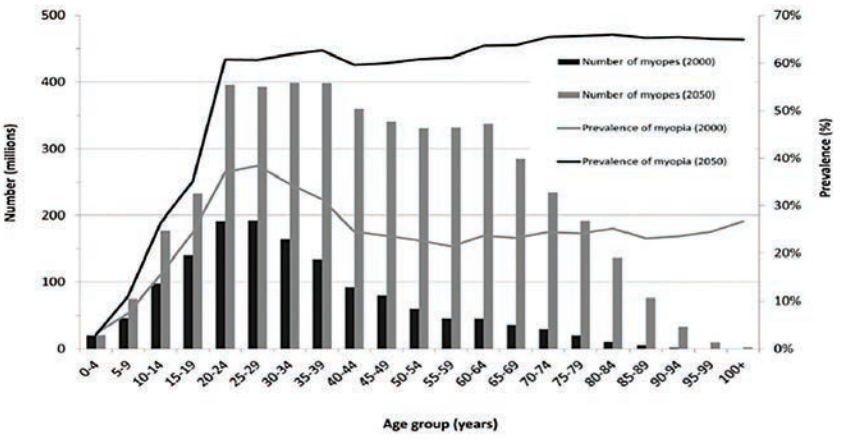
The time to address this growing phenomenon is now. Let's start with the basics.

By Kevin Chan, OD, MS

Optometrists are no strangers to myopia. In fact, maybe we have become too comfortable with treating this condition, flipping on autopilot mode when prescribing glasses and contacts. It is important to realize that passively "correcting" myopia is simply not enough, especially because we are uniquely positioned and have the tools to proactively prevent, detect and treat it.

Due to its widespread impact—the global prevalence of myopia has grown by 66% in the past three decades, and it has been estimated that nearly half of the world's population will be myopic by 2050—myopia has secured a place in the spotlight as a public health issue.[1,2] In the United States alone, the prevalence of childhood myopia has increased from 25% to 44% between 1972 and 2004.[3,4] As the number of young patients affected by childhood myopia increases, the likelihood of developing high myopia with significant retinal complications later on in life also increases.

With myopia on the rise, we cannot afford to turn a blind eye or do the bare minimum when caring for patients whose lives we have the ability to change. We must emphasize early detection and disease progression control, which should be at the cornerstone of myopia management, and be prepared to treat this population. The following article covers the basics practitioners should consider on their quest to learn more about and manage myopia.



The global prevalence of myopia has grown by 66% in the past three decades, and it has been estimated that nearly half of the world's population will be myopic by 2050.[1,2]

1. Educate Yourself

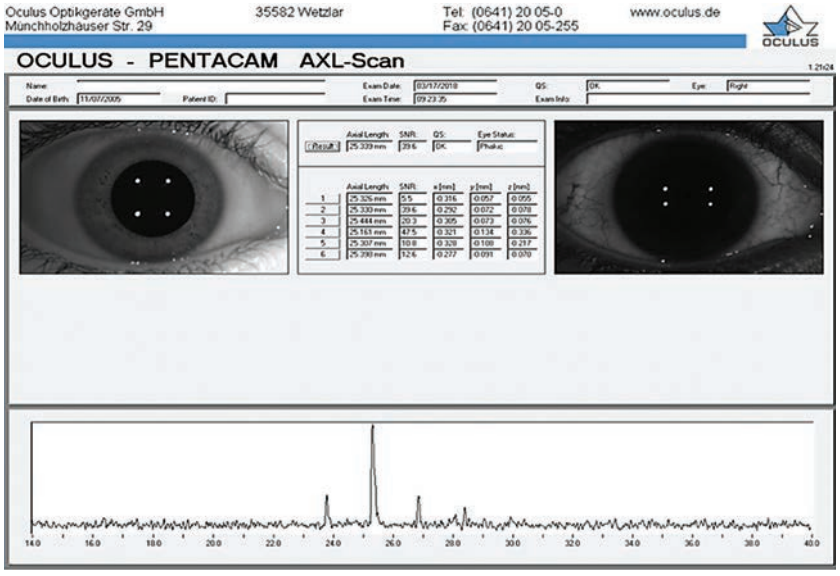
'Why should I practice myopia management?' is one of the most important questions for practitioners to reflect on. Before attempting to answer it, you must first understand what myopia management means. Identifying and diagnosing myopia is generally easy and straightforward. Knowing how to prevent myopia from progressing, however, proves to be harder; it involves clinical expertise and patient compliance.

As you delve into the core of myopia management, be prepared to step out of the 'phoropter zone' and start thinking beyond the fovea. Numerous animal studies have shown that the peripheral retina—not the fovea—plays an integral part in axial length (AL) growth.[5-7] With that being said, this finding does not come without controversies. Currently, a foolproof theory or formula that can completely 'control' myopia does not exist. It is worth noting that, at the moment, there are no FDA-approved treatments for myopia management. Practitioners should be forthright when discussing this with patients and their parents. Rather than making absolute claims and asserting that treatment will prevent myopia from worsening, which is impossible anyway, practitioners must manage myopia based on each patient's myopia profile to render more promising prognoses and treatment outcomes.

When managing myopia, it is important that practitioners set a clear goal and have a focused mindset to develop treatment plans for young patients best suited under the circumstances. Currently, there are three evidence-based approaches that are commonly used for myopia management—orthokeratology (ortho-k), soft multifocal contact lenses and topical compounded atropine. Each modality offers unique benefits and challenges.

Ortho-k is generally geared toward young, athletic patients who show little to no contraindications or corneal irregularities, such as keratoconus. Capturing good quality baseline corneal topography is vital.

Soft multifocal contact lenses with custom designs are typically catered toward patients with high myopia and atypical astigmatism, for which ortho-k may not be able to provide satisfactory uncorrected vision during the day, or those who do not adapt well to rigid gas permeable lenses at night.



The Pentacam Scheimpflug imaging system is an important tool used to analyze the anterior segment and measure AL to help guide myopia management.

Topical compounded atropine is generally used for young patients who manifest atypical, rapid myopia progression and a strong inheritance of myopia and are not compliant with contact lens wear. Aside from the technical expertise necessary to fit contact lenses, myopia management is unique in that it combines technical and behavioral approaches to address the distinct myopia profile of each patient.

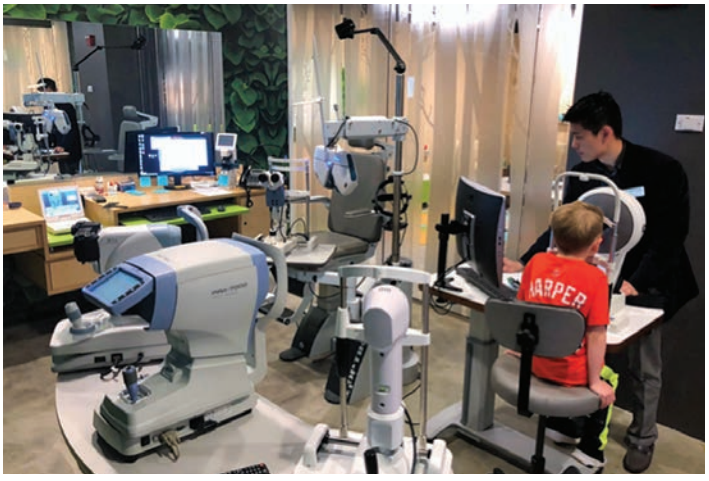
While dabbling in myopia management helps you get your feet wet, unfortunately it does not get you very far. Specializing in myopia management requires practitioners devote significant amounts of time and effort, refine other areas of their practices to move myopia management to the forefront and allocate staff and resources.

2. Stock Your Practice

The process begins with acquiring the resources necessary to manage myopia, starting with tools and technologies. Managing myopia involves using more just a phoropter. Having a corneal topographer is fundamental and helps practitioners understand patients' corneal health and determine which management strategy is best suited under the circumstances. For example, using corneal topography to learn more about a patient's corneal elevation differentials, such as the relationship between sagittal height differentials (SHDs) and corneal toricity, would help guide lens design if corneal reshaping or ortho-k were being considered as treatment modalities. A recent study showed that a higher corneal toricity is more likely to result in lens decentration.[8,9] As a result, custom lens parameters are designed to optimize lens centration. Additional studies have found that toric ortho-k lenses are preferred to minimize lens decentration when SHDs exceed 30µm at a chord length of 8mm.[9,10]

Measuring AL has become the foundation of myopia management. Many sophisticated technologies—such as the IOLMaster (Zeiss), one

of the most popular instruments for obtaining AL measurements—are now able to detect microscopic physiologic variations that often precede subjective dioptric changes manifested by phoropters. Currently, the sonographic A-scan and the non-contact automatic Pentacam corneal tomographer are among the more commonly used pieces of equipment. Both have their advantages and shortcomings. The A-scan is generally light and portable, making it easier to transport between offices, and more affordable. However, operating the A-scan requires direct tactile contact using an ultrasound probe on the cornea under topical anesthesia, steady fixation and proper posture. Using this device while working with children may prove to be difficult; putting a probe onto their eyes can be intimidating and technically challenging.



This child is undergoing Pentacam Scheimpflug imaging.

The Pentacam, on the other hand, uses a high-resolution rotating Scheimpflug imaging system to capture panoramic views of the cornea and provide a precise anterior segment analysis and elevation maps of the anterior and posterior corneal surface and thickness. The newest version of the Pentacam incorporates AL measurement technology, is precise and does not involve tactile contact with the cornea or topical anesthesia. All in all, it is generally more practical and favorable when working with children.

3. Prepare Staff

As you are building your myopia management armamentarium, you should be allocating and educating your staff and determining an action plan. It is worth delegating tasks and designating a few technicians and staff members to a 'core staff team' that has more in-depth education and training and can take on more responsibilities to alleviate the burden on you and enhance the patient experience.

Patient consultations may take an hour to obtain in-depth information to construct effective treatment plans but vary on a case-by-case basis and should not be rushed or associated with a time restraint. To make matters easier, myopia patients should be scheduled on the same day.

In my experience with post-treatment care for myopia management, patients who receive the overnight ortho-k modality are generally seen for monitoring a day after, a week after and a month after treatment, at which point they are seen on a quarterly basis for the remainder of the first year of treatment. Once stable, I follow up with patients on a quarterly or biannual basis depending on the situation. If treated with topical compounded atropine, patients are typically monitored for near vision quality and photophobia issues in quarterly intervals.

Unlike adults, young patients may not always be able to comfortably or effectively express their experience clearly and accurately. Be on the lookout for clues of discomfort or dissatisfaction, which are usually reflected via nonverbal cues and body language. A recent cognitive study found that nonverbal, communicative eye contact yields anticipatory cooperation and commitment in children.[11] This patient population requires more than your typical 'five-minute-check-and-go' follow-up and may greatly benefit from additional face-to-face time with you, the practitioner, to establish a sense of familiarity, comfort and rapport. Practitioners and staff alike need to be prepared to exercise patience and work with this unpredictable demographic—perhaps by implementing an incentive or reward program—otherwise, young patients may feel neglected or misunderstood, which may result in compliance issues and negative visual outcomes.

4. Initiate Patient Acquisition Marketing Efforts

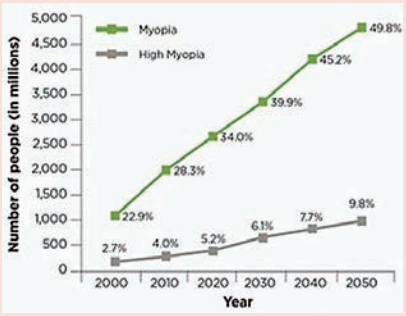
As you equip your practice with educated staff members and appropriate tools, you should begin mapping out and executing your marketing plan. As with raising awareness about anything, digital marketing is a great way to introduce the myopia management services your practice is offering. According to recent statistics, approximately 74% of Internet users actively engage in social media, 80% of who specifically search for medical information and resources.[12] Nevertheless, only 26% of medical professionals in large corporations

The Future is Looking Myopic

By Catherine Manthorp, Associate Editor

Myopia was previously thought to be a simple refractive condition correctable by glasses and contact lenses with limited visual consequences. We know now this is simply not the case; the condition is increasingly associated with a heightened risk of permanent vision impairment.[1]

A groundbreaking 2016 study by the Brien Holden Vision Institute on the global prevalence of myopia and high myopia since 1995 found that myopia affected 23% of the world's population, or 1.5 billion people, in 2000.[2] Using the data they obtained to predict what the future prevalence of myopia looks like, the team cautioned that half of the global population—that's five billion people—could be affected by 2050.[2] To make matters worse, while only 3%, or 163 million people, had high myopia in 2000, this percentage is expected to increase to 10% by 2050.[2] That means one billion people could be at risk of permanent vision impairment and blindness, which would make myopia the leading cause of blindness worldwide.[2]



Estimated global prevalence of myopia and high myopia, 2000 to 2050.[2]

Luckily, the researchers narrow in on the problem by shedding light on what is causing this shift, which demographic is experiencing the greatest impact and what can be done about it. Environmental influences and lifestyle changes—such as reduced time outdoors, increased near activities, higher-pressure educational systems and greater electronic device usage—are two of the biggest factors at play.[3] In 2000, myopia was occurring for the most part in patients younger than 40, indicating that these factors mainly affected this demographic.[2]

Moving forward, ECPs must better understand the risk factors associated with myopia onset and progression by regularly monitoring population trends and characteristics.[1] In terms of treatment, ECPs have several routes they can take, including optical interventions, such as bifocal glasses, multifocal soft contact lenses and ortho-K to slow axial elongation, behavioral strategies, including reduced near work and more time outdoors, and pharmacological agents like low-dose atropine.[1]

If practitioners don't assert control, myopia's inexorable growth could eventually leave most patients either affected directly or at least indirectly feeling its consequences through the effects on a family member.

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or hospitals in the United States use social media as a tool to market their medical services.[12] Clearly, consumers have an appetite for social media, giving us a great opportunity to give them a platform catered toward myopia management.

The success of any marketing campaign depends on how well the audience is targeted. Because myopia management services are primarily geared toward children, it is the baby boomers and the Gen X'ers—the parents (decision-makers) of most of today's myopic kids—that we need to reach. Volunteering to speak at local community events and provide vision screening in schools and pediatricians' offices are good ways to increase public awareness of early preventative eye care and your unique service for children.

Perhaps most importantly, and free of cost to you, you should consider focusing your efforts within your own practice through internal marketing. You will probably find that your patients, many of whom may be young myopes, are your best source of marketing. Raising awareness of the additional services you offer through discussions or handouts leads to word-of-mouth referrals and increases traffic flow to greatly benefit your practice. You can also look toward your colleagues who have myopic children because treating local physicians' kids for myopia management allows you to provide a first-hand experience of your services to medical professionals who can vouch for you when making referrals.

5. Consult and Counsel

After acquiring patients, but before starting myopia management, comes the consultation stage. A thorough myopia consultation typically involves obtaining a detailed case history, which includes gender, ethnicity, age at myopia diagnosis, age at myopia correction, average rate of myopia progression, ocular history and congenital medical conditions. It is also worth taking the time to understand your patient's lifestyle to help facilitate patient education and guide the treatment process.

Based on a patient's history and lifestyle, practitioners can determine who is at risk of myopia progression. Ethnicity plays a large and independent role in the development and progression of myopia. In particular, children of Asian and Southeastern descent are inheritably at a higher risk of early onset of myopia.[13,14] Moreover, several studies have

shown that increasing the amount of near work activities, especially on digital devices, and reducing the amount of time spent outdoors are associated with increased odds of myopia development.[15-22]

Consultation does not end after learning about your patient's background. Many parents are not properly educated about myopia in terms of its risk factors, signs and increasing prevalence and the implications of higher degrees of myopia. While parents acknowledge their children have vision problems, they have yet to associate worsening vision with greater risks of retinal and other eye health complications and understand how to address these issues. As a result, proper and timely intervention for childhood myopia may not occur.

An observational study reported that 55% of participants had never taken notes during a medical appointment, and 41% were reluctant to disclose their medical concerns to their physicians because they felt rushed during their consultation.[23] Therefore, it is crucial to allow 20 to 30 minutes for parents to engage in Q&A sessions and discuss realistic goals and expectations. It is also vital that clinicians carefully delineate the relationship between refractive changes and eye health consequences to help patients and their parents better understand and appreciate the long-term ocular health benefits of myopia management.

6. Discuss the Finances

It is important to discuss the costs of this condition, which may not be on most parents' radar, with already anxious parents who may be trying to decide how they are going to pay for your services or if they are even worth it in the first place.

"Is this covered by insurance?" is one of the questions parents most frequently ask. Currently, myopia management is generally uncovered and is 'elective.' It is also not 'medically necessary' as per the current standard of care. Criteria that qualify as 'medically necessary' include corneal irregularities (e.g., keratoconus and post-surgical corneal complications), high myopia (-10D) and hyperopia (+10D) and aphakia.[24] Contact lenses are usually covered when vision correction by custom lenses yields far superior visual outcomes than conventional spectacles. While conventional contact lenses may be covered by most vision plans, lenses for myopia management are not.

There are, however, other options. flex spending accounts and health spending accounts are generally accepted. Third-party healthcare finance companies can be used as well. Regardless of insurance coverage, it is critical to have candid discussions with parents regarding their financial situations without overwhelming them. Should they decline to proceed with myopia management, parents must be aware of the cumulative costs of conventional spectacles or contact lenses both financially and medically.

You cannot afford to take any shortcuts when developing a first-class myopia management practice and making it your own. Given the growing prevalence of myopia, you must be prepared to work with patients who are interested in enlisting your services and offer them the best care possible through the resources, effort and time you allot. While it has its challenges, managing myopia is also extremely rewarding, sets your practice apart from others and allows you to use your passion and expertise to change the lives and vision of your patients. ■

Dr. Chan is the director of Treehouse Eyes in Tysons Corner, VA, and a fellow of the American Academy of Optometry. He graduated with a doctorate in optometry and a masters in vision sciences from the New England College of Optometry.

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Myopia Education Efforts Online and Abroad

By Mark De Leon, Associate Editor

Myopia may be pervasive, but so are efforts to spread awareness and inform ways to reduce progression. Resources abound.

Essilor has taken big steps in furthering the conversation about myopia on a global scale, with a longstanding commitment to education and intervention, including assistance in launching the first National Myopia Management Convention in Singapore last June.[1] Stateside, Essilor spread myopia awareness last fall with a movie trailer that screened in select theaters nationwide, to inspire parents to be vigilant for warning signs in their children and schedule a comprehensive eye exam.[2] Clinicians can also download a variety of educational tools at essilorshare.com/myopia.

Early this month, the company announced the formation of a Myopia Taskforce comprised of 14 experts "who have pledged their commitment to establishing a preferred method for comprehensive myopia care, including developing a universally accepted care protocol for managing myopia," according to *Vision Monday*.[3] It's part of Essilor's Myopia Initiative in Action, which will be rolling out this year.

In November, **Johnson & Johnson Vision** teamed up with experts, also in Singapore, on a research collaboration to tackle myopia.[4] The aim is to create tools to identify those at risk to develop high myopia, learn about the underlying mechanisms of myopia and work toward developing better treatments.

The **Brien Holden Vision Institute** (BHVI) recently launched its online Global Myopia Centre, which includes numerous free online tools, such as BHVI's Myopia Calculator, Guidelines for Myopia Management and online courses that provide accredited training for practitioners in myopia interventions.[5] You can access them here: www.globalmyopiacentre.org.

At the 2018 Academy of Optometry meeting, the **International Myopia Institute** presented seven white papers on areas such as myopia definitions, evidence for interventions, ethical considerations, clinical trial guidelines and clinical management guidelines.[6] The reports will be available at the beginning of this year in Investigative Ophthalmology and Visual Science.

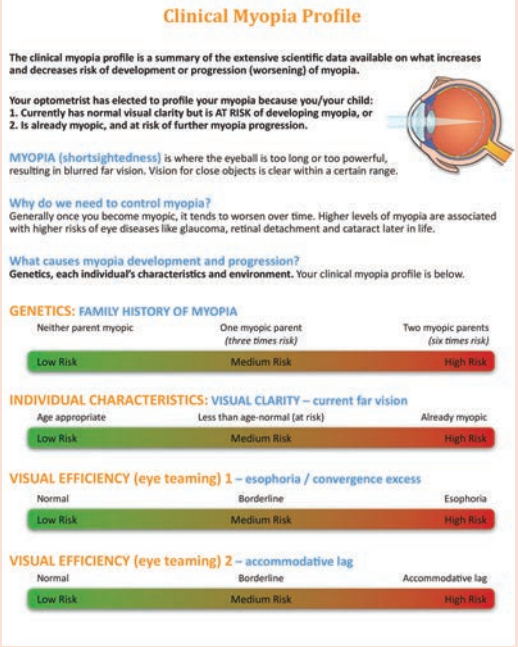
Kate Gifford, PhD, chair of the institute's clinical management committee, has been managing myopia for over 15 years. With her husband, Paul Gifford, PhD, she developed various patient communication resources, including the Myopia Profile tool, available at myopiaprofile.com. Dr. Gifford uses the profile tool whenever she examines a child who is already myopic or shows risk of developing the condition. The duo has also developed My Kid's Vision (www.mykidsvision.org) as another information resource to help parents understand myopia and the steps they can take toward prevention and the slowing of progression.

Visioneering Technologies, which makes the NaturalVue contact lens for myopia control, supported the development of www.managemyopia.org, a website for practitioners looking to better address the needs of myopic patients, which includes screening tools and other resources.

CooperVision offers the MiSight contact lens for myopia control outside the US and is studying it in ongoing trials. The company provides an educational summary for the public with advice on how parents can keep myopia at bay (coopervision.com/blog/three-ways-to-slow-down-myopia) and advice for clinicians as well (coopervision.com/practitioner/clinical-resources/myopia-control-in-youth).

Industry has also been raising awareness of the link between digital screen use and myopia, as well as dry eye. In June, **Shire** launched Screen Responsibly (www.myeyelove.com/screen-responsibly), a consumer-friendly resource to ensure a healthy screen routine.

It doesn't always take an entire industry to generate discourse among an engaged community of optometrists. It just takes commitment and conversation. Each OD can make meaningful differences in patients' lives with a little more attention and emphasis for those at risk.



Dr. Gifford's Myopia Profile tool helps practitioners explain to the child and their parents the outcomes of the exam more effectively.

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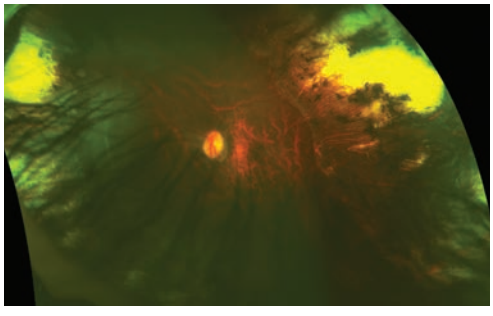
HOW ENVIRONMENT AND GENETICS GIVE RISE TO MYOPIA

More than half of the world's population will be myopic by 2050. What factors are causing this epidemic? **By Erin Tomiyama, OD, and Kathryn Richdale, OD, PhD**

Most parents think genetics are the only reason for their child's nearsightedness. But more and more children with only one myopic parent, or even no myopic parents, are becoming nearsighted. Clearly, more than genetics is involved in the myopia epidemic.

This article reviews the current understanding of both genetic and environmental associations of myopia, and discusses how to educate parents about why more



This fundus photo shows a patient with high myopia, a condition with both genetic and environmental contributors.

Release Date: January 15, 2019
Expiration Date: January 15, 2022
Estimated time to complete activity: 2 hours

Jointly provided by Postgraduate Institute for Medicine and RGVCE

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

- Interpret the worldwide increase in myopia prevalence from an epidemiologic perspective.
- Discuss the normal pathophysiologic process of emmetropization and its relation to myopization.
- Explain the likely environmental and behavioral causes for the increase in myopia prevalence.
- Use knowledge of the genetic factors associated with susceptibility for myopia and high myopia to identify and treat patients.
- Recognize how genetic causes relate to environmental factors in regard to light-induced signaling in myopia development.

Target Audience: This activity is intended for optometrists engaged in the care of patients with myopia.

Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by the Postgraduate Institute for Medicine and RGVCE. 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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Credit Statement: This course is COPE approved for 2 hours of CE credit. Course ID is **60197-PB**. Check with your local state licensing board to see if this counts toward your CE requirement for relicensure.

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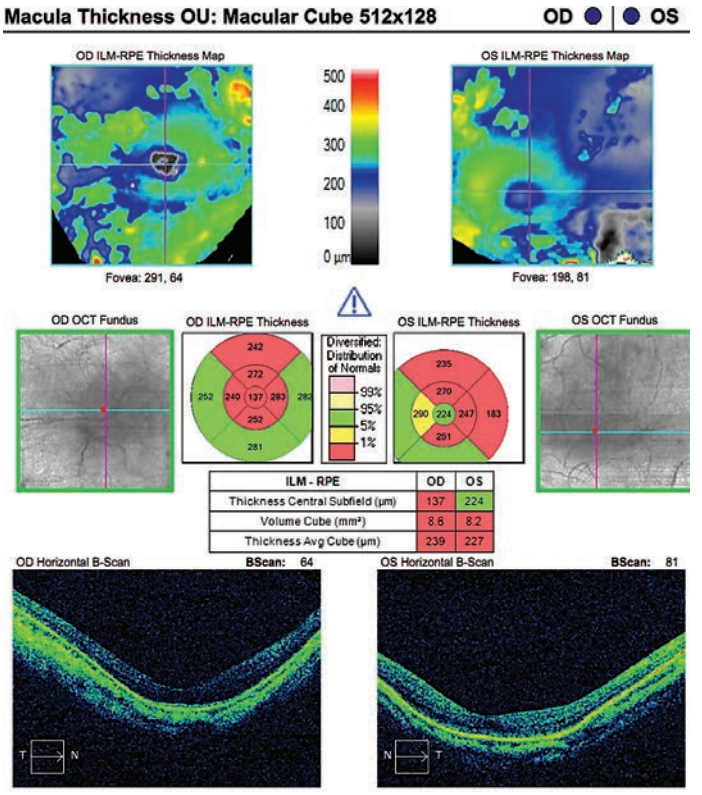
children are becoming nearsighted, and what can be done about it.

Worldwide Increase

Myopia was previously believed to be a benign error of refractive power. But as more children become nearsighted, myopia is being recognized as a worldwide epidemic with significant health consequences. The rapid increase in prevalence of myopia and consequential risk for pathologic visual conditions, such as cataracts, glaucoma and retinal detachment, make myopia a significant concern for today's parents.

Higher levels of myopia increase the risk for ocular disease, but even low levels of myopia (-0.75 to -3.00D) are associated with three to four times greater risk of retinal detachment.[6] The definition of high myopia varies from study to study, but is usually defined as refractive error of -5.00D or -6.00D or higher.[6] Myopia greater than -6.00D increases the risk of retinal detachment by 20 to 80 times that of a non-myope.[6]

Even though high myopia is the greater threat, we must aim to decrease the amount of myopia for all patients to reduce the overall risk for ocular co-morbidities. Reducing the rate of overall progression by 50% would decrease the prevalence of all levels of myopia, and reduce high myopia by up to 90%.[1]



This OCT analysis of a patient with high myopia shows that most of the retinal nerve fiber layer is flagged as abnormal.

Emmetropization and Myopization

The normal pathophysiologic process of emmetropization is such that the eye progresses from hypermetropia in the first years of life until emmetropia is reached in mid-childhood. The goal of emmetropization is for the eye's corneal and lenticular refractions to match the increasing axial length of the eye during growth. In myopia, the process overshoots emmetropization and results in myopic refractive error.[8,9]

Research shows myopic children may begin to experience greater axial elongation than emmetropes even three years before the actual onset of myopia.[10] In fact, the best determinant of the final refractive error for school-aged children is an age-matched comparison of current refractive state, reported researchers in the Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error (CLEERE) study.[11] This was an observational cohort study of ocular development and myopia onset of ethnically diverse children ages six to 13 years. They reported that, if a child is less hyperopic than +0.75D by first grade, the child is at an increased risk to develop myopia.[11]

We now know that hyperopic defocus (focusing light behind the retina) can stimulate axial elongation. Conversely, myopic defocus (in front of the retina) can slow axial growth.[12,13] The peripheral refraction typically varies with the central refraction in that myopes usually have relative hyperopia in the periphery and hyperopes usually have relative myopia in the periphery.[14,15]

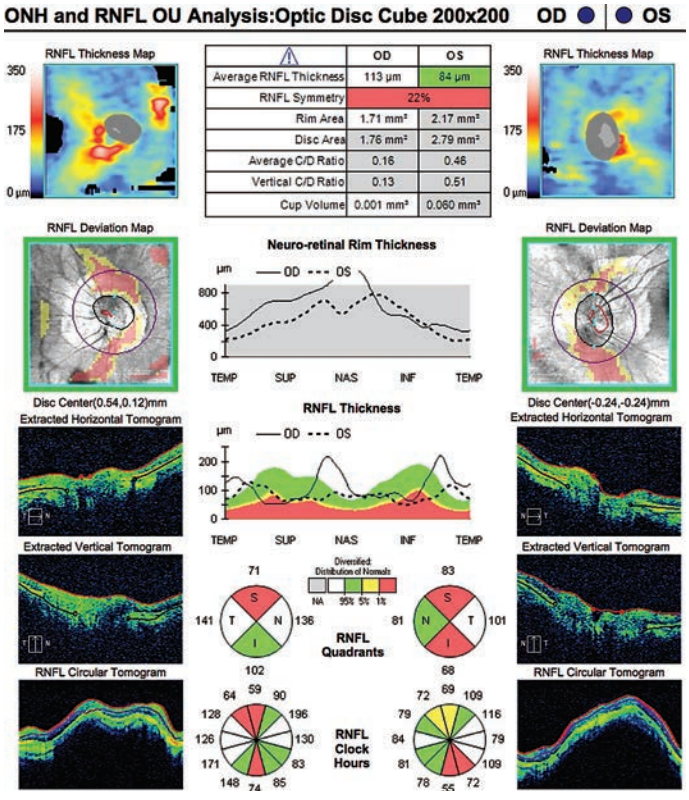
Myopia is Now a Global Public Health Issue

The increasing prevalence of myopia and related cost of vision care are now global public health issues. Currently, 1.9 billion people worldwide (27%) have been diagnosed with myopia, and this number is expected to nearly double to almost five billion people (52%) by 2050.[1] In the United States alone, the prevalence of myopia has increased from 25% in the 1970s to 44% in the early 2000s.[2]

As myopia prevalence escalates, the prevalence of uncorrected refractive error will also increase. Uncorrected refractive error is the leading cause of moderate and severe vision impairment (42%), affecting 108 million people worldwide in 2013.[3]

Not only does uncorrected refractive error affect vision and quality of life, but it also results in global loss of productivity amounting to an estimated $269 billion.[4] In Singapore, the annual cost of optical correction for myopic adults has reached $755 million.[5]

Soft multifocal lenses and orthokeratology lenses are used for myopia control as they shift the peripheral hyperopic defocus to myopic defocus by moving the peripheral focus forward. Corneal topography maps can be used to demonstrate the peripheral plus power that is created with either a soft multifocal lens worn on the eye or after orthokeratology treatment. Multiple studies have now reported good safety and efficacy with these treatment methods.



This OCT crosssection reveals a posterior staphyloma—a hallmark sign of pathologic myopia.

Environmental and Behavioral Influences

The rapid increase in the prevalence of myopia within the past few decades suggests the involvement of environmental causes, especially when we see such dramatic increases among specific populations and in certain regions. Given this increase in such a short time period, geographic and racial/ethnic differences cannot fully explain the sudden rise in myopia. Ultimately, genetic susceptibility and environmental changes have likely worked in combination to produce the greater prevalence of myopia observed today.[1]

Research shows that the amount of time a child spends outdoors is directly related to the odds of the child developing myopia.[16] While time outdoors is certainly a factor, it's not certain what aspect of outdoor time directly decreases the risk—could it be the higher light levels, the spectral composition of outdoor light, or the dioptric demand of outdoor viewing?

One study found that children who spent sufficient time outdoors, quantified as more than two hours per day, decreased their risk of myopia even if they also performed a large amount of near work or had two myopic parents.[18] The protective effect of time outdoors against myopia was not activity-dependent. Interestingly, this study also reported that there was a greater effect of delaying myopia onset when children performed sports outdoors as compared to doing the exact same sport indoors.[18]

While there is no magic number, about two hours per day or at least 10 hours per week of outdoor time can have a positive impact for children who haven't yet developed myopia. Unfortunately, once a child becomes myopic, increasing outdoor time isn't likely to slow the progression of myopia, according to the limited evidence we have.[16]

The dioptric mapping (amount of distant vs. near stimuli) of the visual environment could help to explain why more time outdoors may delay the onset of myopia. The dioptric topography of an indoor environment is generally closer and more heterogenous than an outdoor environment, so the eyes are more likely to experience hyperopic defocus indoors.[6] Conversely, the outdoor visual environment provides a more uniform field of view on the retina with little dioptric demand.[6]

Other research suggests that the bright outdoor light triggers a release of dopamine from the retina, inhibiting signals for axial elongation.[18] Seasonal variation in the progression of myopia would support this theory, as more axial growth occurs during winter months than during summer months.[19]

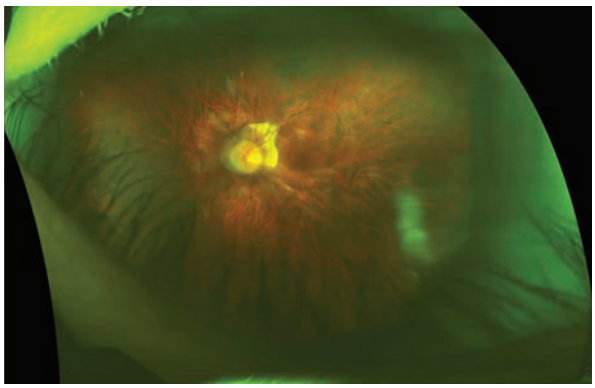
Taken together, studies done in Japan, China and the United States suggest that higher light levels, longer days and higher solar irradiation seem to be associated with slowed myopia progression.[19-21]

Many challenges to establishing a causal relationship between time outdoors and myopia development and progression exist. For instance, increased time outdoors is indirectly

When Myopia Causes Macular Degeneration

Myopic macular degeneration (MMD) is the most common cause of visual impairment in patients with myopia.[7] MMD initially develops with chorioretinal atrophy and lacquer cracks that may ultimately lead to choroidal neovascular membrane and macular atrophy.[1]

proportional to time indoors, since they are mutually exclusive. Also, children's activities performed outdoors often involve more physical movement compared with indoors activities, which tend to be more sedentary and involve closer dioptric stimuli. Lastly, many studies examining the amount of time spent outdoors are conduted by survey and are therefore subject to recall bias.[16]



While high levels of myopia have greater risk for pathologic changes, even low levels of myopia (-0.75 to -3.00D) are associated with increased risk for retinal disease.

Researchers have made efforts to use technological advancements to objectively record time spent outdoors and light levels of exposure, but depending on the placement of these devices, they may not truly quantify visual input to the eyes.[22] New devices are currently being studied to also record the real-time viewing distances.[23]

Further studies are needed to gain a better understanding of what factor, or combination of factors, make outdoor time effective in delaying the onset of myopia. For now, we can only present the facts when educating parents and children: increased time outdoors will help delay the onset of myopia, but will not likely have an effect once a child becomes myopic.

Research also shows that near work and education level can modify the risk of developing myopia. Myopia progression increases approximately 2% for every diopter of near work.[24] The mechanism behind increased near work to increased axial length includes peripheral defocus and biomechanical changes.[25] The risk of development and progression of myopia is more correlated with a closer reading distance (less than 20cm) and longer periods of continuous near work (more than 45 minutes) than total duration of near work.[26,27]

The location in which a child lives can also influence myopia risk. One meta-analysis shows that children in urban environments had 2.6 times increased risk of myopia compared with children in rural areas.[33] This finding may be related to the observation that children in urban environments may spend more time indoors with greater near work demands.

Environmental and behavioral factors appear to play important roles in the development and progression of myopia. But if we recommend modifiable behaviors that may slow the onset of myopia, we must also appreciate the important role that genetics play.

Genetic Factors

The risk of a child becoming myopic when one parent is myopic is approximately 1.5 times that for children with no myopic parents. That risk doubles to three times if both parents are myopic.

The degree of the parents' myopia also plays an important factor in the development and final refractive error of the child.[34] Parents can readily understand these relative risk factors, but, unfortunately, myopia genetics is not as straightforward as one might expect.

Go Play Outside!

A recent meta-analysis recommended that children spend 9 to 10 hours per week outdoors to reduce the risk of developing myopia.[16] This recommendation is based on the finding that the incidence of new myopia cases decreased by 50% in children who spent an additional 80 minutes outdoors per day.[17]

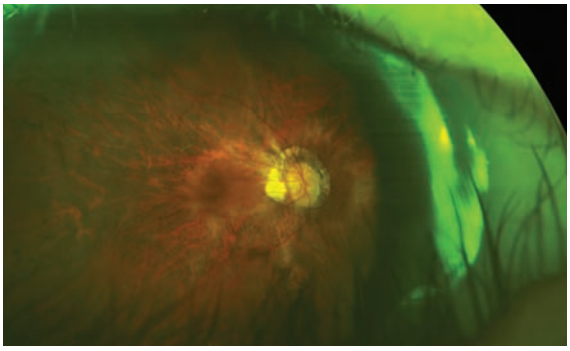
Twin studies. With the exception of rare autosomal dominant conditions such as Marfan syndrome, most "school age" myopia has a complex genetic and environmental etiology.[35] Heritability is the proportion of variation in a trait (myopia) that can be attributed to genetic factors. In fact, the first true twin study of heritability was actually conducted by a German ophthalmologist, Walter Jablonski, who identified a greater "within pair" difference in refractive error for fraternal vs. identical twins.[36]

Some of the more recent twin studies reported myopia heritability ranging from 75% to 95%; however, twin studies are known to overestimate true heritability due to model assumptions and confounding factors such as a largely shared environment.[37] Family studies can add more information about the complexity of genetic covariance and environmental factors. Large family studies have reported heritability of refractive error to be 50% to 60%, and a meta-analysis of both twin and family studies suggest a myopia heritability of about 71%.[37]

Genetic linkage studies. These use familial information to map locations on genes that may be related to a trait. The first myopia-related locus identified was MYP1, which is related to high myopia and located on the X chromosome.[38] While other loci have been identified, linkage studies

often highlight large regions of the gene, making further analysis challenging.

Candidate gene analyses have identified more than 100 potential myopia-related genes based on known biological function. For example, genes involved in ocular development and growth, as well as scleral remodeling, have been identified as potential candidate genes for myopia.[38]



High levels of myopia (-5.00D or -6.00D or greater) increase the risk for ocular disease. Note the peripapillary atrophy and macular changes in this myopic eye.

Unfortunately, the candidate gene approach is limited by current knowledge of gene function. Both linkage studies and candidate gene studies have reported a possible association between high myopia and the paired box 6 (PAX6) gene.[39,40] The PAX6 gene is considered a master gene involved in many facets of normal eye development.

Genome-wide association studies. (GWAS). This approach compares the genetic information across thousands (or tens or hundreds of thousands) of individuals to identify small differences potentially related to a specified trait. The strength of this approach is that it can explore the entire genome, and thus doesn't rely on a priori knowledge of function.

Two of the largest GWAS groups in myopia, the commercial 23andMe and the academic Consortium for Refractive Error and Myopia (CREAM), recently combined efforts to conduct a GWAS meta-analysis of more than 160,000 participants, which identified 161 potential gene loci involved in myopia.[41] Some of the loci identified include those involved in dopamine and light processing. These genetic findings provide further support for mechanisms of myopia development involving light exposure, defocus and contrast.

Despite these important findings informing potential genetic locations and mechanisms of myopia development, these studies have identified less than 5% of the variation in refractive error due to genetic variants.[42] Clearly, the dramatic rise in myopia cannot be due to genetics alone, but is likely due to a combination of both genetic susceptibility and increased environmental triggers.

Gene-environment interaction studies. To study both genetic susceptibility and increased environmental triggers, gene-environment interaction studies have been used to explore some of the more well-established links with myopia, and have found associations with educational level and near work.[42] Mendelian randomization uses genes with known functions as surrogates for environmental factors to explore the cause and effect of environmental exposure on the trait of interest. This is the technique that was used to estimate that every additional year of education was associated with a -0.27D increase in myopia.[32]

Epigenetics. The study of epigenetics has also been used to explore modifications in gene expression or gene activity due to environmental factors. Of note, one recent epigenetics study found that variants in a micro RNA (miRNA 328) modified PAX6 expression, which may be a potential target for myopia treatment.[43]

The contribution of genetics directly on the development of myopia may not be comprehensive, but one's genes may play an important role in one's susceptibility to environmental factors.[44]

Discussing 'Nature and Nurture' with Parents

When discussing myopia with parents, establish what physicians know about myopia and its progression. Parents should also understand the risks and consequences of developing myopia, specifically the risk for developing retinal holes, tears or detachments, myopic maculopathy, glaucoma and early cataracts.[6]

While myopia has a genetic component, modifiable behaviors impact the development as well as the rate of progression of myopia. Explaining these modifications can help parents understand that environment does play a role in the development of myopia and actions can be taken to help delay the onset and progression of their child's nearsightedness.

Online calculators are available to help communicate with patients and parents about the need for initiating treatment. Three primary forms of off-label treatment are available in the United States for myopia control: atropine, soft multifocal contact lenses and orthokeratology. As practitioners, it is our responsibility to

Asia Leads the Way in Myopia

Myopia is more prevalent in Asian and South East Asian countries, where they strongly emphasize education and academic achievement.[28,29] This intense educational system requires high near work demands and therefore may be correlated with the higher prevalence of myopia and faster rate of progression.[30,31]

A recent international study reported that every additional year of education was associated with a -0.27D higher level of refractive error.[32]

educate parents on what myopia is, how it develops, what causes it to progress, and how we may be able to delay its onset and slow its progression. Present all treatment options, as well as their associated risks and benefits.

We may never fully understand the exact causal relationship between our genetics and the many environmental factors related to myopia due to their tight inter-relationship and our inability to restrict natural human development. However, continued genetic and environmental research will likely shed more light on the interaction between nature and nurture, and perhaps bring this epidemic under control. ■

Dr. Tomiyama recently completed a cornea and contact lens residency at the University of Houston College of Optometry (UHCO) in Houston.

Dr. Richdale is an associate professor at UHCO.

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OSC QUIZ

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1. Which of the following is not a pathologic concern due to myopia?
a. Cataracts.
b. Glaucoma.
c. Myopic macular degeneration.
d. Optic neuritis.

2. Myopia is considered a worldwide epidemic for all of the following reasons except_____.
a. It affects more than a quarter of the population.

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OSC QUIZ

b. It affects patients' quality of life.
c. It results in losses of productivity and money.
d. It indicates children spend less time outdoors.

3. What is the goal of emmetropization?
a. The eye's corneal and lenticular refraction matches the axial length of the eye.
b. The eye does not grow longer than 24mm.
c. The corneal power is not greater 40D.
d. The lenticular power does not exceed 20D.

4. What is known to slow axial elongation?
a. Hyperopic defocus.
b. Myopic defocus.
c. Matched peripheral refraction.
d. All of the above.

5. Which myopia control treatment options employ the principle of myopic defocus?
a. Atropine, orthokeratology.
b. Soft multifocal contact lenses, atropine.
c. Orthokeratology, soft multifocal contact lenses.
d. Soft multifocal contact lenses only.

6. Which activity best delays myopia onset in children?
a. Practicing karate indoors.
b. Playing video games indoors.
c. Playing basketball outdoors.
d. Watching television indoors.

7. Which of these behavioral factors is most effective in delaying the onset of myopia?
a. Increased time outdoors.
b. Decreased near work.
c. Living in an urban environment.
d. Modifying the PAX6 gene.

8. In one study, the incidence of new myopia cases decreased by 50% in children who spent about how much additional time per day outdoors?
a. 30 minutes.
b. 80 minutes.
c. 100 minutes.
d. 300 minutes.

9. Once a child becomes myopic, which modification is likely to slow the progression of myopia?
a. Increasing daily outdoor time.
b. Increasing outdoor time in summer months.
c. Increasing outdoor activity.
d. No known behavioral factors can slow progression after myopia onset.

10. Which issue complicates monitoring the amount of time children spend outdoors?
a. Surveys performed are subject to recall bias.
b. Recording devices cannot measure the time spent outdoors but can measure the amount of light reaching the eye.
c. Current devices record the viewing distance only to the nearest meter.
d. Schools regulate the amount of time children spend outdoors.

11. Which factor associated with increased near work has been shown to lead to increased myopia progression?
a. Closer reading distance.
b. Smaller print size.
c. Higher prevalence in Europeans, where there is an emphasis on education.
d. Living in a rural environment.

12. What is the expected increased refractive error for a student who completed four years of college compared with someone who did not attend college?
a. No difference.
b. -1.00DS.
c. -2.00DS.
d. -3.00DS.

13. What is the factor of increased risk of developing myopia for a child who has one myopic parent compared with a child who has no myopic parents?
a. Unknown.
b. 1.5x.
c. 3x.
d. 7x.

14. What is heritability?
a. A way to measure genetic information across thousands of individuals to identify small differences related to a specific trait.
b. The use of familial information to map locations of genes that may be related to a trait.
c. The proportion of variation in a trait that can be attributed to genetic factors.
d. Greater "within pair" difference in refractive error among identical twins vs fraternal twins.

15. Why are twin studies not as valuable as family studies for estimating myopia heritability?
a. Twin studies show a greater "within pair" difference in refractive error.
b. Twin studies overestimate true heritability due to model assumptions and confounding factors.
c. Family studies do not add any more information about the complexity of genetic covariance and environmental factors.
d. Family studies report higher heritability of refractive error.

16. Which gene shows an association with high myopia?
a. PAX6.
b. MYP1.
c. miRNA 328.
d. X chromosome.

17. What is the main benefit of genome-wide association studies?
a. Results are commercially available through 23andMe.
b. Studies can identify one specific gene of interest.
c. Variations are directly related to the trait of interest.
d. Studies can identify potential gene loci without knowledge of function

18. What area of interest have gene-environment interaction studies explored?
a. Time indoors.
b. Light levels and composition.
c. Near work.
d. Seasonal variations of daylight.

19. Potential genetic locations and mechanisms of myopia development can account for approximately what percent of the variation in refractive error?
a. 5%.
b. 10%.
c. 50%.
d. 71%.

20. When discussing myopia control with parents, all of the following key points should be mentioned EXCEPT:
a. Consequences of having high myopia (i.e., retinal detachments, glaucoma, cataracts, etc.).
b. Risk factors for developing myopia or for myopia progression.
c. Risks and benefits of all available treatment options.
d. Gene therapy for myopia control.

Examination Answer Sheet

How Environment and Genetics Give Rise to Myopia

Valid for credit through January 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.

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

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Answers to CE exam:

1. Ⓐ Ⓑ Ⓒ Ⓓ
2. Ⓐ Ⓑ Ⓒ Ⓓ
3. Ⓐ Ⓑ Ⓒ Ⓓ
4. Ⓐ Ⓑ Ⓒ Ⓓ
5. Ⓐ Ⓑ Ⓒ Ⓓ
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18. Ⓐ Ⓑ Ⓒ Ⓓ
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. Interpret the worldwide increase in myopia prevalence from an epidemiologic perspective. ① ② ③ ④ ⑤

22. Discuss the normal pathophysiologic process of emmetropization and its relation to myopization. ① ② ③ ④ ⑤

23. Explain the likely environmental and behavioral causes for the increase in myopia prevalence. ① ② ③ ④ ⑤

24. Use knowledge of the genetic factors associated with susceptibility for myopia and high myopia to identify and treat patients. ① ② ③ ④ ⑤

25. Recognize how genetic causes relate to environmental factors in regard to light-induced signaling in myopia development. ① ② ③ ④ ⑤

25. Recognize how genetic causes relate to environmental factors in regard to light-induced signaling in myopia development. ① ② ③ ④ ⑤

26. 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.

27. 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): ☐

28. 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: _____

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

ⓐ very confident ⓑ somewhat confident ⓒ unsure ⓓ not confident

30. 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:

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

Signature _____ Date _____

Lesson 117525 RO-OSC-0119

31. Additional comments on this course:

_____

_____

_____

_____

_____

Rate the quality of the material provided:

1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

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

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

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

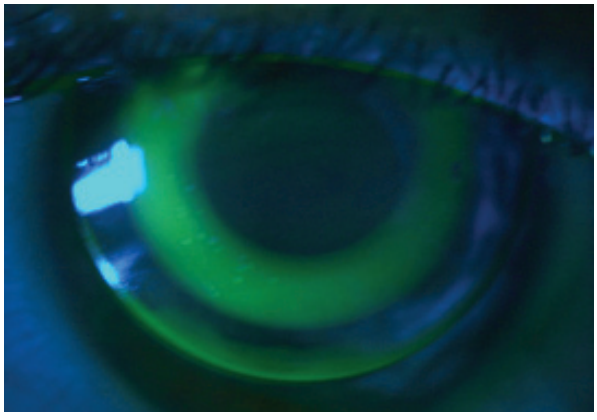
35. Based upon your participation in this activity, do you intend to change your practice behavior?
① ② ③ ④ ⑤

Myopia Treatments:
How to Choose and When to Use?

Effective management in children and young adults may require different interventions. Learn which works best for your case.

By Julie Tyler, OD, and Heidi Wagner, OD, MPH

We are at a tipping point whereby treatments to curtail the progression of myopia are moving beyond the research and specialty clinic setting into the primary eye care practice environment. It is clear that myopia is on the rise—likely for a variety of reasons including, but not limited to, an increase in near-work demands and decrease in time outdoors.[1,2] By 2050, nearly five billion people worldwide will have myopia compared with two billion in 2010.[3] While it is also clear that genetics play a role in the risk for developing myopia, inheritance patterns alone do not seem to account for the significantly increasing rate of myopic refractive error.[4,5]



Ortho-K lenses allow patients to enjoy correction-free vision until they reapply them at bedtime, as corneal reshaping occurs overnight.

Furthermore, the rise in myopia has public health significance: In addition to the daily increased reliance on spectacles and other vision correction by people with myopia, there is also an inherent risk of ocular comorbidities associated with this type of refractive error that may permanently and negatively affect vision, including increased risk of peripheral retinal degeneration, retinal detachments, myopic maculopathy, choroidal neovascular membrane and glaucoma.[6]

This discussion of pharmaceutical interventions, prescription modifications, orthokeratology (ortho-K) and soft bifocal/multifocal contact lens prescriptions is focused on the management of children and young adults. With regards to myopia interventions, recent research presents the most compelling evidence for ortho-K, bifocal/multifocal soft contact lenses and low-dose atropine.[7–9] It should be noted that these interventions are not approved by the United States Food and Drug Administration (FDA); therefore, these options are off-label applications in the United States. Optometrists should present to parents such information as part of the informed consent process.

Atropine

The exact mechanism of action for slowing the progression of myopia with atropine is not well understood; notably, researchers have observed that low-dose atropine significantly reduces the progression of refractive error but has a lesser effect on axial length.[10]

Atropine is an anticholinergic agent (muscarinic antagonist) that competitively binds to receptors with acetylcholine or other agonists. In the eye, atropine is known to induce mydriasis by blocking contraction of the circular pupillary sphincter muscle—allowing the radial iris dilator muscle to contract and cause paralysis of accommodation (cycloplegia). For years, atropine and other mydriatic-cycloplegic agents have been used as part of the comprehensive management of anterior uveitis, but more recent studies have considered and compared treatments with atropine at levels ranging from 0.01%, 0.1% and 1.0% for myopia progression/control.[11,12]

Initially, 1% atropine was proposed as a method to delay or deter development of myopia.[13] Unfortunately, side effects, such as burning or stinging of the eyes, conjunctival injection, blurred vision and systemic symptoms including flushing, tachycardia and restlessness, irritability or anxiety proved to be clinically significant enough to limit customary treatment. Additionally, higher concentrations of atropine were associated with a greater rebound effect when the medication was discontinued.[10,14,15] Fortunately, continuing research has shown that lowering the concentration of atropine continues to result in measurable slowing of myopia progression while also allowing for tolerable side effects and a sustained effect in modulating the refractive error. [10,11,14,15] Thus, low-dose (0.01%) atropine can help slow myopia progression while minimizing the rebound effect and without noticeably increasing pupil size or blurring near vision.

Additional study is needed to understand how to optimize treatment regimens for individual patients based upon age, refractive error and other patient characteristics.[10] It remains to be seen whether this treatment option will be less protective for ocular pathology if it exerts less effect on axial length.

While using atropine to manage myopia progression, patients also require correction for their current level of ametropia; therefore, spectacles are still necessary for many children on atropine therapy who already have developed myopia. For some children and parents who would prefer only one "method" of managing their refractive status, optical corrections/contact lens management may be a preferable myopia control option. However, other children (and their parents) may not embrace the concept of wearing contact lenses and the associated lens care and adverse events such as infectious keratitis. In that subpopulation, spectacle wear and atropine would be the most effective treatment option.

Low-dose atropine requires compounding at a pharmacy, making availability and cost a potential deterrent. Patients or parents who choose this option should instill one drop in each eye at bedtime for optimal myopia control and minimal side effects. While most young patients do not report blurred near vision or photophobia with lower concentrations of atropine, practitioners may wish to prescribe progressive addition lenses and photochromic lenses for the occasional symptomatic patient. For example, in a previous study with 0.05%, 0.025% and 0.01% atropine eye drops, the accommodation amplitude was reduced by 1.98±2.82D, 1.61±2.61 D, and 0.26±3.04D, respectively.[12]

Further research may provide additional understanding regarding patients who may benefit from additional spectacles support.

Spectacles

Historically, many proposed that the effect of "myopic defocus" in some patients may contribute to the development or progression of myopia. If a child's spectacles were undercorrected (one study proposed blur at +0.75DS), the patient might have a delay in myopic changes in the eye, including axial length measurements and refractive status.[16] However, multiple studies evaluating undercorrection in children have suggested that underprescribing either enhances or has no effect on myopia progression.[16,17]

Additional considerations for spectacle prescribing include the use of bifocal and single vision lenses to slow myopic progression. One study demonstrated some benefit of executive bifocals in children—especially in combination with prism—for mild myopia control with results comparable with the ortho-K and multifocal soft contact lens studies, in terms of axial length and refractive myopia control.[18] The benefit was more effective in patients with low lags of accommodation. The results with executive bifocals differed from the COMET study, which used progressive lenses and also demonstrated minimal clinical applications.[19] These results not only help support the theory of peripheral defocus as a contributor to myopic progression but also identify that some subsets of children may have clinically relevant benefits with spectacles to deter myopic progression.

While often not the primary myopia management strategy, the necessity of spectacles as an adjunct to therapy is still evident. Children who wear contact lenses should always have a back-up pair of glasses available in the case of an infection, ocular surface irrita-

tion and visual correction when not wearing contact lenses. Moreover, when binocular issues occur concurrently with myopia, spectacles—including the use of prism or bifocal lens types—may be key to achieving overall normal, single, clear binocular vision for patients prescribed low-dose atropine or contact lenses as their primary myopia management protocol.

Contact Lens Wear

In addition to slowing the progression of myopia, contact lens wear provides other benefits to children and adolescents who don't enjoy wearing spectacles. It provides a necessary vision correction and has been shown to improve vision-specific quality of life in myopic children younger than 12 years of age.[20] Research also shows that contact lens wear boosts self-esteem with regards to physical appearance, athletic competence and social acceptance.[21] Soft multifocals and ortho-K contact lenses provide comparable efficacy (approaching 50%) with regards to slowing the progression of myopia.[22] Given that the mechanism for myopia control with atropine appears to be different than the mechanism with contact lenses, combination methods may prove to be effective and are currently being investigated.[23]

Table 1. Considerations for Management of Myopia Progression

Management Type	Pros	Cons
Atropine (recommended dosage of 0.01%)	• Limited side effects • Can be used with conventional spectacles • Parents can manage • Avoids contact lens wear	• Compounded medication and costs may be a challenge • Mechanism of action unknown
Modified Spectacle Prescription	• Potential high effect on concurrent associated near signs/symptoms • May be used in tandem with ortho-K for higher refractive errors	• Standardization for prescribing difficult • Undercorrection has not proven to be effective
Orthokeratology (Ortho-K)	• Correction-free vision during waking hours • Ideal for activities where water exposure is inevitable or lens dislocation is likely • Parents can assume responsibility for lens application, removal and care • Well-established treatment regimen in some global regions • Minimal reliance on spectacles	• Risk of infectious keratitis • Only corrects low to moderate refractive error • Variable responses • Fluctuating vision possible • Compliance/consistent wear schedule important • More exacting lens parameters
Bifocal/Multifocal Soft Contact Lenses	• Commercially available options • Wide parameters availability for correcting high ametropia	• Risk of infectious keratitis • Increased expense compared with single-vision contact lenses • Back-up spectacles add to the expense of the regimen

Orthokeratology

This myopia control method uses custom-designed gas permeable contact lenses that reshape the cornea to temporarily reduce refractive error. Paragon's corneal reshaping treatment received FDA approval in 2002, while Bausch + Lomb's Vision Shaping Treatment was approved in 2004. Notably, these approvals were for lenses prescribed for the correction of refractive error rather than for devices intended to reduce the progression of myopia.

While the exact lens parameters are proprietary, common characteristics of commercially available lens designs include a large diameter, small optic zone and a secondary (reverse) curve that is steeper than the base curve radius. This design enhances lens centration and promotes central epithelial cell compression. Fluid moves from area of central compression to surrounding areas of relief. The resultant mid-peripheral corneal thickening and shift of peripheral retinal defocus has been proposed as the primary myopia-inhibiting stimulus, although a cause-and-effect relationship has yet to be established.

Ortho-K is primarily used as a correction for low-to-moderate myopia (up to -6.00D) with or without astigmatism (up to -1.75D). Alternatively, the practitioner may elect to partially correct higher refractive errors with ortho-K lenses and prescribe spectacle over-correction for daytime use.[24] At the other end of the refractive spectrum, low (less than or equal to 1.25D) refractive error is relatively easy to correct but the amount of

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peripheral defocus generated will be modest and possibly less effective in reducing myopic progression.

Initial lens selection may be based on empirical methods (spectacle prescription, keratometry, horizontal visible iris diameter), diagnostic lens fitting and/or corneal topographic analysis. Corneal topography allows for a more nuanced approach to identifying optimal candidates and is essential for monitoring lens position in overnight wear and performance over time. Such measures allow practitioners to assess corneal eccentricity and evaluate whether a toric peripheral curve system is indicated to optimize the lens-to-cornea fitting relationship. Toric lens designs are based on the sagittal height difference between corneal meridians rather than the manifest astigmatic refractive error. With-the-rule corneas are generally more amenable to treatment, as are steeper corneas.

Other prefit considerations include the patient's and parents' interest and motivation in wearing lenses. By design, ortho-K lenses entail applying lenses before bedtime. While the patient may initially exhibit apprehension in wearing rigid lenses, closed-eye wear minimizes lens awareness. Corneal reshaping occurs overnight so that patients enjoy correction-free vision until they reapply the lenses. Remaining lens-free during the day may be especially desirable for patients participating in sports such as swimming, where water exposure is inevitable, or other activities where lens dislocation is likely.

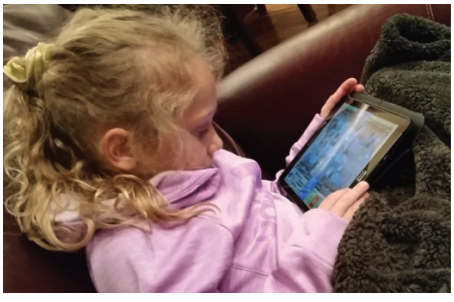
While more severe anterior segment disease is a contraindication to ortho-K lens wear, patients with mild dry eye or allergic symptoms may prefer overnight ortho-K to daily contact lens wear. The benefits of ortho-K must be considered against the risks (*Table 1*). Finally, ortho-K is a well-accepted treatment in some regions while relatively unknown in others; consequently, treatment decisions may also be influenced by the patient's family and peers.

Uncorrected visual acuity may fluctuate during the fitting process or if the lenses are not worn on a consistent schedule. This may be of greater concern for older adolescents or young adults who are driving. Similarly, college-aged students may have erratic schedules and sleep patterns that result in an inconsistent wear schedule and variable vision.

Finally, the more exact fitting process may be appealing to some practitioners while off-putting for others. At minimum, the initial fitting process involves a baseline examination and fitting, dispensing visit, morning-after visit and follow-up visits until the desired endpoint is reached.

Periodic follow-up care is similar to that of other contact lens wearing subjects, with the caveat that more frequent visits may be merited, based upon on the overnight wear schedule and the needs of the individual patient.

In a previous retrospective study in the United States, the risk of microbial keratitis in ortho-K was similar to that of overnight wear of soft contact lenses.[25] Corneal staining and lens binding have also been reported with this modality. Thus, successful ortho-K lens wear relies upon precise lens fitting, proper wear and care practices, periodic follow-up care and timely treatment of adverse events.



Limiting time spent on electronic devices and length of near-distance work may reduce the chances of developing myopia.

Multifocal Prescribing

The use of both multifocal spectacles and multifocal contact lenses for the management of myopia is something that many optometrists may be more comfortable with for the medical management of contact lens corneal reshaping therapy. In part, this is because most doctors write prescriptions for refractive error regularly. Compared with ordering compounded medications and the exam time and nuance of ortho-K, spectacle lenses are relatively easy to be made and multifocal contact lenses are available "off the shelf" (*Table 2*).

Soft multifocals. Center-distance daily wear contact lenses provide an alternative contact lens management option for reducing the progression of myopia.[26-31] The central portion of the lens provides the distance myopic vision correction while the peripheral surround reduces hyperopic defocus. This, in turn, minimizes the stimulus for myopia progression by focusing the light in front of the peripheral retina. Commercially available lenses in the United States include CooperVision's Biofinity and Proclear "D" monthly replacement lenses and Visioneering Technologies' NaturalVue Multifocal 1-Day. Other products such as CooperVision's dual-focus MiSight lens are available in Canada.

Soft multifocals are an excellent option for patients averse to overnight wear, those with refractive error beyond commercially available ortho-K parameters, or ortho-K dropouts. These

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REVIEW® OF OPTOMETRY

Myopia and Use of Electronic Devices

By Raman P. Sah, B.Optom

The rapid increase in myopia prevalence over the last 50 years has occurred simultaneously during a period of rapid technological advances, resulting in the dominant use of electronic devices for displaying everything from text, pictures, movies, games and more. As technology advanced, the spatial resolution of these displays improved from Video Home System (VHS) in the 1970s with resolutions of roughly 333 pixels by 480 pixels to current Macintosh retinal displays with native resolution up to 2,880 pixels by 1,800 pixels in 2018.[1,2] However, as screen resolution improved, display sizes have reduced, and we now have high resolution (up to 2,436 pixels by 1,125 pixels) displays that are handheld (14cm by 7cm) that can be viewed at close distances without visual detection of individual pixels.

Also, as might be expected with these great advances, the prevalence of handheld electronic devices that children and teenagers use has grown exponentially. A 2017 report by the Common Sense Media, a nonprofit organization, suggests that 98% of households with kids under eight have a mobile device.[3] It is also reported that 42% of children of age eight and younger now have their own mobile devices, with the average usage being two hours and 19 minutes.

A similar report cites that a majority of teenagers spend more than four hours per day with screen media.[4] Therefore, children and teenagers are exposed to these short viewing distances and electronic displays for a significant amount of time. Could an unintended consequence of the improved display resolution in modern electronic displays be a worldwide myopia epidemic?

In a lab study, we examined the accommodative behavior of young children (ages seven to 16) when binocularly viewing different targets presented on electronic displays (as they do in real life), and monocularly. We quantified the accommodative lag in diopters of hyperopic defocus. Our results revealed that both emmetropic and myopic children experience typical lags of accommodation (mean +0.54D and +0.32D, respectively) at the routinely experienced viewing distances of these devices between 33cm and 20cm.[5]

Research with infant monkeys has shown that exposing young primates to artificially induced chronic hyperopic defocus will trigger compensating axial myopic eye growth.[6,7] In our study, however, we did not find larger accommodative lags in children viewing the electronic displays than those shown in reports published previously for children viewing printed text materials.

Therefore, if the electronic displays are a contributing factor in myopia development, it is likely that they do so by increasing the amount of time children are exposed to hyperopic defocus and not the magnitude of the hyperopic defocus. In particular, recent studies on myopia control suggest that spending time outdoors might be a preventive factor for myopia.[8] These electronic devices could indirectly be contributing to myopia development by influencing children to spend more time inside and less outside.

In general, myopia is considered to have a multifactorial etiology (combining genetic, environmental and hereditary influences), and studies show that early onset is often linked to increased myopia in adulthood. Thus, it is sensible to adopt preventive measures early in life with regular comprehensive eye examinations. Further research is needed to know what part, if any, controlling digital device use may ultimately play as a clinically recommended preventative measure in possibly minimizing myopia progression.

Dr. Sah is currently a Vision Science PhD student at Indiana University Bloomington and presented the study, "Accommodative Behavior and Behavior Defocus in Children Viewing Electronic Devices" at the 2018 AAO Meeting in San Antonio.

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lenses are also feasible for patients who wear contact lenses part-time, although reduced wearing time likely limits the lenses' dampening effect on myopia progression. A previous study reported that the myopia modulating effect increased with daily wearing time, but the benefit plateaued after eight hours.[30] The fitting process is relatively straightforward and comparable to other soft lens modalities with regards to chair time and follow-up schedule. Therefore, patients, parents and, in some cases, practitioners, may be more familiar with soft lenses and more readily embrace this treatment regimen.

The Biofinity multifocal is available in spherical powers up to 8D and four add powers (+1.00D, +1.50D, +2.00D, +2.50D).

Proclear lenses provide even wider parameters. In clinical practice, the higher add power is initially selected to enhance the peripheral defocus, but the minus distance power may need to be increased slightly (typically -0.50D to -0.75D) to enhance distance vision. Occasionally, the add power may need to be reduced to optimize distance visual acuity.

The NaturalVue 1-day multifocal contact lenses, with an extended depth-of-focus center distance design, are manufactured with one add power up to +3.00D.

They are currently available in spherical power up to -12.25D. Troubleshooting typically involves adding minus lens power to enhance distance vision.

In contrast, CooperVision's MiSight is a daily disposable lens with two zones for vision correction and two for treatment (2D myopic defocus). It is not yet available in the United States but is available in Canada.[31] The risks of adverse events are comparable to other soft lenses worn for daily wear.[32]

Beyond the specific therapies discussed previously, there are additional recommendations that we can make for our young patients when specific "additional" myopia management is not pursued. Notably, children who spend more time outdoors are less likely to develop myopia.[33] While there is an emerging consensus that environment plays a factor, it is not well understood. Future recommendations for children might include participating in activities that encourage outdoor play for a period of time daily ("prescribed play") as well as limiting time spent on electronic devices such as smartphones and tablets. It is also important to increase the working distance when using these devices.

Myopia, now commonly described as an epidemic, is a public health concern and common cause of visual impairment. Therefore, interventions designed to slow the onset of myopia and curtail its progression are of critical concern. Eye care professionals can potentially play an active role in modifying myopic outcomes. The needs and lifestyle of the individual patient should be taken into consideration when developing a treatment plan. By embracing strategies to reduce myopia progression, optometrists have the opportunity to reduce the economic burden of vision loss due to the condition. ■

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Dr. Wagner is a professor of clinical optometry and director of extern programs at Ohio State University.

Table 2. Commercially Available Contact Lens Products in the US

Orthokeratology Lenses	Soft Multifocal Contact Lenses
• Paragon CRT	• CooperVision Biofinity "D"
• B+L Vision Shaping Treatment	• CooperVision Proclear "D"
	• Visioneering Technologies NaturalVue 1-Day

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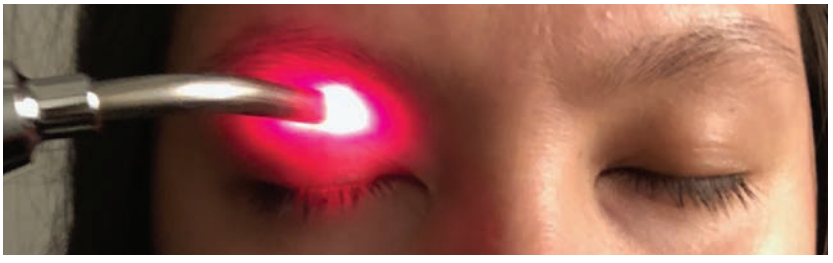
What You Can Learn from Lids

A proper evaluation can lead you to targeted dry eye treatment. **By Azinda Morrow, OD**

With everyone's eyes perpetually scanning the myriad of glowing screens around us, dry eye symptoms are getting worse and affecting younger demographics than ever.[1] For the optometrist, these habits make it more likely that a patient with dry eye will appear in your chair complaining of fluctuating vision, redness, foreign body sensation and even pain.

According to the most recent Tear Film and Ocular Surface Society International Dry Eye Workshop II (TFOS DEWS II), dry eye disease (DED) is characterized by a loss of homeostasis of the tear film.[2] By discovering and understanding the underlying etiology—whether due to hormonal changes, contact lens intolerance, dysfunction of the meibomian glands or other reasons—using a targeted work-up and approach towards management is the only effective way to improve patient symptoms and clinical signs.

Management of these patients is not "one size fits all," and requires significant tailoring to each patient's symptoms, severity and lifestyle. Often, practitioners advise patients to first use artificial tears, up to four times a day. If used properly, tear supplementation therapy may increase lubrication; however, in some cases, they alone may not alleviate all complaints.



Korb-Blackie test indicating incomplete closure of the eyelids due to light being seen emanating from the lid margin of the right eye.

In most dry eye cases, practitioners will often apply vital dyes to assess for corneal staining to determine tear break-up time. Although this offers valuable information, a thorough eyelid assessment is also necessary to determine additional contributors to the patient's signs and symptoms. This article provides a guide through a variety of eyelid findings and evaluation techniques that can alter your typical management approach and provide the relief your patients are seeking.

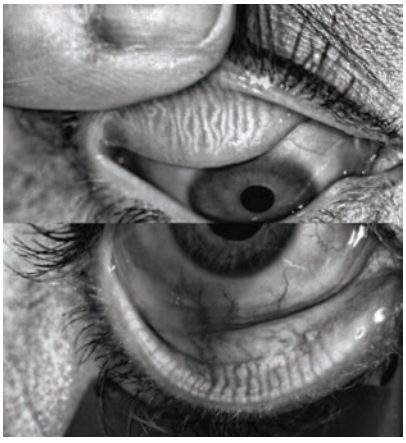
Incomplete Blink

Corneal staining with sodium fluorescein, particularly in the inferior one-third of the cornea, often arises from incomplete blink and closure. Even just asking a patient to close their eyes under high magnification, or watching their blink rate and pattern, can provide insight. If poor closure, incomplete blink or infrequent blinking is present, the patient may be suffering from the effect of the lids poorly distributing the tears along the ocular surface, or from lagophthalmos during sleeping hours, both causing chronic dryness.

Even if the eyelids appear to have complete closure, the Korb-Blackie light test can discern if the patient has incomplete closure, if any, and to what degree.[1] By resting a transilluminator along the patient's closed eye, light escaping from the lid margin indicates a lack of closure. Thicker artificial tears in the form of gels or ointments for overnight use are available for these cases. Lid-taping regimens when the patient is asleep, and moisture goggles for more severe cases, can also help.

Meibomian Glands

Keratinized—or capped—meibomian glands are an easily discoverable finding, which can indicate meibomian gland dysfunction (MGD) as a component to the patient's dryness. If the glands themselves are clogged and unable to secrete the lipid component of the tear film, tear evaporation time will likely be compromised. Heat masks may be effective in liquefying material trapped in the glands and help express meibum.[13]



Meibography images of the upper and lower eyelids indicating mild gland tortuosity and dilation.

Lipiview (Tearscience), Keratograph (Oculus) and Meibox (Box Medical Solutions) are some of the options doctors use to evaluate the glands for tortuosity, dropout or atrophy. However it's accomplished, meibomian gland imaging should be part of your work-up on all patients, no matter the age.[5] This protocol can help establish baseline information and potentially uncover signs prior to the onset of symptoms.

Lipid-based artificial tears can help patients battle underlying gland dropout or dysfunction, as the oily components in these drops can help thwart evaporation of tears from the ocular surface.

Even prior to performing any dry eye work-up or instillation of dyes, using the high magnification with biomicroscopy to view the eyelids is helpful. In addition, by

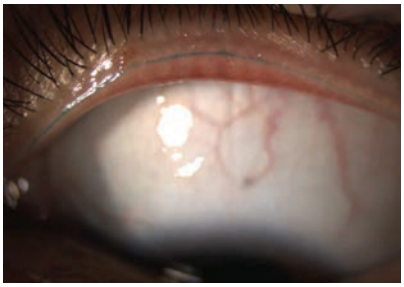
manually moving the eyelids and assessing the meibomian glands and palpebral conjunctiva, complete with lid eversion, more details are revealed. It is critical not only to assess the cornea with fluorescein staining, but also to assess the eye with lissamine green stain. Unlike sodium fluorescein, lissamine will allow for visualization of the devitalized cells of the bulbar and palpebral conjunctiva. Staining of these areas may indicate extreme global dryness that can be missed on standard fluorescein staining.

Papillary Reaction

Foreign body sensation or irritation in the eyes can masquerade as a papillary reaction, with or without seasonal allergies. Although papillary reactions are often associated with symptoms such as ocular itching, some patients may have difficulty in expressing their symptoms, and if papillae are present, it is important to consider an antihistamine/mast cell stabilizer combination drop to improve ocular comfort. More severe presentations—namely, giant papillary conjunctivitis—may occur, typically with soft contact lens wear, due to frictional forces or protein hypersensitivity. A steroid may be warranted in these cases, depending on the level of severity of patient symptoms and clinical signs. Monitoring the appearance of the palpebral conjunctiva is critical to a patient's success and specifically in avoiding contact lens intolerance.

On the Lashes

Often overlooked, the lashes too can offer clues on what might be exacerbating a patient's symptoms. For example, if trichiasis exists, positive sodium fluorescein staining near the lash margin may be confused with lagophthalmos or incomplete blink, based on corneal signs alone.[8] However, if rogue lashes are noted, epilation or (in severe cases) surgical manipulation may be necessary to relieve the globe from irritation.



Staining shows lid wiper epitheliopathy present on the upper eyelid.

If debris is present, depending on the shape of the scurf (or collarettes), a practitioner must determine whether the underlying etiology is seborrheic (flaky debris) or secondary to *Demodex* (round, tubular debris found at the base of the lashes). Although *Demodex* presence was originally thought to be a symbiotic relationship, patients with more prominent *Demodex* are found to have a higher likelihood of inflammatory markers in the tear films, with increased symptoms of dryness compared to those with reduced *Demodex* levels.[7] Lid scrubs, such as from Ocusoft or Avenova, are options to assist in eyelid hygiene in cases of blepharitis. Similarly, if *Demodex* is noted, research shows tea tree oil scrubs can be effective in reducing a patient's ocular surface discomfort index (OSDI) score and improve symptoms.[7]

Lid Wiper Epitheliopathy

This disturbance of the eyelid margin affects the portion of the lid that is essential for spreading tears across the globe. This finding can be seen in patients with abnormal eyelid structures, dry ocular surfaces or a history of contact lens wear.[3] By using lissamine green staining, significant dryness can be discovered secondary to frictional and mechanical forces of the eyelids against the globe.

Unfortunately, findings of lid wiper epitheliopathy may not be seen with sodium fluorescein, as the condition has no correlation to tear break-up time or Schirmer testing.[3] Therefore, instillation of lissamine green staining is essential in those patients who are symptomatic but showing no signs of dryness.[3]

These patients may benefit from lid exfoliation with lid scrubs, as well as tear supplementation therapy to decrease friction. In addition, switching soft contact lens materials or adding coatings to rigid lenses may help to alleviate these signs and symptoms.

Ocular Rosacea

Whether present with a diagnosis of systemic rosacea or not, underlying inflammation due to ocular rosacea can cause a patient to have discomfort and irritation.[10] Telangiectatic vasculature present on the eyelid margins is pathognomonic for the condition, but can also be present alongside blepharitis, keratitis or conjunctivitis.[10] This condition most commonly manifests between the ages of 40 to 60 and can have significant detrimental effects on ocular comfort and productivity.[10] Targeted therapies toward eliminating or decreasing the inflammation, such as with low-dose oral doxycycline, is necessary in these cases.

Trauma

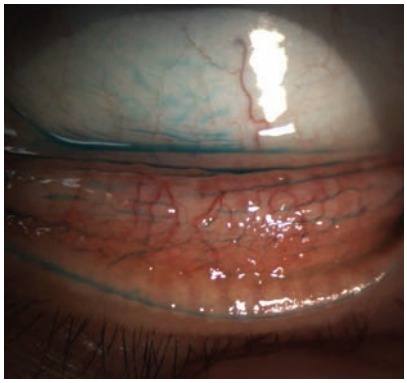
Lid anatomy can vary widely after trauma, which includes surgical interventions. For example, after correction of an entropion or ectropion, removal of lid lesions or with

blepharoplasties, normal eyelid architecture may be compromised. This in turn can impact the apposition of the eyelid to the globe, disrupt the correct placement of the meibum component into the tear film, as well as the anatomy of the glands themselves.

Careful case history and communication with other eye care providers will assist in determining if trauma or surgery may be confounding factors in a patient's dry eye symptoms.

Contact Lens Discomfort

Although all of the above lid findings can be noted in any patient, patients who wear contact lenses require an especially careful assessment, as these findings may be the determining factor between whether a patient is successful or discontinues lens wear. Forty-one



This patient displays moderate papillary reaction with lid wiper epitheliopathy on the lower eyelid.

percent of patients drop out of contact lenses due to visual complaints, and 36% due to ocular discomfort, with both of these complaints potentially being secondary to dryness.[9] However, most sources report that up to half of all contact lens wearers have some discomfort.[4] Materials and coatings can influence comfort, but lens wear itself also has effects on the ocular surface and adnexa.[4] The palpebral conjunctiva, which directly abuts a contact lens, has been found to yield more prominent lid wiper epitheliopathy, especially in lenses with low lubricity.[4]

While more studies are necessary, current research also indicates that contact lens wear can cause dilation and inflammation of the meibomian glands, as well as partial or complete loss, with contact lens wearers having gland length that is half of that observed in non-contact lens wearers.[4] These effects were noted in both rigid and soft lenses, with more gland dropout occurring in the upper and lower eyelids, respectively.[4] In addition, wearers have more microbes colonized on their lid margins, foam-

ing of the inferior lid margin and decreased blink rate compared with non-contact lens wearers.[4] Therefore, as these patients are more prone to having pertinent eyelid findings, take extra care to enhance lubrication and overall comfort with the proper recommendations.

Regardless of whether a patient wears contact lenses, prescribed home therapies can significantly help to treat dry eye. However, it is also essential to consider in-office therapies as an adjunct in cases where patients are still symptomatic. The variety of these in-office procedures have expanded significantly, and options for patients range from relatively simple to more complex involving purchased equipment in your office.

In-office Procedures

Optometrists have a number of potential interventions to consider and offer in their practices:

Gland expression. With the use of in-office warm compresses, and the high magnification achieved with biomicroscopy, a practitioner is able to express the meibomian glands manually or with the use of expressor paddles. With this technique, you can not only assess the quality and consistency of the meibum expressed but also encourage healthy expression once the patient is out of the office. Meibomian gland probing has also recently gained popularity due to case reports showing regrowth and restoration of atrophied glands.[6]

Lid debridement/Blephex (Blephex). Running a spud along the meibomian gland orifices to remove keratinization, as well as assist in exfoliation the eyelids, can eliminate capped glands, lid wiper epitheliopathy and promote healthy expression of meibum afterwards.

Another option is the Blephex procedure, which involves a rotating sponge that the practitioner uses to clean and exfoliate the eyelid margins. The process takes approximately seven minutes and is performed on each individual eyelid. Results are optimal if the procedure is repeated every four to six months.

Lipiflow/Mibo Thermoflo (Mibo Medical). These systems are a way of adapting a common home therapy (warm compresses) into a pulse-dosed in-office therapy. Both involve heating of the eyelids with concurrent massage. The procedures differ in that the Mibo Thermoflo involves a probe operated by a technician or doctor on the outside of the lids whereas Lipiflow is an automated system with an applicator that provides massage of the lids both on the underside and outside. Research shows these procedures are about equal in efficacy in improving signs and symptoms compared to three months of twice daily home warm compresses.[11]

iLux (Alcon). This handheld device uses light-based technology to heat the meibum from the inner and outer portions of the eyelids, allowing for expression of the glands, all under high magnification. This therapy allows manual gland expression outside of the slit lamp and provides specific measurements regarding length of treatment as well as the actual temperature when performing meibum expression.

Bandage contact lenses/scleral lenses. Depending on the severity of the ocular surface disease, bandage contact lenses and scleral lenses can be useful for patients who are unsuccessful with other therapies. By providing a mechanical barrier from the eyelids and environmental conditions, contact lenses can be extremely useful not only to provide symptomatic relief for patients, but also to protect and promote healing of the cornea and ocular tissues.

Although many patients may present with similar symptoms of dryness, burning or ocular irritation, it is important to evaluate the lids for clues. Many eyelid findings will direct you to very specific treatments targeting the underlying etiologies, in cases where typical artificial tear supplementation may have been unsuccessful.

There are many options for us to consider for our dry eye patients with lid disease. From improvements in lid hygiene to medical management ranging from ocular lubricants to steroid therapy, careful observation and examination may allow you to discover the targeted therapy necessary to finally be able to help these patients achieve the relief they are looking for. ■

Dr. Morrow is an assistant clinical professor at the State University of New York College of Optometry.

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Antibiosis Abroad

New Zealand indication makes the case for a drug's use in the United States.

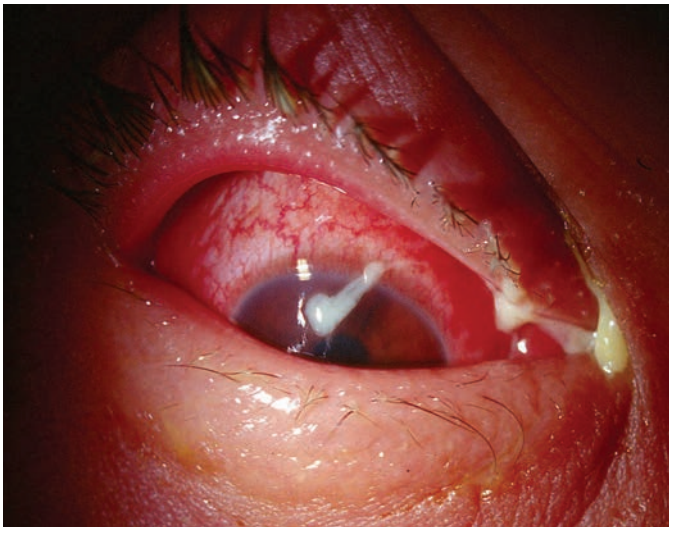
By Joseph W. Sowka, OD

I recently had the opportunity to again lecture to the New Zealand Association of Optometrists. My last time there, 10 years earlier, therapeutic drug laws had recently been passed and optometrists were going through the education and certification process to obtain prescribing privileges. Now, a decade later, optometrists in New Zealand are registered prescribers (the same as medical doctors) and are allowed to use virtually any medication available within their specialty.

While in New Zealand, my wife and I enjoyed the beauty of the Bay of Islands, the history of the Waitangi Treaty Grounds, and the natural beauty of Fjordland National Park and Milford Sound. We saw unique fauna including the kiwi and the tuatara. However, there were some things that I didn't see. I did not see anyone dead or dying in the streets. I did not hear the sirens of ambulances racing through the streets, nor did I see any funeral processionals heading to overfilled cemeteries. Indeed, I saw none of these horrendous sights, despite the fact that the most commonly prescribed topical ophthalmic antibiotic in New Zealand is chloramphenicol.

What is Chloramphenicol?

This bacteriostatic antibiotic inhibits bacterial protein synthesis and, when used in high doses or against highly susceptible organisms, it can be bactericidal (in high concentrations).[1] Chloramphenicol is available in systemic form as well as a topical ophthalmic 0.5% solution and 1% ointment. Dosing is recommended at five times per day.[1]



A case of bacterial conjunctivitis, as seen here, is frequently treated with chloramphenicol outside the United States.

The drug has a broad spectrum of both gram positive and gram negative antibacterial activity.[2-6] It is effective in treating anaerobic organisms, mycoplasma, rickettsia and chlamydia.[7] More than 95% of *Haemophilus influenzae, Neisseria meningitides, Neisseria gonorhoeae, Salmonella typhi*, Brucella species and *Bordetella pertussis* are susceptible to chloramphenicol.[7] Especially noteworthy is the low rate of clinical and microbiologic resistance and its ability to conquer organisms that demonstrate resistance to more commonly available antibiotics.[8-10]

Research shows chloramphenicol is also effective against ocular methicillin-resistant *Staphylococcus aureus* (MRSA) infections.[10] It is used extensively throughout the world (with the exception of the United States) for treatment of acute bacterial infections, corneal trauma and surgical prophylaxis.[10-15] Recent resistance studies have shown that chloramphenicol has a strong susceptibility profile and a low resistance.[16,17] Notable is that isolates of *Streptococcus pneumoniae* exhibit non-susceptibility to azithromycin (31%) and penicillin (38%) while remaining susceptible to fluoroquinolones and chloramphenicol.[16,17]

The Fall of Chloramphenicol

Since the inception of systemic chloramphenicol, there has been a reported association with several blood dyscrasias, the most notable of which is aplastic anemia. Fatal aplastic anemia has been associated with systemic chloramphenicol use.[1,7,18]

The first case of aplastic anemia associated with topical use was

reported in the 1960s.[7] In 1980 and 1982, the first two cases of fatal aplastic anemia that were thought to be associated with topical chloramphenicol use were reported.[19,20] Another fatal case believed to be associated with topical use was reported in 1992.[21]

To date, there have been 23 cases of aplastic anemia (the majority of which were not fatal) associated with the use of topical chloramphenicol.[7] While topical chloramphenicol use is widespread throughout the world, this possible association with aplastic anemia curtailed use of the drug in the United States. In fact, American prescribing information for chloramphenicol issues a warning that it should only be used if there are no other options available.

Another Look

Aplastic anemia is a condition in which bone marrow does not produce sufficient new cells to replenish dying and aging blood cells.[22] The marrow suffers from an aplasia that renders it unable to function properly. This results in anemia, with fewer erythrocytes, leukocytes and platelets than normal or fewer than are needed to function properly. Aplastic anemia is life-threatening, demonstrating a 50% mortality rate. The condition can be associated with certain medications or it may occur idiopathically.[22]

Despite the reported association between chloramphenicol and aplastic anemia, a causal relationship has yet to be conclusively established. Many in the medical community are skeptical of claims that aplastic anemia is actually caused by chloramphenicol.[1,7,23-25]

Of the 23 possible cases of topical chloramphenicol-induced aplastic anemia, only seven were actually published; the remainder were never fully investigated.[7] And of those seven, compelling evidence actually de-emphasized the role of chloramphenicol. For instance, all but one patient had long periods of topical therapy (13 months average), and three patients concurrently used other marrow-toxic medications. Two of these patients had concomitant liver disease. Possible genetic predispositions were also found in three patients (family history of aplastic anemia, pernicious anemia and leukemia).[7]

During a 10-year span in the United Kingdom, only 11 suspected cases of non-fatal topical chloramphenicol-induced blood dyscrasia were reported, vs. more than 200 million uses.[18] This frequency of approximately 1:20 million cases is far less than the 1:100,000 frequency of penicillin-induced anaphylaxis.[18] In one report, the incidence of aplastic anemia among users of topical chloramphenicol was 0.36 cases per million weeks of treatment.[24] The incidence of idiopathic aplastic anemia among nonusers was 0.04 cases per million weeks.[24]

Based upon this information, an association between ocular chloramphenicol and aplastic anemia could not be excluded, but the risk was less than one per million treatment courses.[24] The minimum total dose of topical chloramphenicol associated with marrow toxicity is 30mg and the minimum duration of exposure is 18 days.[1] A typical course of topical chloramphenicol therapy delivers only 10mg to the eye.

Mythbusting

In their editorial based upon the literature and worldwide use, one research team postulated that topical ocular chloramphenicol "possibly" can cause blood dyscrasias and aplastic anemia, and the latter is frequently fatal but is not possible

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to quantify the risk, which appears to be very rare (approximately one in a million based on a very small number of case reports).[26] They also noted that it is likely that there are genetically susceptible individuals to this idiosyncratic reaction, and it is not possible to identify who these individuals are. They remarked that if some medications are not available based on cost alone, drugs like chloramphenicol eye drops could be considered as a viable treatment option.[26]

The point of this month's column is not to induce resurgence in chloramphenicol use, but to challenge the myths about this drug. Though difficult to obtain, it is still topically available and can be vital in patients with resistant (including MRSA) ocular infections. By avoiding chloramphenicol use in patients with a family history of aplastic anemia, liver disease or known chloramphenicol sensitivities, this drug could be applied safely and effectively in a wide range of patients.

Chloramphenicol is used extensively in New Zealand, Australia and several other countries. How worried are they about the so-called "deadly effects?" Apparently, not very. Until recently, topical chloramphenicol was available over-the-counter in these countries and any parent could pick up a bottle for a child's red eye. More recently, it has been moved behind the counter, but is available without a prescription after a brief consultation with a pharmacist (no biomicroscopy required) to ensure that patients didn't have symptoms of uveitis.

While in New Zealand, I picked up the beginnings of a conjunctivitis myself and foolishly had no samples with me. I went to a local pharmacy where I obtained, not surprisingly, a bottle of chloramphenicol.

Somehow, I survived. ■

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Edited By Carlo Pelino, OD, and Joseph J. Pizzimenti, OD

All Vessels, Great and Small

In vasculitis, inflammation can lead to serious problems in multiple body systems, including the eye. **By Alexandra Zuercher, OD**

Although the diverse nature of vasculitis presents a diagnostic challenge, optometrists are up to the task. A careful patient history, ophthalmic testing and a systemic workup are integral to detecting and managing signs of this heterogeneous group of disorders that includes rare forms such as granulomatosis with polyangiitis (GPA), also known as Wegener's granulomatosis.

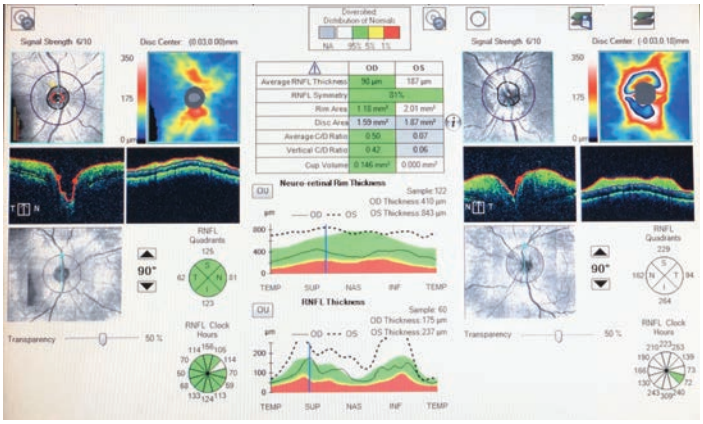

Fig. 1. OCT of the left optic nerve the day this patient first presented with GPA reveals optic disc and retinal nerve fiber layer edema.

Vasculitis Essentials

The term *vasculitis* refers to a group of relatively uncommon conditions characterized by inflammation and damage in blood vessel walls, leading to tissue necrosis. Vasculitis has a reported annual incidence of 40 to 54 cases per one million persons and appears to vary with geographical location, patient age and seasonal changes.[1]

Blood vessels of any type, including capillaries, veins/venules and arteries/arterioles, can be affected, resulting in a broad spectrum of signs and symptoms. While vasculitis may be limited to the skin or other individual organs, it may also be a multisystem disorder with several manifestations and complications. There are 10 primary vasculitides based on vessel size (small, medium and large).[1]

The ABCs of GPA

GPA is an inflammatory disease that affects multiple organ systems and is believed to be autoimmune in origin.

Although it can occur almost anywhere in the body, GPA most frequently impacts the kidneys and respiratory tract, and the associated vasculitis has a predilection for smaller arterial blood vessels. GPA most commonly occurs around the age of 50 and is equally common in males and females. It occurs in around five to 10 cases per million people every year.[1,2]

Because typical cases of GPA show renal or respiratory signs, patients commonly present with symptoms similar to pneumonia or a sinus infection. GPA may also cause generalized symptoms of fatigue, a low-grade fever and weight loss. The diagnosis is based on the results of blood tests, radiologic (x-ray and computed tomography) findings and a thorough physical exam. A biopsy of the affected tissue can help confirm the diagnosis when necessary. Lab results typically show elevated ESR and CRP, and elevated anti–neutrophil cytoplasmic antibodies (ANCA) levels. ANCA testing with C-ANCA/PR-3 in particular shows high sensitivity and specificity for the disease.[1,3]

The prognosis in patients with GPA depends on when treatment starts and how much damage has already occurred at the time of diagnosis. It also varies depending on whether the GPA is C-ANCA or P-ANCA related, as higher levels of C-ANCA/PR-3 correlate with a higher mortality rate.[1,3,4] A patient treated before any damage can occur may be able to achieve remission. However, if left untreated, the survival rate is one to two years.[1,5]

The Eye in GPA

Ocular manifestations may be the presenting sign of GPA, as ocular involvement is between 29% and 79% in a patient's lifetime.[6] Findings are similar to those of other vasculitides and may include any of the following:[4,6]

- Blurred vision
- Necrotizing scleritis
- Uveitis (usually associated

with the scleritis)
- Keratitis
- Retinitis
- Retinal vasculitis
- Optic nerve head edema and atrophy
- Orbital disease
- Visual field loss (defects correlate with optic neuropathy)

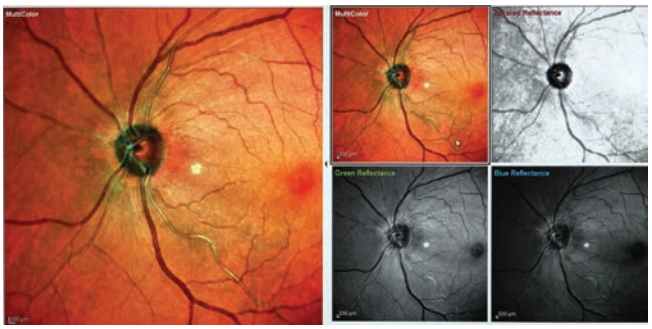

Fig. 2. One month after initiating treatment, the patient's disc edema and vasculitis have mostly resolved (the white spot temporal to the nerve is an artifact).

GPA Management

Treatment varies depending on the severity and the stage of the disease. GPA is categorized into two stages: the induction (or presentation) phase and the remission maintenance phase.

The presentation phase is further broken down into early systemic, generalized and severe stages, all of which are treated with immune suppressants and monoclonal antibodies.[1] Pharmacotherapies and methods may include methotrexate, cyclophospomide, rituximab, corticosteroids and, if the presentation is severe, plasma exchange.[1,7,8]

The remission maintenance phase includes major relapse, refractory disease and maintenance therapy. Patients in any of these stages can be placed on intravenous immunoglobulin or different monoclonal antibodies and immune suppressants.[1,8]

A patient diagnosed with GPA should be managed by an interprofessional team that includes ODs. As vital members of that team, optometrists can be involved with every step of the process from diagnosis to treatment and comanagement. GPA is serious but treatable and requires prompt diagnosis and treatment to prevent complications. ■

Dr. Zuercher is a 2018 alumna of University of the Incarnate Word Rosenberg School of Optometry, where she is now a resident in primary care optometry.

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Case Report

A 59-year-old black female presented with complaints of hazy peripheral vision in her left eye for the past week that was constant and had been worsening. Her medical history was positive for diabetes mellitus type 2, depression and hypertension. Her medications included metformin, Celexa (citalopram, Allergan), acetaminophen/codeine, bumetanide and trazadone. She reported symptoms of mild fatigue, shortness of breath and mild coughing.

Her visual acuities measured 20/20 OD and 20/20-2 OS. We documented a grade 1+ relative afferent pupillary defect in the left eye. Her confrontation fields showed mild generalized constriction in all four quadrants in the left eye. The dilated fundus exam revealed left optic disc edema with hemorrhage, a flat macula and arteriolar vasculitis. Threshold perimetry showed a mild superior rim defect in the right eye and a repeatable inferior and superior arcuate defect in the left eye, as well as an enlarged blind spot with slightly high fixation losses and high false positives. We took OCT and multimodal ophthalmic imaging (*figures 1 and 2*). Her lab results included several abnormalities:

- P-ANCA – *high*
- Anti-myeloperoxidase (MPO) Abs – *high*
- Rheumatoid arthritis factor – *high*
- C-reactive protein, quant – *high*
- Hemoglobin – *low*
- Hematocrit – *low*
- MCV – *low*
- MCH – *low*
- Platelets – *high*
- Monocytes – *high*
- Serum sodium – *low*
- Serum chloride – *low*
- Serum albumin/globulin ratio – *low*
- Plasma glucose – *high*
- Sedimentation rate, westergren – *high*
- EKG – *new left bundle branch block*

Due to the high level of P-ANCA with MPO, increased ESR and CRP, inflammation of the optic nerve head, vasculitis in the retinal arterioles and pneumonitis-like symptoms, we made a tentative diagnosis of Wegener's granulomatosis and started the patient on a steroid pulse of 80mg PO QD.

We communicated the results with her rheumatologist and cardiologist and scheduled follow up. One month later, we observed resolution of the optic nerve head edema and retinal vasculitis, although the visual field defect remains. Team-based care is ongoing.

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Through a Child's Eye

A teenage boy reported no vision problems, but his retina told a different tale.

By Alan Levitt, OD, and Mark Dunbar, OD

A 14-year-old boy from Jamaica was referred for the evaluation and management of retinal changes discovered in his right eye during a routine eye exam. When he presented a year earlier, everything was normal at that time. He reported no vision problems and says he's in excellent health and takes no medications. He was born full-term and is the youngest of three siblings.

On examination, his entering visual acuity measured 20/20 OD and OS. His extraocular motility testing was normal. Confrontation visual fields were full to careful finger counting OU and the pupils were equally round and reactive; no afferent papillary defect was detected and his anterior segment exam was unremarkable.

A dilated fundus exam of both eyes showed a small cup with good rim coloration and perfusion in both eyes. The macula appeared normal in both eyes. Changes were noted in the peripheral retina of the right eye (*Figure 1*). A widefield fluorescein angiography (FA) was performed and is also available for review (*Figure 2*). The optical coherence tomography (OCT) of the macula was normal in both eyes.

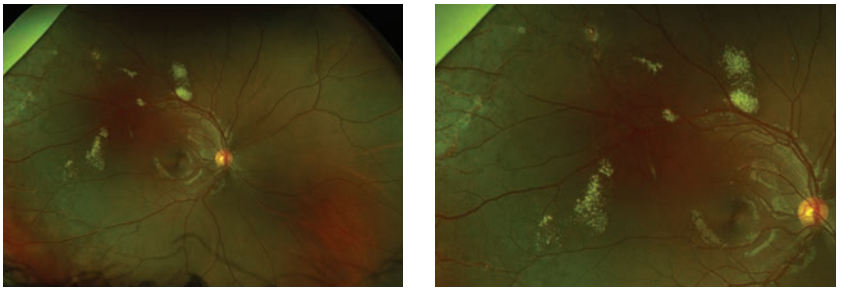

Fig. 1. This wide-angle montage view of the right eye shows peculiar retinal changes.

Take the Quiz

1. Which best describes the peripheral retinal changes based on the fundus photo?
a. Retinal neovascularization.
b. Aneurismal dilations and retinal telangiectasia.
c. Capillary angiomas.
d. Retinal arterial macroaneurisms.

2. What does intravenous FA show?
a. Capillary dropout.
b. Retinal neovascularization.
c. Quiet choroid with diffuse leakage.
d. Avascular retina with leakage and saccular dilations.

3. What is the correct diagnosis?
a. Resolving branch retinal vein occlusion.
b. Von Hippel disease.
c. Coats' disease.
d. Sickle cell retinopathy.

4. How should this patient be treated?
a. Observation.
b. Laser photocoagulation.
c. Intravitreal injection of triamcinolone.
d. Intravitreal injection of anti-VEGF medication.

Diagnosis

On the clinical exam, we noted a large area of exudate superior to the arcade, as well as temporal beyond the macula. Beyond the arcades, we noted numerous saccular aneurismal dilated vessels, as well as extensive smaller telangiectatic retinal vessels. The widefield FA highlights the extensive retinal telangiectasia, as well as extensive capillary dropout with peripheral areas of non-perfusion. The good news: even though we saw significant exudate and peripheral ischemia, we saw no exudative retinal detachment or macular edema. Of note, these retinal changes were limited to the right eye; the left eye was normal.

Based on the unilateral retinal changes and the fact our patient is male, our patient was diagnosed with Coats' disease.

Discussion

Coats' disease is an idiopathic vascular anomaly characterized by aneurismal dilations and telangiectasia of the retinal vessels.[1-2] It tends to be unilateral and occurs in males 90% of the time during the first to second decade of life.[1-2] The retinal capillaries tend to be most affected, but changes can also be seen in the major retinal vessels, particularly the arteries, as was the

case for our patient. These vessels leak fluid in the form of exudate. The hallmark of this condition is exudative retinopathy. The extent of involvement and degree of severity can vary.

In milder forms, only isolated areas of the retina may be involved and the prognosis is good. In severe forms, large areas of the retina are involved and patients can present with large amounts of exudate that can lead to an exudative retinal detachment or macular edema.

Investigators have stratified the disease into five main stages and further subclassified some stages (see *Table 1*).[3]

In a 2001 study of 124 eyes of 104 patients, the visual outcomes were good in stage 1—with no patients' visual acuities worse then 20/200 after treatment. In stage 2, 53% had vision worse than 20/200; however, among those with no foveal exudation, only 30% had a poor visual outcome.[3] For those with foveal exudation present in stage 2, 86% had a poor visual outcome.[3] In patients with stage 3 disease, 74% ended with visual acuity worse than 20/200.[3] All patients in stage 4 and 5 ended with visual acuity worse than 20/200.[3] Of note, only 18 patients had stage 1 or 2 disease, whereas 106 patients had stage 3 disease or higher by the time they were diagnosed.[3]

Based on these classifications, our patient would be categorized as stage 2a because no foveal exudation was noted. We expect a good prognosis, especially since he was diagnosed early in the course of the disease.

Treatment

Treatment for Coats' is indicated if the exudate is extensive and if it seems to be progressive. It is also indicated if central vision becomes affected due to macular edema, or in patients that develop retinal detachment.[1] Indirect laser photocoagulation is often a first-line treatment with the goal being to cauterize the affected retinal vessels.[4] Laser may also be used to create a barrier between normal and abnormal retina with hopes of sealing off the area of exudation.

Cryotherapy was once a mainstay of treatment but is not used as much anymore except as an adjunct or in more advanced cases. Intravitreal triamcinolone injections has been shown to reduce subretinal fluid and macular exudates and may even facilitate future treatments with laser photocoagulation. Finally, anti-vascular endothelial growth factor therapy may also have great success in reducing subretinal fluid and macular edema but it carries the risk of tractional retinal detachment from the treatment crunch-effect that occurs from regression of neovascularization and scarring that may develop.[5] It is, therefore, often reserved for more severe cases.

Vitreoretinal surgery may be required in more advanced cases that do not respond to traditional therapies. Even enucleation may be necessary. In one study, 20 eyes (16%) ultimately required enucleation.[3]

No immediate treatment was recommended for our patient; instead, he was scheduled to have an examination under anesthesia in approximately six weeks. At that time, treatment with indirect laser photocoagulation or an injection of intravitreal triamcinolone will be strongly considered. ■

Dr. Levitt is in private practice in Miami.

Table 1. Disease Classifications and Subclassifications

Stage	Clinical Findings
Stage 1	Retinal telangiectasia only
Stage 2a	Telangiectasia and extrafoveal exudation
Stage 2b	Telangiectasia and foveal exudation
Stage 3a1	Subtotal, extrafoveal, exudative retinal detachment
Stage 3a2	Subtotal, foveal, exudative retinal detachment
Stage 3b	Total exudative retinal detachment
Stage 4	Total retinal detachment and glaucoma
Stage 5	Advanced end-stage disease

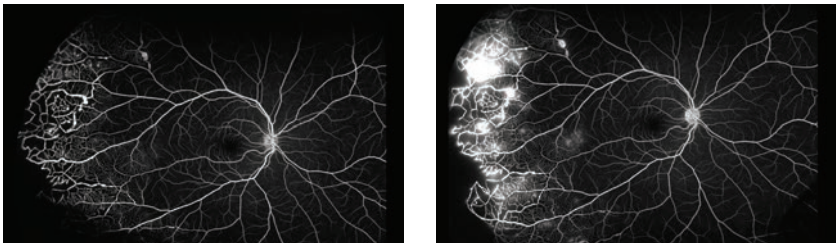

Fig. 2. These mid-phase and late phase fluorescein angiograms show our patient's peripheral retina.

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Anaphylaxis in Your Exam Lane

When a patient is gasping for air, will you be prepared?

By Matthew Krein, OD, Sarah Krein, OD, and Richard Mangan, OD

Anaphylaxis is an acute, potentially lethal multisystem syndrome. There are two primary anaphylactic reactions. One type, known as immunogenic, is a classic Type 1 hypersensitivity reaction most commonly mediated by IgE. The other, formerly called anaphylactoid, is triggered independently of an immune reaction.[1]

These two entities are indistinguishable clinically and the World Allergy Association has proposed referring to any severe, life-threatening, systemic hypersensitivity reaction as "anaphylaxis."[1] The World Allergy Association also proposes using "allergic anaphylaxis" when referring to immune-mediated reactions (IgE/immune complex) and "non-allergic anaphylaxis" when referring to immune-independent reactions.[1]

While the exact prevalence of anaphylaxis is unknown due to widespread underreporting, it is estimated between 1% and 2% of people worldwide are at risk for anaphylactic reactions.[2-3] Furthermore, the incidence of anaphylactic reactions appear to be increasing in the industrialized world.[1] Several known triggers for allergic anaphylaxis are shown in the literature, but any agent could potentially trigger an event and as many as 50% to 60% of cases are idiopathic.[1,4] The most common triggers are foods, particularly nuts/peanuts, fish/shellfish and mammalian dairy.[4] The second most common trigger is certain drugs, with beta-lactam antibiotics (penicillin and cephalosporin) and nonsteroidal anti-inflammatory drugs (NSAIDs) implicated.[1] Other common triggers are insect bite/sting, latex and exercise.[1,4-5]

This is the inside of an emergency kit used at Northeastern State University Oklahoma College of Optometry.

With 1% to 2% of the population at risk, the variety and ubiquity of triggering agents, the rising prevalence and lethal potential of allergic anaphylaxis it is critical for all health care workers, including optometrists, to understand how to recognize and triage a patient in anaphylaxis.

Pathophysiology

Allergic anaphylaxis is a classic IgE-mediated Type 1 hypersensitivity. An antigen triggers B-cells to produce antigen-specific IgE, the IgE coats mast cells and basophils, and these cells become activated. Additional antigen exposure crosslinks the bound IgE and triggers massive degranulation of mast cell and basophil cell contents. The systemwide effect of this massive degranulation causes the morbidity associated with anaphylaxis.

Two of the most important factors released are histamine and products of arachidonic acid metabolism (prostaglandins, leukotrienes and platelet-activating factor). These chemicals cause smooth-muscle contraction, vasodilation and increased vascular permeability.[1] Numerous neural proteases, proteoglycans and chemoattractants are also released, causing large-scale immune cell recruitment, further degranulation and magnification of the immune response and reduced cardiac function.[1,5-6]

While allergic anaphylaxis is a multisystem disorder, the primary organs affected during a reaction are those of the cardiovascular and respiratory systems. The principal causes of death from anaphylactic shock are

A convenient medical kit should include an epinephrine pen for anaphylaxis emergencies.

circulatory collapse and respiratory distress.[1] The extensive smooth-muscle contraction can affect any level of the airway and lead to bronchiole constriction of the lower airway and laryngeal edema of the upper airway, both of which can cause asphyxiation.[1] Extensive vasodilation and increased vascular permeability lead to massive extravasation of fluid out of the circulatory system, leading to a decrease in venous return, drop in blood pressure and clinical features consistent with hypovolemic shock.[1,3]

Diagnosis

Due to the multisystem nature of the reaction, symptoms may vary, and not all patients will present the same symptoms.

Any time you administer medication, particularly if administering parenterally, monitor patients for symptoms of anaphylaxis. Symptoms will typically begin within minutes, but could potentially take hours to manifest. Common skin/mucosa symptoms include itch, generalized flushing, hives, angioedema (swelling of lips, eyes, tongue). Symptoms of respiratory compromise include coughing, sneezing, runny or congested nose, tightness of throat or chest, dyspnea, changes in voice, hoarseness, stridor, wheezing and hypoxemia.[1,7] Symptoms of cardiovascular compromise include fainting, dizziness, sudden weakness, incontinence, chest pains, tunnel vision, difficulty hearing and tachycardia (heart racing) or bradycardia.[1,7] Other symptoms include cramping, nausea, difficulty swallowing, mood changes and headache.[1,7]

The key to successful treatment of allergic anaphylaxis is rapid diagnosis. The faster treatment is administered the better. In 2006, researchers developed guidelines to help simplify the diagnosing process (See "*Anaphylaxis Guidelines*").[7]

Management

The goal of in-office treatment for allergic anaphylaxis is to maintain cardiovascular and respiratory function. Prompt treatment is vital. Treatment should be catered to the severity of the reaction. If anaphylaxis is suspected, the first step is to have a staff member call 9-1-1. While calling, the patient should be given 0.2cc to 0.5cc 1:1000 epinephrine IM. This is most commonly administered with a preloaded injection pen on the lateral thigh. Epinephrine (adrenaline) causes venous dilation and increased cardiac contractility countering the effects of cardiac insufficiency. Additionally, it causes smooth-muscle relaxation countering airway constriction. Epinephrine injection can be repeated every 10 minutes to 30 minutes as needed.

In the face of anaphylaxis, there is no absolute contraindication for using epinephrine.[1] Following injection,

Anaphylaxis Guidelines

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissues or both (e.g., generalized hives, pruritus or flushing, swollen lips-tongue-uvula) And at least one of the following:

a. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow [PEF], hypoxemia).

b. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence).

2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):

a. Involvement of skin-mucosal tissue (e.g., generalized hives, itch-flush, swollen lips-tongue-uvula).

b. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia).

c. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence).

d. Persistent gastrointestinal symptoms (e.g., cramp/abdominal pain, vomiting).

3. Reduced blood pressure after exposure to known allergen for that patient (minutes to several hours):

a. Infants and children: low systolic blood pressure (age specific) or greater than 30% decrease in systolic blood pressure. Low systolic blood pressure for children is defined as less than 70mm Hg from one month to one year, less than (70mm Hg + [2 × age]) from 1 to 10 years, and less than 90mm Hg from 11 to 17 years.

b. Adults: systolic blood pressure less than 90mm Hg or greater than 30% decrease from their baseline.

immediately assess the patient's airway, breathing and cardiac function (known as the "ABCs"). Administer CPR/BLS, including the use of an AED, if needed.

Assuming the patient is breathing and their heart is beating, continue in-office treatment. Place the patient in the Trendelberg position (lay on back with feet 15 to 30 degrees above the heart) to combat hypotension and maintain perfusion of vital organs. If the anaphylaxis stems from a medication injected into a limb, apply a tourniquet to that limb. Administer a combination of H1- and H2-blocking antihistamines. Diphenhydramine (Benadryl) 25mg to 50mg by mouth for H1 receptors. If the patient is unable to swallow, administer the drug subcutaneously or intramuscularly. Ranitidine (1mg/kg/dose) or cimetidine (adult 5mg to 10mg/dose) can be given by mouth, or parenterally if the patient cannot swallow.

If significant bronchospasm occurs, treat with two puffs of inhaled β-2 agonist (albuterol). While treatments are administered, monitor the patient's vitals at least every five minutes until emergency medical help arrive. Due to the widespread systemic response and the large amount of chemotaxis of immune cells, relapse of symptoms is not uncommon. The patient may require additional monitoring and possible treatment in the emergency department. Such efforts may include IV fluids to reduce hypovolemia, IV epinephrine 1:10,000, vasopressive agents and systemic steroids to reduce inflammatory relapse.

Allergic anaphylaxis is a serious and life-threatening condition. With the prevalence at 1% to 2% of the population and the flow of patients through our practices, it is likely you will encounter it. As health care professionals, Optometrists are responsible for not just visual well-being but the health and wellness of the entire patient. For this reason, we need to be prepared to recognize and confront anaphylactic shock efficiently and effectively to save more than just the patient's eyesight. ■

Drs. Krein and Krein are assistant professors at Northeastern State University in Oklahoma.

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■ **8-10.** *Annual Palm Beach Winter Seminar*. PGA National Resort & Spa,West Palm Beach, FL. Hosted by: Palm Beach County Optometric Association. Key faculty: Walter Whitley, Steven Ferrucci, Joseph Sowka, Barry Frauens. CE hours: 21. For more information, email Tamara Maule at pbwinterseminar@gmail.com, call (561) 477-3524 or go to www.2019pbws.eventbrite.com.

■ **9-16.** *Tropical CE–Secrets Akumal*. Playa del Carmen, Mexico. Hosted by: Tropical CE. Key faculty: Carlo Pelino, Derek Cunningham. CE hours: 20. For more information, email Stuart Autry at sautry@tropicalce.com, call (281) 808-5763 or go to www.tropicalce.com.

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

a) **Teaching**
- Developing and delivering lectures
- and/or laboratories for related areas, as assigned;
- Embracing and enhancing the didactic philosophies in the O.D. program;
- Maintaining and expanding the high quality clinical practice environment for optometry students on rotation;
- Precepting students on clinical rotation at the Midwestern University Eye Institute where applicable;

b) **Service**
- Helping to maintain and grow the state of the art optometry program with a strong interdisciplinary focus that meets the needs of patients in the surrounding community; is efficient, patient friendly, and cost-effective;
- Working closely together with all optometry and ophthalmology faculty to provide a complete range of eye and vision care services;
- Participating in leadership roles in state, regional, and national optometry organizations;
- Participating on College and University committees, as assigned;
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c) **Scholarly activity**
Engaging in research and scholarly activity, including presentations at scientific meetings, research, and publication in peer reviewed journals sufficient to qualify for academic advancement in a non-tenure or tenure track position.

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

Salary will be commensurate with qualifications and experience
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Contact information: Interested applicants should apply online at www.midwestern.edu and include curriculum vitae and letter of interest specifying the position and college that he/she wishes to be considered for. Application packet should include curriculum vitae and letter of interest. Inquiries may be directed to Dr. Melissa Suckow, Dean; Midwestern University: msucko@midwestern.edu.

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Diagnostic **Quiz**

Lost in a Fog

By Andrew S. Gurwood, OD

History

A 27-year-old Caucasian female reported to the office emergently with a chief complaint of painful vision loss in her left eye for a duration of two days. She explained that her eye became red and painful with "foggy" blurring of her vision.

Her systemic history was remarkable for migraine headaches—for which she had just started the medication Topamax (topiramate, 25mg) PO BID.

Her ocular history was unremarkable and she denied exposure to chemicals or allergies of any kind.

Diagnostic Data

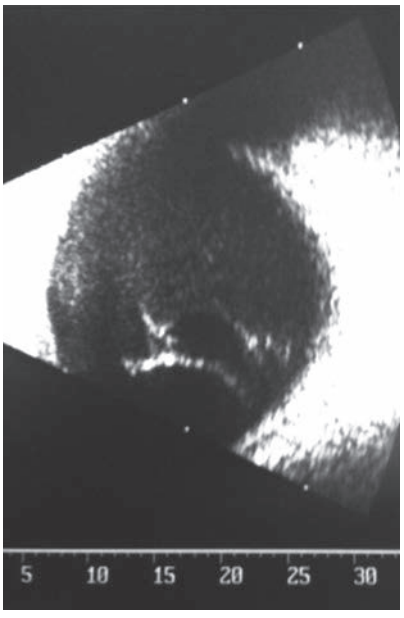
Her best corrected entering visual acuities were 20/20 OD and 20/200 OS at distance and near. Her external examination demonstrated grade III conjunctival injection, tearing with mild corneal clouding in her right eye. No evidence of afferent pupil defect was seen. The biomicroscopic examination of the anterior segment of the right eye demonstrated conjunctival injection, corneal bullae, a narrow angle with shallow anterior chamber and evidence of cell and flare grade II.

The left eye was normal. Goldmann applanation tonometry measured 55mm Hg in the right eye and 15mm Hg in the left eye.

The B-scan of the patient's right eye is available for review.

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 do you think is the most likely diagnosis? What is the patient's likely prognosis? To find out, visit us online at www.reviewofoptometry.com. ■



This B-scan shows the right eye of our 27-year-old patient who had "foggy," blurring vision.

Retina Quiz Answers *(from page 68):* 1) b; 2) d; 3) c; 4) b.

Next Month in the Mag

In February, *Review of Optometry* presents its Annual Innovations in Eye Care issue.

Topics include:

- *These Glasses Lower IOP for Glaucoma Patients—Really!*
- *Can a Contact Lens Treat Diabetic Retinopathy?*
- *DARC: A Radical New Way to Diagnose Glaucoma and Other Retinal Neurodegenerative Diseases*
- *What to Expect from Iontophoresis Drug Delivery For Uveitis, Dry Eye and Post-Cataract Care*

Also in this issue:

- *Managing Anterior Uveitis with Steroids, NSAIDs and More*
- *How Are You Faring with Your Plaquenil Screenings?*

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