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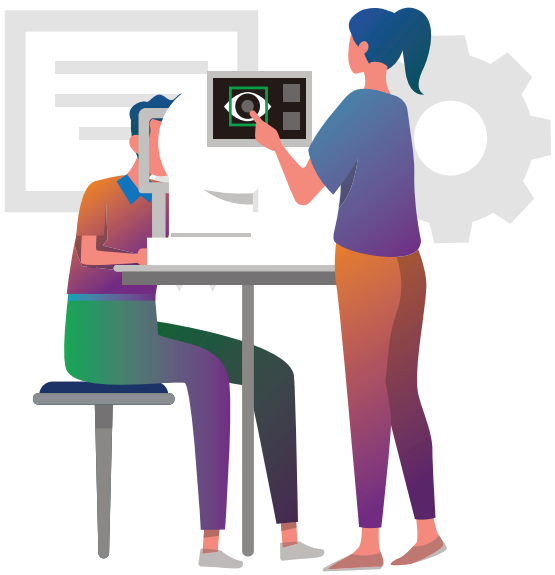

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Treatment for Mild Amblyopia Effective Half the Time

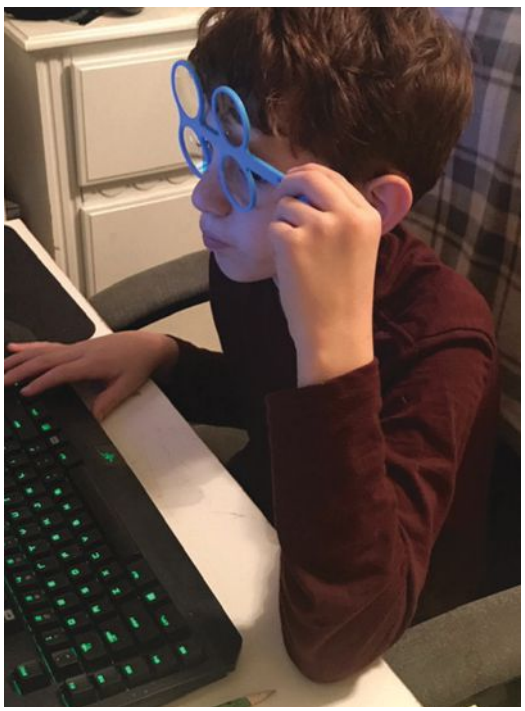
These patients—including a third of those treated in the past—achieved 20/20 vision and improved stereopsis.

Clinical studies on amblyopia typically only include patients with three or more lines of visual acuity difference between eyes, or roughly 20/20 in one eye and 20/40 or worse in the other—the official Academy of Ophthalmology cut-off for diagnosis. However, these analyses exclude patients who are identified as having unexplained vision loss despite failing to meet the traditional amblyopia criterion. Aiming to shed some light on the incidence and treatment of this so-called "subthreshold amblyopia," researchers recently evaluated a patient population at Boston Children's Hospital. They found that, when treated, half of these patients achieved 20/20 vision in both eyes and improved stereopsis, suggesting that intervention is still worthwhile for parents of affected children to consider.

In the single-center analysis, the team included data on a total of 2,311 amblyopic patients aged two to 12 diagnosed at the hospital during a four-year period. Of these, 20.1% had what this research team characterized as subthreshold amblyopia, the majority having an amblyogenic factor (61.7%)—most commonly anisometropia (32.8%). The average follow-up among this cohort was 3.1 years, and 97.5% received treatment.

The researchers reported in their paper on the study, recently published in the *American Journal of Ophthalmology*, that among the three-quarters of patients with subthreshold amblyopia who returned for follow-up, "47.8% achieved resolution, including 55.7% of treatment-naïve patients, and 62.5% (five of eight patients) offered observation alone." They added that the "median stereopsis improved by 0.4 logMAR in those who achieved resolution, with no change in those with persistent amblyopia." A multivariate analysis of the data also showed a significant association between a longer follow-up duration and the resolution of subthreshold amblyopia (odds ratio: 1.38).

Photo: Marie Bodack, OD



Amblyopia patients with vision better than 20/40 in one or both eyes can still achieve 20/20 vision after treatment in approximately 50% of cases, study finds.

Of the patients with subthreshold amblyopia, 41.1% had received prior treatment elsewhere, referred to in the study as having "residual" amblyopia. Fewer of these patients improved with treatment compared with those who were treatment-naive; however, 33.9% still achieved 20/20 vision in both eyes by their final visit. "This is in line with recent studies investigating visual acuity gains in patients treated for residual amblyopia that found 22% to 28% of patients improved 0.2 logMAR or more," the researchers explained in their paper.

As this data shows, children who don't meet the diagnostic criteria for amblyopia may still benefit from treatment, with nearly half in this cohort recovering 20/20 vision and achieving improved stereopsis, including a third of whom were treated in the past. For affected patients, this could mean opening up "a broader range of future occupations, some of which have a minimum requirement of 20/20 corrected visual acuity in both eyes," the researchers pointed out.

The team suggests that future studies should assess whether treatment vs. observation alone would result in similar outcomes.

Michalak SM, Chinn RN, Shoshany TN, et al. Subthreshold amblyopia: characterization of a new cohort. Am J Ophthalmol. December 18, 2022. [Epub ahead of print].

20-20-20 Still Not Enough to Alleviate Eye Strain

A break from near work is good, but the parameters of this suggested exercise lack statistically significant evidence to confirm its positive impact as an intervention, recent evidence suggests.

Given that success in today's society requires children to perform sustained near work most days of the week, the only workable option to help curb near work-related myopia development/progression is to suggest periodic breaks. Practitioners commonly recommend the 20/20/20 rule with hopes that, if patients follow it, they will reduce their risk of myopic progression. However, the results of a recent study provide little or no support for the use of 20-second breaks to alleviate symptoms of digital eye strain.

The research determined that, if the goal of the work breaks is to reduce both the accommodation and vergence responses following sustained fixation on a near object, then 20 seconds of distance viewing may not be sufficient enough to allow these sustained responses to dissipate fully.[1] A commentary on the same topic (but not this specific recent study) that looked at current animal model data also suggests repeated episodes of 20 seconds were ineffective at reducing myopia development, and instead indicated that sustained breaks of five or more minutes every hour are needed to negate myopiagenic effects.[2] Both were published recently in *Optometry and Vision Science.*

The study evaluated the effect of different break schedules during the course of a highly demanding word search task. Following the 20-20-20 rule, individuals were advised to fixate on an object at least 20 feet away for at least 20 seconds every 20 minutes. This research was carried out on 30 young subjects who performed a 40-minute cognitively demanding reading task from a tablet computer. The task was undertaken on four separate occasions, with 20-second breaks allowed every five, 10, 20 or 40 (*i.e.*, no break) minutes, respectively. Before and after each trial, subjects completed a questionnaire on ocular and visual symptoms experienced during the session. Reading speed and task accuracy were also quantified during each trial.[1]

A significant increase in post-task symptoms (with respect to the pre-task value) was observed for all four trials. However, there was no significant effect of scheduled breaks on reported symptoms, reading speed or task accuracy.

"These findings should not be interpreted as evidence that taking breaks isn't helpful," the authors of the first study wrote in their paper. "Rather, it seems likely that longer break durations or a different frequency of breaks may be required to produce significant effects," they explained.

"Providing more specific instructions on appropriate fixation targets during the break intervals would likely be helpful, rather than a simple statement of look into the distance," the researchers suggested. While participants were asked to look out of the window at a distant target about 20 feet away during the break interval, one cannot be certain that they actually were focused on the far stimulus. Because the participants were not asked to view or comment on a specific detail of the distant target, the researchers believe this may have represented a poor stimulus to accommodation and vergence, especially during the later stages of the trial when subjects knew they would be returning to the reading task.

The team noted that even simply having the subject close their eyes during the break interval, rather than having them fixate on a distant target, might be beneficial. "This allows a layer of tears to be spread over the anterior surface of the cornea, increasing the subject's comfort," they explained.[1]

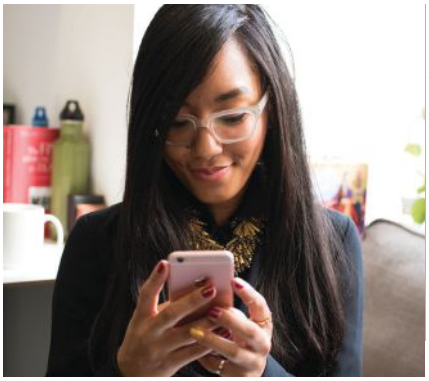


Photo: Getty Images

Researchers from this study suggest that shorter, more frequent breaks from near work may be more effective than 20-second breaks every 20 minutes.

Longer Break Times Needed

The commentary was a clinical perspective on recent myopia research. These researchers' review of myopia animal model paradigms led them to propose that the ideal break would not just consist of not doing near work but also include going outside and looking at distant objects for a minimum sustained break of five or more minutes. The data suggested that instead of taking a 20-second break every 20 minutes, it may be more effective to take a five-minute break every hour. However, the second research team believes that breaks likely do not need to be as closely spaced as every 20 minutes.[2]

"Clearly more research needs to be done, both in further determining the temporal parameters of anti-myopia stimuli, especially in species more closely related to humans, and also in targeted epidemiological work that looks more carefully at how the pattern of children taking breaks from near work correlates to the development and progression of myopia," the commentary authors wrote.

1. Johnson S, Rosenfield M. 20-20-20 rule: are these numbers justified? Optom Vis Sci. 2022. [Epub ahead of print].

2. Pucker AD, Gawne, TJ. Fighting myopia with intermittent near-work breaks: 20 seconds every 20 minutes might not be enough time. Optom Vis Sci. December 5, 2022. [Epub ahead of print].

Daily Disposable Wearers Understand Risks but Don't Modify Behavior in Response

Study shows that more individualized patient education may help curb improper usage with this modality, such as lens reuse and overnight wear.

Since the 1950s, a concept called the health belief model has been used in a wide variety of medical fields and populations to explain patterns of health-related behaviors. The premise is that, for a person to take action to improve or maintain their health, they need to believe they are personally susceptible to the disease in question and that it would negatively affect them. In a recent study, researchers used this model to understand the attitudes and beliefs of daily disposable soft contact lens wearers.

A total of 100 daily disposable wearers between ages 18 and 33 were enrolled and asked about demographics, lens wear and various aspects of their beliefs and actions germane to the health belief model. The team found an association between health belief survey scores and education level, overnight lens wear and lens reuse.

Of the study participants, 37% reported sleeping in their contact lenses, which may reflect the wearing habits of a young adult population, or a changing demographic of lens wearers as daily disposable lenses become more widely prescribed, with 25% reporting reusing them.

As the study authors predicted, these lens wearers saw fewer benefits and more barriers to following recommended health actions. But to their surprise, they found that soft lens wearers who reported sleeping in lenses perceived themselves as more susceptible to contact lens–related adverse events.

"This suggests that patients may recognize that certain health behaviors are risky but do not modify their behaviors, conceivably because they do not believe that the consequences of high-risk behavior will individually happen to them," the authors explained in their paper on the work.

They suggested that providers consider the permeability of the lenses they are prescribing for their daily disposable patients, as well as the potential for corneal inflammatory events since many may be sleeping in the lenses.

Although contact lens wear is widely considered to be a safe and effective way to manage refractive error, the authors noted that adverse events still occur and are more likely with improper use. Providers should reinforce best practices and "in the context of the health belief model, reinforce the benefits of wearing daily disposable soft contact lenses as prescribed."

The team concludes by suggesting that the health belief model "allows providers to identify opportunities for patient education and interventions to promote healthy contact lens wear."

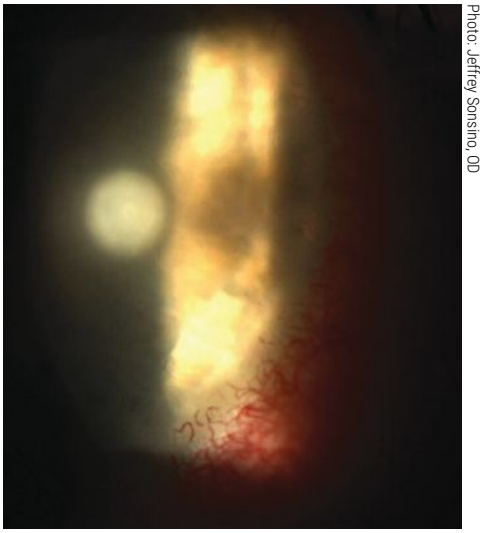


Photo: Jeffrey Sonsino, OD

Not replacing soft contact lenses as directed led to this case of infiltrative keratitis. Education should stress not just the possibility of adverse effects but the individual's vulnerability as well.

Lutmer KM, Wagner H, Richdale K, et al. Examining daily disposable soft contact lens wearers' attitudes and beliefs using the Health Belief Model. Ophthalmic Physiol Opt. December 4, 2022. [Epub ahead of print].

IN BRIEF

DR Nomogram Predicts VA Loss. Retinal structural abnormalities are associated with diabetic retinopathy severity and vision loss. In a recently published paper, **researchers scored various retinal risk factors correlated with vision loss in DR to create a nomogram that predicts visual impairment.**

They retrospectively enrolled patients who had undergone OCT-A, fundus fluorescein angiography and swept-source OCT of the macula to observe retinal blood flow, stage disease and define retinal structures. The researchers considered visual acuity ≥0.5 logMAR as impaired. They screened characteristics that correlated with visual acuity using binary logistic regression. These factors were input into a multivariate binary stepwise regression to create a nomogram, which was then validated.

In total, the researchers analyzed 29 parameters and 13 characteristics for their model. They reported that **the following were statistically significant: diabetic macular ischemia grade, disorganization of the retinal inner layers, outer layer disruption and vessel density of the inferior aspect of the choriocapillaris layer.**

The validated model had an area under the curve of 0.931. The researchers noted in their paper that analysis confirmed that risk threshold probabilities can be used as clinical practice guides. The clinical impact curve of the model shows the proportion of individuals at risk at each threshold probability.

Importantly, the study used patient data from a single visit, so the **"model can only predict the current risk of visual impairment and not changes in visual acuity at a future time,"** the researchers pointed out in their paper. They recommended future prospective studies with external validation to improve the model.

Zhao Y, Yu R, Sun C, et al. Nomogram model predicts the risk of visual impairment in diabetic retinopathy: a retrospective study. BMC Ophthalmol. 2022;22:478.

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New Findings Link Kidney Disease to DME

Study documents subretinal fluid in patients with renal disorders and identifies vascular hyperpermeability as the common mechanism.

While diabetic macular edema comes (DME) with the risk of developing subretinal fluid (SRF), the association between DME, abnormal renal profiles and development of SRF is less well explored. One new study examined just that, looking at potential risk factors for fluid development and linking it with renal function, since diabetic kidney disease and DME may share similar microvascular pathophysiology.

The retrospective study included 66 DME patients and the researchers evaluated systemic and renal parameters along with ocular factors. Systemic parameters included hypertension, glycosylated hemoglobin, serum fasting glucose diabetic kidney disease and others. Renal parameters included serum albumin, albuminuria and estimated glomerular filtration rate. OCT measured for central subretinal fluid thickness and presence of SRF.

Main findings from the research included a higher albuminuria level association with the presence of SRF in DME patients and lower serum albumin levels associated with an increase in SRF thickness.

In line with these results, the researchers suggest in their paper published in *Ophthalmology* that "diabetic kidney disease plays an important role in the occurrence of SRF in DME." They additionally mention that higher albuminuria had a better association with SRF presence than HbA1c levels, but both were in fact important risk factors when determining SRF presence. Even further, serum albumin was not significantly different between patients with and without SRF.

As for why a negative correlation was observed between SRF severity and serum albumin levels, the researchers postulate that the connection may be due to a shared pathogenic mechanism between the kidney (albuminuria) and the eye (SRF), that being vascular hyperpermeability.

As pressure gradients determine fluid movement, lower intravascular osmotic pressure and higher hydrostatic pressure may result from marked protein loss seen in advanced proteinuria. As such, fluid retention of the subretinal space would occur, and in early stages, increased albumin molecule production in the liver would serve as a compensatory mechanism for deficits of serum albumin. At later stages, though, the serum deficits that could not be compensated for, could be associated with the observed SRF thickness.

The researchers believe fluid leakage from choroidal vessels may be more prone to patients with albuminuria, as well as a damaged retinal pigment epithelium, leading to the SRF seen in DME.

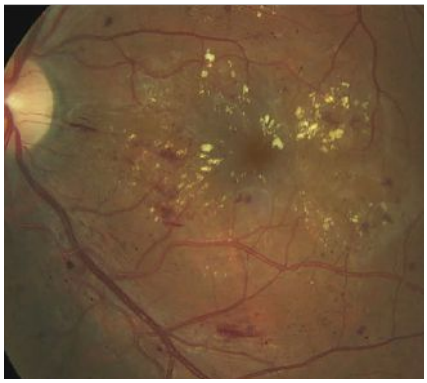

Photo: Jarett Mazzarella, OD

Serum protein loss in kidney disease raises hydrostatic pressure, causing fluid retention in the subretinal space that can lead to DME, the researchers posited.

Finally, they point out that a lower HbA1c level was more likely to result in SRF, contrary to what some might think. This may be due to an early worsening effect caused by rapid HbA1c level reduction.

Because of the apparent connection between DME and renal capacity, the authors of the study advise that "screening for SRF in DME in all patients with diabetic kidney disease should be emphasized" and screening "patients with higher albuminuria and lower serum albumin levels should be routinely performed."

Zhang X, Hao X, Wang L, Xie L. Association of abnormal renal profiles with subretinal fluid in diabetic macular edema. J Opthalmol. December 13, 2022. [Epub ahead of print].

IN BRIEF

Systolic Hypertension May Pose Modifiable Risk Factor for POAG. In an effort to better pinpoint the association between systemic blood pressure and incident primary open-angle glaucoma (POAG) using a large open-access database, **researchers recently found that higher systolic blood pressure and pulse pressure were associated with an increased risk of incident POAG.**

The prospective cohort study included 484,268 participants from the UK Biobank without glaucoma at enrollment who were followed for a median of 12 years. Incident POAG events were recorded through assessment visits, hospital inpatient admissions and primary care data. Blood pressure measures included systolic, diastolic, pulse and mean arterial pressure.

There were 2,390 incident POAG events over 5,715,480 person-years of follow-up. Multivariable analysis showed that, compared with systolic and pulse pressure in the normal range (systolic: 120-130mm Hg, pulse: 40-50mm Hg), higher systolic and pulse readings were associated with an increased risk of incident POAG. Specifically, **systolic blood pressure of 130-140mm Hg or 140-150mm Hg was associated with a 1.16x higher risk of incident POAG, whereas a pulse pressure of greater than 70mm Hg was associated with a 1.13x higher risk.** No statistically significant associations were found between diastolic or mean arterial pressure and incident glaucoma.

"Systolic hypertension may thus represent a potential modifiable risk factor for POAG, although further studies are required to better characterize these associations," the study authors wrote in their paper.

Macri C, Wong CX, Tu SJ, et al. Blood pressure measures and incident primary open-angle glaucoma. Invest Ophthalmol Vis Sci. 2022;63(13):3.

First COVID-19 Vaccine Dose Increases Uveitis Flare Risk

COVID-19 vaccines are generally safe and their protective effects typically outweigh potential side effects, but certain populations with pre-existing disease should take a more guarded approach. A new study showed an increased risk of uveitis flare following vaccination. This risk was highest among those with previous recurrence, chronic uveitis and a shorter period of quiescence.

The retrospective study identified participants with uveitis from the Inflammatory Eye Disease Registry. The rate of flare was calculated for three months prior to and three months after each vaccination. Uveitis flare was defined as new or increased uveitis activity that required a treatment change. In total, 3,008 patients (4,184 eyes) were included, with a total of 8,474 vaccines given during the study period (median age: 55 years; 49% female).

Noninfectious etiology was most common, occurring in 76.3% of patients, with infectious etiology in 23.7%. The rate of uveitis flare was 12.3 per 1,000 patient months at baseline, 20.7 after the first dose, 15.0 after the second, 12.8 after the third and 23.9 after the fourth. The median period of quiescence prior to flare was 3.9 years. An increase in uveitis flare was seen both in infectious uveitis (13.1 at baseline compared with 20.2 after first dose, a 154% increase) and noninfectious uveitis (12.4 at baseline compared with 20.9 after first dose, a 169% increase).

Risk factors for uveitis flare were identified to be recurrent uveitis, chronic uveitis, a shorter period of quiescence and the first dose of the COVID-19 vaccine. Median time to uveitis flare was 0.53 months following the first vaccination, 1.74 months following the second and 1.35 months following the third.

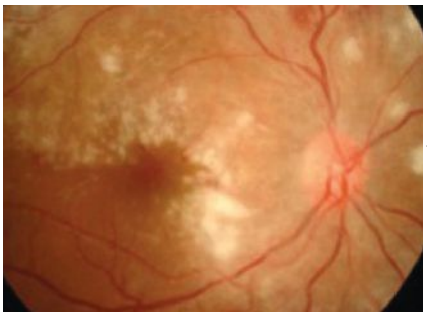

Photo: Michael Trottini, OD

Uveitis incidence followed the COVID vaccine more often among those with previous recurrence and chronic uveitis, study finds.

Jordan CA, Townend S, Allen N, et al. Navigating COVID-19 vaccination and uveitis. Ophthalmol. December 16, 2022. [Epub ahead of print].

DR Lasting Beyond Five Years Tied to Dementia, Alzheimer's

Upon investigating if associations between diabetic retinopathy (DR) and dementia and Alzheimer's disease (AD) remain significant after adjusting for diabetes severity, researchers found that among people with type 2 diabetes (T2D), DR and resulting glycemic and renal complications seem to be an important biomarker of dementia risk.

This retrospective cohort study included 536 adults ≥65 years who were dementia-free at enrollment and followed biennially until incident dementia developed. Participants either had or developed T2D.

The team found significant associations between DR of greater than five years duration and both dementia and AD. "The strong association," the researchers wrote in their paper, "despite controlling for several markers of diabetes severity suggests that the factors accounting for this association may be specific to the retina and the brain." Although the pathophysiologic mechanisms differ considerably between AD and non-AD dementia, a history of DR more than five years "appears to be an important biomarker associated with increased risks of both AD and all-cause dementia development," they explained.

Damage to the neurovascular unit, including the inner blood-retinal barrier (BRB) and the blood-brain barrier (BBB), may be a key mechanism underlying both DR and dementia, the researchers suggest. "The breakdown of the BRB is a feature of DR, and BRB pathology has also been found in AD. Additionally, BBB breakdown has been implicated as an early event in the AD pathology cascade." Citing a prior study, the authors explained, "T2D patients with retinal neuro- and vasodegeneration were at higher risk for rapid cognitive decline and AD," while those without these retinal pathologies had lower rates of cognitive decline.

"These findings suggest the course of DR in diabetes patients may provide useful information on dementia risk," the authors concluded. "A deeper understanding of the causal pathways underlying the association between DR >five years and dementia could offer useful insights on dementia."

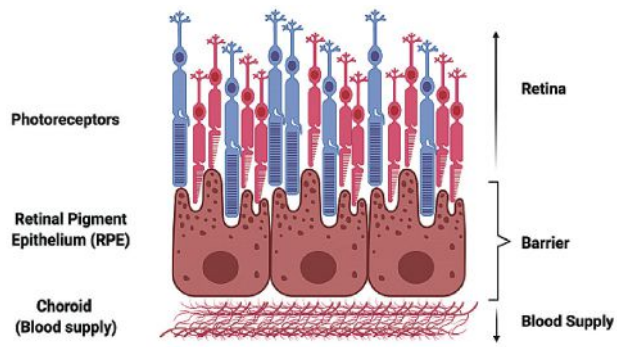

Photo: Uni College London Institute of Ophthalmology

Damage to the blood-retinal barrier is present in both DR and several forms of dementia, likely leading to the association found in this study.

Lee CS, Krakauer C, Su YR, et al. Diabetic retinopathy and dementia association, beyond diabetes severity. Am J Ophthalmol. December 10, 2022. [Epub ahead of print].

AAOph Gives Cautious Endorsement to Hysteresis

A literature review concluded that it complements tonometry and offers additional nuance in clinical assessment but can be difficult to untangle from other factors at play.

While IOP is central to the diagnosis and treatment of glaucoma, its significance is likely overstated, due to the dearth of other easy-to-obtain biomarkers. For years, proponents have argued that corneal hysteresis adds much-needed nuance to the clinical apprehension of IOP and hence the patient's glaucoma status. To help, the American Academy of Ophthalmology recently weighed in, issuing one of its Ophthalmic Technology Assessment papers on the topic.

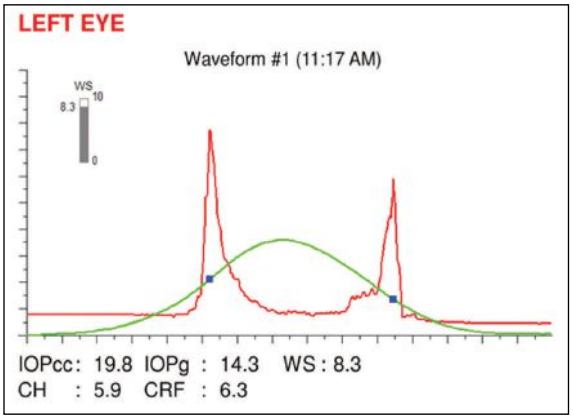

Photo: Sarah B. Klein, OD

Although various forms of glaucoma can result in lower hysteresis values, interpretation of corneal hysteresis measurements in an individual patient is complicated by IOP effects, study finds.

An expert panel reviewed published literature on the utility of corneal hysteresis in diagnosing glaucoma and assessing the disease's progression in existing patients with glaucoma. Data was gathered through searching peer-reviewed literature in the PubMed database. Included in the retrospective analysis were 19 articles, rated for level of evidence by the panel methodologist. Eight were rated level I and five were rated level II.

The researchers broke the articles down into two general categories: corneal hysteresis use in glaucoma screening and in disease development/progression. Corneal hysteresis values between normal and glaucomatous eyes were further subdivided into different glaucoma types, including primary open-angle glaucoma (POAG), primary angle-closure glaucoma (PACG), pseudoexfoliative glaucoma, normal-tension glaucoma (NTG) and ocular hypertension.

After reviewing the articles included, the researchers came to find that corneal hysteresis was lower in patients with POAG, PACG, pseudoexfoliative glaucoma and pseudoexfoliation syndrome when compared with normal, control eyes. Even further, lower hysteresis values were associated with an increased risk of disease progression.

Determining the significance of a lower observed corneal hysteresis is complicated by patients with higher IOP or who are on topical hypotensive medication because these confounding factors influence the parameters of corneal hysteresis measurements. Despite this, corneal hysteresis was additionally observed to be lower in treatment-naïve, NTG patients compared to controls with similar IOP.

Out of the five articles that looked at POAG, four indicated level I evidence and four found that POAG patients had lower corneal hysteresis compared with controls. Four articles compared corneal hysteresis with PACG patients, all providing level I evidence. Within this subgroup, limitations existed between with different IOP levels between groups and the effects that topical hypotensive medications may have on hysteresis.

Three articles were included looking at corneal hysteresis in psedoexfoliative glaucoma or pseudoexfoliation syndrome, all providing level II evidence. All three found lower corneal hysteresis with pseudoexfoliative glaucoma, while patients with pseudoexfoliative syndrome (without glaucoma evidence) displayed higher corneal hysteresis than pseudoexfoliative glaucoma patients but lower corneal hysteresis than normal controls.

Two articles were included in the NTG subgroup, both providing level I evidence. This subgroup also proved interesting because untreated NTG can be matched to healthy eyes for IOP, effectively getting rid of confounding factors of IOP and topical hypotensive medication. With that possibility, both articles found lower corneal hysteresis in NTG compared to controls.

Despite the consensus across all subgroups displaying lower corneal hysteresis, the authors caution that "interpretation of corneal hysteresis measurements in an individual patient is complicated by IOP effects as well as medical, laser and surgical therapy, as well as other influencing parameters such as age, central corneal thickness and axial length."

The authors still posit that, "nevertheless, most evidence suggests that the corneal hysteresis measurement is a potential adjunct in identifying glaucoma patients and those who may be at increased risk for disease progression" and that the measurement of corneal hysteresis "complements current structural and functional assessments in determining disease risk in glaucoma suspects and patients."

Sit AJ, Chen TC, Takusagawa HL, et al. Corneal hysteresis for the diagnosis of glaucoma and assessment of progression risk. Ophthalmol. December 16, 2022. [Epub ahead of print].

Alcohol Consumption Linked to Higher Glaucoma Rates

This held true for all categories of intake level, and with no protective benefit observed with lower intake.

Though drinking in moderation can provide some health benefits, one new study to appear in *Ophthalmology Glaucoma* outlines that this is not the case when considering glaucoma and its related traits. The study sought to clarify any association between alcohol consumption and glaucoma as well as assess whether a genetic predisposition to glaucoma modified the association.

The researchers conducted a retrospective, cross-sectional study using data from the UK Biobank. They performed Mendelian randomization (MR) experiments to probe causal effects of the substance.

A total of 173,407 glaucoma patients were included. Of those, data for IOP was included for a total of 109,097 participants and the researchers had access to 46,236 macular OCT scans. Researchers compared the categories of self-reported alcohol consumption (never, infrequent, regular and former drinkers), then assessed a dose-response effect for the regular drinkers. Then, researchers assessed if any associations were modified by a multi-trait polygenic risk score for glaucoma.

They found that regular drinkers displayed both a higher IOP and thinner macular ganglion cell-inner plexiform layer (mGCIPL) thickness when compared to infrequent drinkers. Regular drinkers' alcohol intake was associated adversely with all outcomes dose-dependently. The MR analyses concluded a causal relationship with mGCIPL thickness.

Those with a stronger genetic susceptibility to developing glaucoma had much stronger alcohol-IOP associations. Alarmingly, researchers found that alcohol intake observed with adverse effects was at a lower level than the current UK and US guidelines.

Photo: Stanislav Ivanitskiy on Unsplash

Compared to infrequent drinkers in this study, regular drinkers had a higher IOP and thinner mGCIPL.

Following that, there was no observed protective association with any outcome, which is interesting, given that there's evidence of neuroprotective properties of polyphenols, found in high concentration in red wine, which also contains anti-inflammatory and antioxidant compounds. The researchers propose this might be due to the detrimental effects of alcohol on glaucoma outweighing any of its potential benefits, even at low intake.

Alcohol Use Categories in This Study

- Never drinkers (no past or present use)
- Infrequent drinkers (special occasions only)
- Regular drinkers (1-3x/month or greater)
- Former drinkers (no current use but have previously)

The researchers weigh in on the potential biological mechanisms underpinning the association. One theory involves the association representing a combination of causative factors linked to chronic alcohol usage, including biomechanical and physiological differences as well as neurodegenerative, cardiovascular and endocrine disorders. In this way, it may not be a single mechanism contributing to the observed association.

The authors point out in their paper that "while we cannot infer causality definitively, these results are of interest to people with or at risk of glaucoma and their advising physicians. The presence of an underlying causal association may have important clinical and public implications and lead to targeted lifestyle recommendations for glaucoma," they conclude.

Stuart KV, Luben RN, Warwick AN, et al. The association of alcohol consumption with glaucoma and related traits: findings from the UK Biobank. Ophthalmol Glauc. December 5, 2022. [Epub ahead of print].

IN BRIEF

■ ***Patient Height Inversely Associated with Steep Cornea.*** A recent study published in *Cornea* assessed the associations of anthropometric features with the presence of steep cornea. The team reported a strong inverse relationship between height (adjusted for weight) and steep cornea in the overall population and in women specifically. **For each one-inch increase in height, there was a 16% lower odds of steep cornea in the overall population and a 20% reduction in women. For each one-inch increase in height, there was also a 0.10D decrease in corneal refractive power in the overall population, independent of gender.**

The researchers assessed these relationships using logistic regression models with steep corneas defined by corneal power ≥48D as used in previous studies due its high specificity and sensitivity for ectatic corneal disease. The study included participants from the US National Health and Nutrition Examination Survey. The team found 171 cases with a mean dioptric power ≥48D. **There was no significant association between BMI or weight and steep cornea. Height had the strongest association with steep cornea** even after adjusting for education, race and socioeconomic status.

The team had no data on nutrition and physical activity—two formative factors that influence height and weight. They believe this information may be needed to better understand their role in this association.

"Further study of the relationship of height with steep cornea may yield mechanistic insight into the pathogenesis of corneal ectasias and genetic correlations," the team concluded in their paper.

Valluru G, Henick D, Klawe J, et al. Anthropometric measures and their relationship to steep cornea in the US population. Cornea. December 13, 2022. [Epub ahead of print].

FEATURES

REVIEW OF OPTOMETRY • Vol. 160, No. 1 • JANUARY 15, 2023

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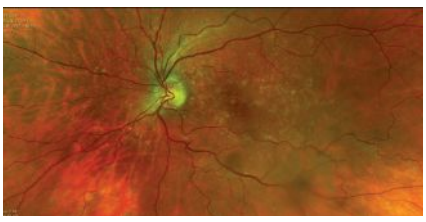

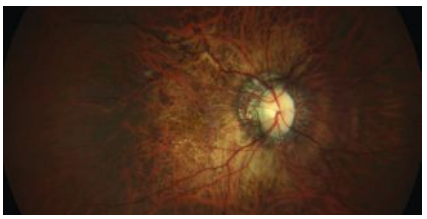

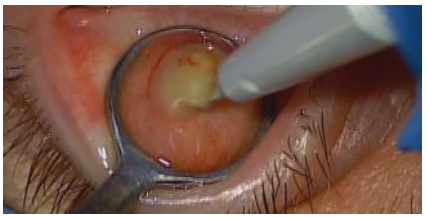

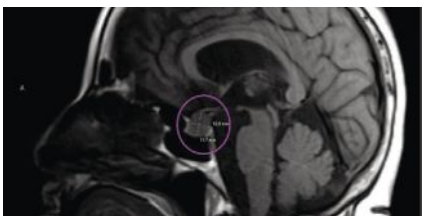

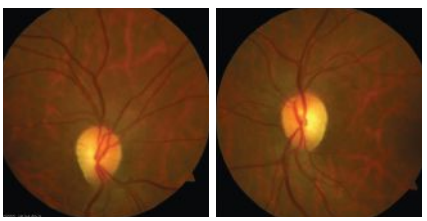

We Welcome Your Comments

Feedback from within the community provides important insights about clinical practice. If you would like to share your thoughts on the topics discussed in this issue—or the wider field of optometry at large—write to: ***editor@reviewofoptometry.com***

Prevent AMD Vision Loss with Digital Healthcare

IRIS REGISTRY

20/83 VA

Average at wet AMD diagnosis according to IRIS Registry real-world data[1]

HOME STUDY

≥20/40 VA

Average at wet AMD diagnosis with ForeseeHome[2]

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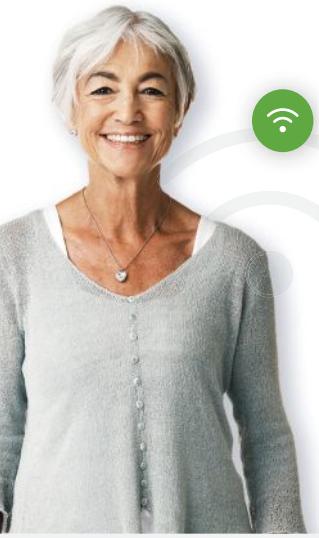
- Strengthen your referral relationships with qualified wet AMD referrals



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BY JACK PERSICO
EDITOR-IN-CHIEF
OUTLOOK

Knowing is (Only) Half the Battle

Sophisticated devices like an OCT will give you a wealth of data on your patient. But you're the one who decides what to do with it.

When we surveyed readers last summer about their priorities for new technology purchases, OCT topped the list: 32% of respondents said they planned to buy one within a year. And since 67% percent of those responding said they *already* had an OCT, it's safe to say this technology is well on its way to being standard operating equipment in optometric practices.

To help newcomers and experienced users alike, we've put together this special issue, which includes a whopping 40 editorial pages devoted to OCT technology and its role in clinical practice. Weighing in at 18,000 words and over 100 images, this special collection of articles is, I daresay, "a keeper." In the pages to follow, over a dozen expert clinicians will walk you through the buying decisions and countless clinical applications of eyecare's Swiss Army knife.

One thing to keep in mind, however, as you dig in to this massive collection of material: technology alone is never enough. You're the one who has to make the call. Much has been written about "red disease" and "green disease" in OCT—those false positive and false negative readings that can lead your clinical judgment astray, usually because the so-called normative database the device compares your patient to lacks appropriate representation of the population's ethnic heterogeneity and the influence of age differences. In fact, Andrew Rixon points out in his article this month on OCT for glaucoma that those are better thought of as "reference" databases, since "normative" is a loaded and misleading word.

But the devices themselves can also just flat-out produce garbage if scans are done incorrectly or if concomitant pathology obscures the structure being imaged. As Henrietta Wang points out on page 30, "The guiding principle in all of healthcare is *primum non nocere*, or 'first, do no harm.' While we usually think of that in a treatment context, this can also be applied to the diagnostic techniques we employ on a day-to-day basis. If the device itself introduces errors, this can undermine the clinical care we provide by confounding accurate diagnosis." In the first article of this series, Dr. Wang shares a guide to avoiding OCT artifacts that we encourage you to tear out or download for use in your practice.

Of course, even when everything goes perfectly and you receive a beautiful scan and accurate analytics, the machine doesn't tell you what to do with it all. Nor should it. People sometimes worry that the increasingly high-tech nature of medicine is going to make doctors glorified techs who administer tests but leave the mental heavy-lifting to computers. Far from it—all that added diagnostic nuance that OCT brings requires you to up your game in how to interpret and make sense of it. There are countless clinical and personal factors you need to synthesize to come up with a management plan that is clinically prudent and also a pragmatic reflection of insurance constraints, patient motivation and other external factors.

So, the OCT may have earned its place alongside the phoropter and slit lamp, but it's still just there to feed data into the most high-powered computer you'll ever use: your brain. ■


ENOUGH
IS ENOUGH
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ADVANCING REFRACTIVE SURGERY

Strategies for EVO ICL Patient Success

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Scottsdale, AZ

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Summit Eye Care
Milwaukee, WI

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Prism Eye Institute & TLC Laser Vision
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A New Refractive Surgery Option: EVO ICL™

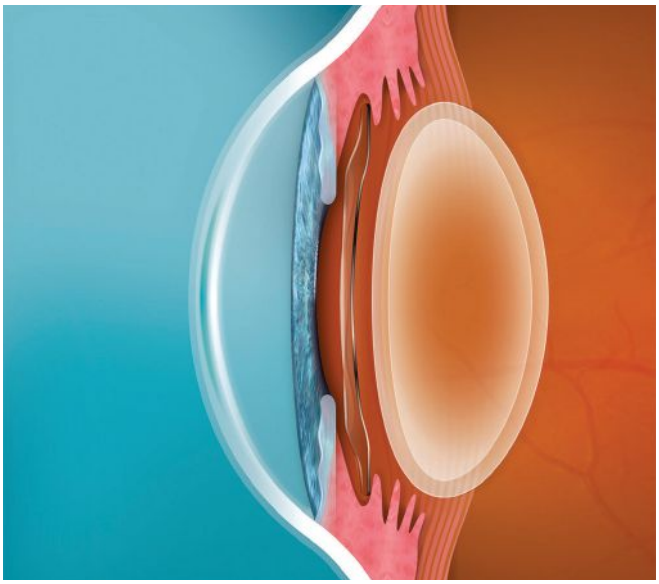
By Marc R. Bloomenstein, OD, FAAO

Interest in refractive surgery is booming again as the huge Millennial generation reaches their prime refractive surgery years. As Millennial patients, who are now age 25 to 40, come to our offices seeking freedom from glasses or contact lenses, it's important to be aware of new options. Also, consider the 6 million frequent replacement contact lens wearers that drop out each year in the U.S. alone.*

The recently FDA-approved EVO ICL™ from STAAR Surgical is a phakic IOL designed to be implanted behind the iris and in front of the natural lens. Surgeons around the world have already been using the EVO ICL for 10 years, so there is a significant body of literature demonstrating outstanding safety and effectiveness and very high rates of patient satisfaction.[1] In a survey of 1,542 patients implanted with the EVO ICL, 99.4% said they would elect to have EVO surgery again.

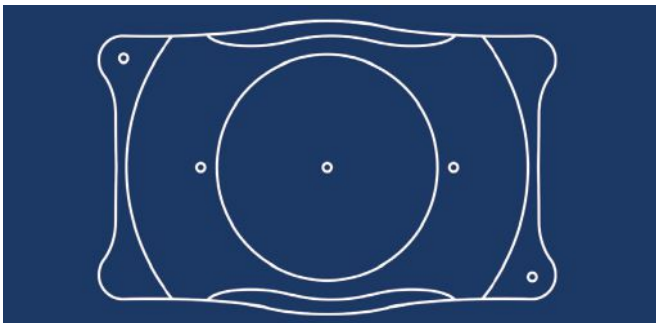
EVO ICL is made from STAAR's Collamer® material, a copolymer of poly-HEMA and collagen that offers UV protection and excellent biocompatibility.[2,3] The Collamer material, used in both the EVO ICL and its predecessor, the Visian ICL, now has a proven history of more than 20 years and more than 2 million ICL lenses sold worldwide.

A significant barrier to implanting Visian phakic IOLs in the past was that they required a separate procedure, a peripheral iridotomy (PI), usually performed 1-2 weeks before the lens implantation. PIs can be uncomfortable and carry some risk of IOP elevation, pupillary block, angle closure, or postoperative glare. The new EVO ICL has a full-thickness, 0.36 mm-diameter central port designed to allow physiologic aqueous flow through the lens. The central port eliminates the need for a preoperative PI. The other ports in the footplates and perioptic area, and the axis alignment marks facilitate orientation and alignment of the lens.



Who Are Candidates for ICL?

In my opinion, the EVO ICL should be considered for any moderate or higher myope. By correcting vision very close to the nodal point of the eye, it offers superb optical results.



The lens is indicated for phakic patients aged 21-45 with stable refractions who are seeking correction or reduction of myopia, with or without astigmatism, with SE from -3.0 to -20.0 D and cylinder from 1.0 to 4.0 D at the spectacle plane. The surgeon will check for an appropriate endothelial cell count and anterior chamber angle and depth to confirm candidacy.

For our very high myopes, EVO ICL is typically the only refractive surgery option. Increasingly, though, we are seeing it becoming the refractive procedure of choice for or many myopes, including any patient with thin or compromised corneas, preoperative dry eye concerns, topography unsuited for laser vision correction, a projected high rate of tissue removal with laser vision correction, or any other concerns around creating a flap.

The EVO ICL procedure doesn't induce or worsen dry eye[4] syndrome or night vision problems[5] and there is no risk of corneal ectasia.[6] Another significant advantage is that the EVO ICL is removable by a surgeon, if necessary. It doesn't change the corneal curvature or make IOL power calculation more challenging in the future. We all know post-LASIK patients who are just learning that they don't qualify for premium IOLs, or can't be assured of good outcomes, due to the difficulty of making accurate IOL calculations without the preoperative corneal measurements. The EVO ICL preserves the opportunity for these patients to achieve the best possible outcomes with whatever IOL technologies are available in the future.

EVO represents an important evolution of phakic IOL technology and an exciting new chapter in refractive surgery for our patients.

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*clspectrum.com/issues/2019/july-2019/where-have-all-of-the-contact-lens-wearers-gone

EVO ICL Outcomes: What To Expect For Your Patients

By Nicholas J. Bruns, OD, FAAO

The outcomes I see in our practice from the EVO ICL represent a tremendous step up from its predecessor, the Visian ICL. With the new EVO ICL, recently approved for use in the U.S., we are seeing the same outstanding levels of visual quality, with a much lower chance of side effects or complications. And with no requirement for preoperative peripheral iridotomies, the procedure is easier for patients and clinicians alike.

I used to think of the ICL as a great option for patients who weren't candidates for corneal refractive surgery—in other words, those whose myopia was too high or whose corneas were too thin for LASIK. Over time, my threshold for who is a good candidate has changed considerably. Today, I would argue that the overlap between EVO ICL and LASIK is larger than most practitioners realize.

FDA Clinical Trial Results

There are several ways to judge the results of refractive surgery procedures. The simplest is the visual acuity. For the 619 eyes implanted with the EVO ICL in the FDA clinical trial available for analysis, the mean uncorrected visual acuity (UDVA) 6 months after surgery was -0.059 logMAR, or 20/17 Snellen acuity.[1] UDVA was 20/16 or better in 58.5% of eyes and 20/20 or better in 87.6% of eyes. That's an amazing achievement, especially considering that patients started out with significant

Figure 1. Predictability

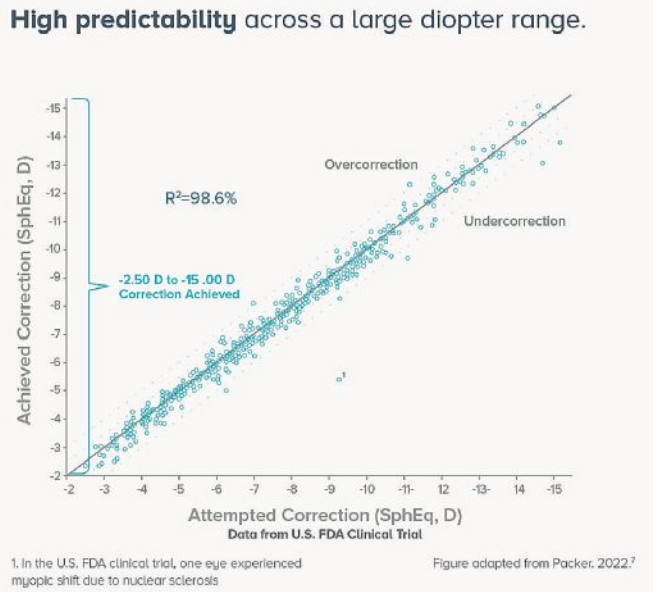
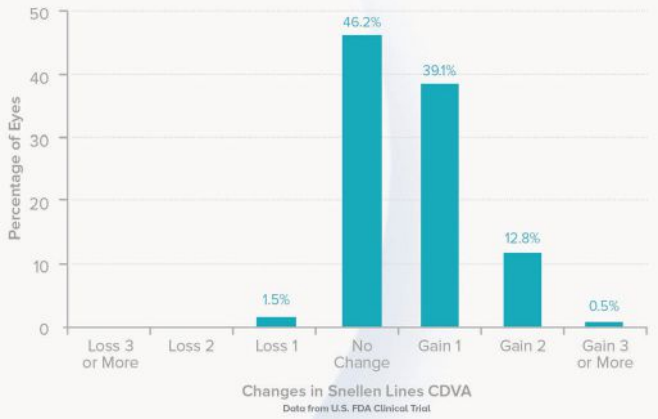


Figure 2. Change in Corrected Distance Visual Acuity



refractive error: Mean preop manifest refraction spherical equivalent (MRSE) in the study was -7.62 D, with myopia up to -15.00 D and cylinder up to +4.00 D.

The predictability of the correction was also excellent, with 90.5% of eyes within 0.50 D of the target refraction and 98.9% within 1.00 D (Figure 1).[1] What I have seen clinically was reflected in these trial results. While patients see very well shortly after surgery, the refractive outcome improves during the first postoperative month and remains very stable thereafter. The results of the 6-month FDA study also mirror what has been published in the international literature with much longer followup.[2]

What impressed me the most about the clinical trial results was that more than half the patients (52.3%) gained one or more lines of corrected distance visual acuity (CDVA)(Figure 2). I attribute this gain in lines of vision to the optical advantages of correcting vision so close to the nodal point of the eye. In our experience, it is difficult to achieve the same results with glasses or contact lenses.

Quality of Vision and Safety

Quality of vision also matters a great deal with elective refractive procedures. Patients have been dissatisfied with some technologies in the marketplace despite objectively good visual acuity, because they had poor quality vision under certain conditions. From the literature, we know that EVO ICL provides improved mesopic contrast sensitivity from preop to 6 months, with and without glare.[3] Moreover, the induction of higher-order aberrations (HOA) is low to nonexistent. There is no induction of spherical-like aberrations, and very low induction of coma-like and total HOA (0.05 and 0.04 µm, respectively).[4]

Finally, I am impressed by the very low rate of adverse events. In the FDA clinical trial, there were zero cases of pupillary block or anterior subcapsular cataract.[1] Only one eye implanted with a toric EVO ICL required surgical repositioning due to residual astigmatism. While there were some cases of transient IOP increase due to retained OVD or a postoperative steroid response, there were no IOP spikes related to blockage of aqueous flow through the central port, angle narrowing, pigment dispersion, or inflammation.[1]

We have experienced excellent refractive outcomes with no adverse events since introducing the EVO ICL in our practice in April. Both the safety and efficacy outcomes we have experienced, along with the published literature, give me great confidence in recommending EVO ICL as a refractive surgery option for a wide range of patients.

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1. Packer M. Evaluation of the EVO/EVO+ sphere and toric Visian ICL: Six month results from the United States Food and Drug Administration clinical trial. Clin Ophthalmol 2022;16:1541-53.

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Pearls For Successful EVO ICL Management

By Anu Ondhia, OD

With the recent FDA approval of the EVO ICL family of lenses, American optometrists will start seeing many more patients implanted with this technology. This presents new opportunities for collaborative care that are well within the capabilities of any optometrist who is currently, or is interested in, co-managing surgical cases.

The first step is to ascertain which patients are ideal candidates for this lens and what to expect in terms of refractive outcomes. As with any new technology, this can mean challenging your historical criteria and flow to keep up and evolve in the best way. Expectations for monitoring and follow-up should be reviewed with the surgeons to whom you refer. Patients will typically be seen at the surgery center for their Day 1 postop visit, then often return to their optometrist for the 1-week or subsequent follow-up visits, depending on surgeon recommendations.

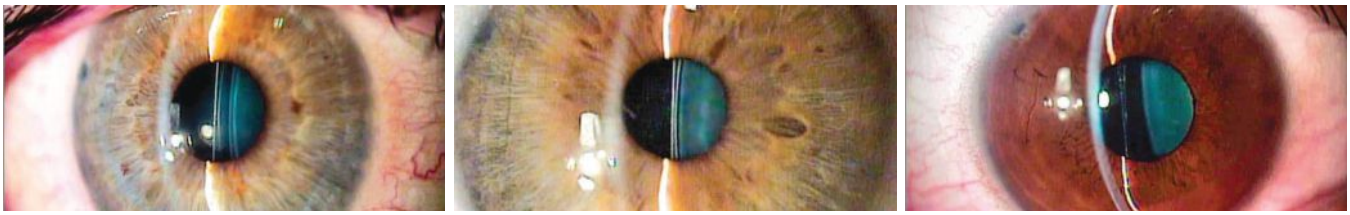
Here are four key elements for evaluation in ICL cases:

Intraocular Pressure (IOP)

A spike in intraocular pressure can occur following any surgical procedure. An IOP spike early in the postoperative period (Day 1-2) is rare, but is usually due to incomplete removal of ocular viscoelastics (OVD). When this occurs, you may see high pressure, and the patient may experience some ocular pain or nausea, although they could also be asymptomatic. In our practice, the surgeon would typically perform an anterior chamber paracentesis, or 'tap,' and release the extra fluid, followed by oral or topical pharmaceutical use to manage. The patient would then be followed within a few days to a week to ensure pressure has not become elevated again. In the rare case when an IOP spike occurs, an open line of communication with the surgeon regarding next steps is advisable.

Lens vault

Evaluation of the lens vault, the distance between the posterior surface of the EVO ICL and the anterior surface of the crystalline lens, is important and may take some practice to be able to judge accurately.



Figures 3, 4, and 5. Normal vault (3), Shallow vault (4) and High vault (5).

Vault should be assessed at every postoperative visit.

The optimal vault is 250-900 µm, or approximately 50% to 150% of the corneal thickness.[1] In the absence of symptoms, a shallower vault may be acceptable.[2,3] If the angle is closing or an anterior subcapsular cataract (ASC) is seen, removal may be necessary. Fortunately, these events are not commonplace. The occurrence of cataract with the current model is very rare. There were no cases (0.0%) of ASC in the FDA clinical trial for the EVO ICL at 6 months and none reported in a review of the worldwide literature covering 4,196 eyes implanted with EVO ICLs.[4] If you are concerned that the vault is too shallow, check the iridocorneal angle gonioscopically and then examine the crystalline lens carefully.

As with anything, there is a learning curve with comanaging ICLs and particularly assessing optimal vault. I would encourage you to work closely with your preferred surgeon and surgical center optometric colleagues to build efficiency and confidence in your new skills as well as seek secondary opinion and guidance. It will all be second nature before you know it!

Endothelial cell density

A major concern with earlier phakic IOL models was the potential for increased rate of endothelial cell loss. Given that the EVO ICL is a posterior chamber lens that sits in the ciliary sulcus, behind the iris, as opposed to being iris fixated as in older ICLs, endothelial touch is highly unlikely. In the FDA clinical trial of the EVO ICL, mean endothelial cell density declined 2.3 ± 4.0% from preoperative to 6 months. Two papers evaluating endothelial cell loss in ICL eyes found no significant change in endothelial cell density over 5 years.[5,6]

Other surgical complications

Post-ICL implantation, it is important to monitor patients for any surgical complications that should be referred back to the operating surgeon for management. Although, complications can occur after any intraocular surgery.

Post-Operative Observations

Optometrists can easily co-manage ICL patients with a skilled surgeon. During the early postoperative period, some patients may describe a ring-shaped dysphotopsia.[7] This symptom rarely persists beyond the first few weeks after surgery.

Post-operative patients, particularly former high myopes, typically experience a huge wow factor quite often, with gains noted in best-corrected vision. These near-instant outcomes are not dissimilar to those experienced with LASIK, in my opinion. A very high percentage (99.4%) of ICL patients surveyed have said they would choose to have the procedure again. The EVO ICL is available in a wide range of available powers, to meet the refractive needs of our patients. Based on data from the U.S. clinical trial and from international experience, we know that complications are exceedingly uncommon, and that the quality of vision patients can achieve with this technology is unparalleled. For all these reasons, I find it very gratifying to manage EVO ICL patients peri-operatively and trust that you will enjoy expanding your comanagement offerings, as well. ●

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BY PAUL M. KARPECKI, OD
CHIEF CLINICAL EDITOR

Bouncing Back

It looks like 2023 could be a blockbuster year for optometry.

In many ways, 2022 was tough for our profession, but a new year brings hope for a major turnaround—and it's very likely to occur. One reason is that optometry is the top prescribing profession of topical ophthalmic medications, accounting for almost 75% of presbyopia drop prescriptions and almost 70% of dry eye medications.

The good news: 2023 serves up more potential and exciting FDA approvals than any recent year. Let's quickly review the year that was and then take a broad look at the great therapeutic opportunities 2023 should bring for you and your patients.

A Year of Loss

By most standards, 2022 can be epitomized as a year of great loss, including the profession's leaders to finances and everything in between. Optometry practices showed little to no growth and salaries look to be the same or less compared to the previous year. Loss of staff was a major theme impacting growth in optometric practice, research recruitment and even optical sales.

We lost two icons in the profession, Art Epstein and Stuart Richer. Dr. Epstein, a frequent author and educator, had a great following. His online publication, *Optometric Physician*, has some of the highest readership of any online product to date. Dr. Richer was known as the "godfather of nutrition" and one of the most intelligent physicians in our profession. Both will be greatly missed for years to come.

Hope on the Horizon

We have many reasons for hope in 2023, including more potential ophthalmic therapeutic approvals than any year in history. There are an unprecedented six new drug applications likely to achieve FDA approval, including two for dry age-related macular degeneration (AMD)/geographic atrophy (GA), three for dry eye disease (DED), one for blepharitis and possibly an additional one for myopia management.

> **"It's time to bounce back from 2022 and get ready for all the possibilities that lie ahead for you and your patients."**

• ***Dry AMD/GA.*** Drug candidates for these diseases are showing a significant slowing of progression with hints that long-term treatment could potentially halt further cell loss completely. It's possible that early and continuous treatment could stop the advancement of any remaining viable cells in the dreaded diagnosis of AMD.

• ***DED.*** The first of these promising therapies is NOV03 (Bausch + Lomb), which appears to inhibit evaporation 80 times greater than human meibum, and by mixing with a patient's existing meibum, it creates an anti-evaporative layer that lasts four to six hours. It also was found to reside in meibomian glands for more than 24 hours.

The second therapeutic is Cycl-A-Sol (Novaliq), a sister product to NOV03. It contains 0.1% cyclosporine, giving the drop a combination of a novel and comfortable vehicle with an effective immunomodulator. It has been shown to provide faster improvement in signs and symptoms, including corneal staining.

• ***Blepharitis.*** TP-03 (Tarsus Pharmaceuticals), the first drug specifically for *Demodex* blepharitis, showed an improvement in clinically meaningful collarette cure rates of almost 90%, compared to 33% for the vehicle after six weeks of BID treatment. It also demonstrated statistically significant mite eradication and lid erythema improvement. *Demodex* blepharitis may affect over 20 million Americans, giving this therapeutic the potential to serve many patients as a drug for DED.

Later in the year, we may see reproxalap (Aldeyra Pharmaceuticals) with a first-ever RASP (reactive aldehyde species) inhibitor. RASP are reactive molecules that covalently bind to cells, disrupting their function and activating pro-inflammatory mediators. It's an upstream approach, much like steroids, but without the risks. It seems to affect tear production in addition to multi-level inflammation control, potentially making it one of the fastest-acting dry eye therapeutics.

Finally, although likely in early 2024, it is possible we could see 0.1% atropine commercialized (Vyluma) as a prescription option for myopia management in pediatrics. This is something to keep our eye on.

It's time to bounce back from 2022 and get ready for all the possibilities that lie ahead for you and your patients. This year may go down as the most prosperous year in eye care history for novel therapeutics, optometry practices and patient success in the areas of dry AMD/GA, DED and *Demodex* blepharitis. ■

About Dr. Karpecki

Dr. Karpecki is the director of Cornea and External Disease for Kentucky Eye Institute, associate professor at KYCO and medical director for the Dry Eye Institutes of Kentucky and Indiana. He is the Chief Clinical Editor for Review of Optometry and chair of the New Technologies & Treatments conferences. A fixture in optometric clinical education, he consults for a wide array of ophthalmic clients, including ones discussed in this article. Dr. Karpecki's full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.

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FOUNDATION FIGHTING BLINDNESS

FightingBlindness.org

BY MONTGOMERY VICKERS, OD

CHAIRSIDE

Pen to Paper

First, I'll teach you what a letter is. Then, I'll show you how to write one.

We don't write enough letters. In fact, these days we don't write any letters at all. And it's really too bad, isn't it?

When I open my mailbox… you know, the one that is used by the US Postal Service? (I put that in here for the young doctors who thought I meant Gmail or something). When I open my mailbox, it is filled with catalogs, grocery store flyers, offers for cheaper insurance and, at my age, stuff about Medicare and Social Security. Yuck is right.

> **I can't remember the last letter I received that wasn't sent to tell me about something I should buy or someone I should vote for. My guess is that you feel the same way I do.**

Oh, I get a bill every once in a while too, but this happens very rarely since everything is set to autopay these days. It's sad that the most personal thing I get is some random bill I forgot to pay.

I can't remember the last letter I received that wasn't sent to tell me about something I should buy or someone I should vote for. My guess is that you feel the same way I do, so rather than write to each of you, I decided to send you something herein (Mad Libs style).

Tempting Templates

Letter to anyone.

Dear _____,

Just checking in to see how your _____ is going. Were you able to _____ after all? I know sometimes _____ can be a _____, but I just wanted you to know that, in me, you will always have someone who thinks you're a _____. Don't forget to get your _____ checked every year. They are irreplaceable.

Your _____,

_____

Letter to your patient.

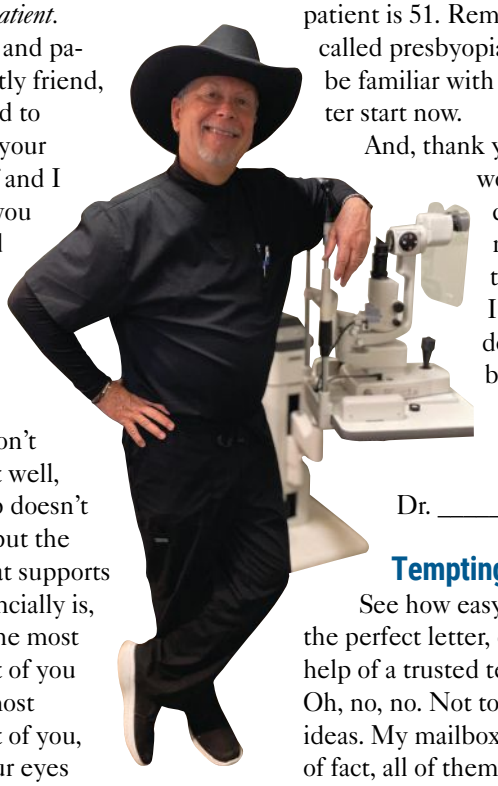
Dear friend and patient, but mostly friend,

I just wanted to thank you for your trust. My staff and I enjoy having you as a friend and patient, but mostly a friend. Although, when I think about it, I actually don't know you that well, and friendship doesn't pay the bills, but the part of you that supports our office financially is, to us all, not the most important part of you because the most important part of you, to us all, is your eyes since that's the reason you come to see us, which makes all of us happy to also be your friends.

OK, whew! Stay in touch.

Sincerely,

Dr. _____



Letter to your staffers.

Dear staffers,

For some of you, I have an idea! Smile. It won't crack your face, I promise. If you already smile, I have another idea! Be serious. What you do is very important and not funny. Posterior detachments matter, I promise.

For my techs, I have an idea! At least dress like you have an iron at your house. Please tell me you don't sleep in your scrubs.

For my opticians, I have an idea! Don't give me a blank stare when I say the patient needs computer glasses. You remember computers, right? I know you're 30, but the patient is 51. Remember that thing called presbyopia? Trust me, you'll be familiar with it soon enough. Better start now.

And, thank you for your hard work. I really appreciate it very, very much! Donuts are in the break room, but I have an idea! You don't have time for a break. What are you thinking, anyway? Get back to work.

Much love,

Dr. _____

Tempting Takeaways

See how easy it is to simply write the perfect letter, especially with the help of a trusted template or three? Oh, no, no. Not to me. Don't get any ideas. My mailbox is full. As a matter of fact, all of them are. ■

About Dr. Vickers **Dr. Vickers** received his optometry degree from the Pennsylvania College of Optometry in 1979 and was clinical director at Vision Associates in St. Albans, WV, for 36 years. He is now in private practice in Dallas, where he continues to practice full-scope optometry. He has no financial interests to disclose.

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†To limit blurriness when using contact lenses, remove contacts, apply drops, then insert contacts.

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Not Cut and Dry

Optometrists must continue to discuss the risk of conversion with patients in the early stages of AMD.

Q A 66-year-old patient with dry age-related macular degeneration (AMD) presented with new symptoms. What are the best next steps to evaluate, educate and manage?

A "Just knowing the right diagnosis is not enough to ensure the best care for our patients," says Jeff Gerson, OD, of Grin Eyecare in Kansas City, KS. "We need to let them know what to expect and how things could progress on the journey ahead of them."

The patient that was being followed for dry AMD was using a digital home monitoring system. She was called and asked to come in for an evaluation because the system sent an alert. Dr. Gerson uses Notal Vision ForeSee Home on all patients with intermediate AMD. It is approved for intermediate AMD with vision of 20/50 or better.

Stay on Top of AMD

When Dr. Gerson called her, she said that her vision seemed fine, but she came in the next day and her best corrected vision was 20/30 in the affected eye, down from 20/25. Examination with optical coherence tomography (OCT) revealed that her left eye had converted from dry to wet AMD. A choroidal neovascular membrane with subretinal fluid was noted.

"We discussed the findings, happy that this was being detected before anything catastrophic had occurred," Dr. Gerson says. "While in the office, we called the retina specialist we trust and scheduled an appointment for her the next day, not leaving it to chance."

When evaluating patients with a progressive eye disease like AMD, it's crucial to remain aware of clinical signs that may indicate potential advancement. Educate all patients with AMD on symptoms of exudative conversion and the need for prompt examination, typically within 24 hours of onset.

To differentiate between stages of AMD, recall that the AREDS defined intermediate AMD as being any retinal pigment epithelium changes or the presence of drusen 125µm or more in diameter.[1]

Dr. Gerson discussed the patient's prognosis and what the retina doctor would do regarding diagnosis and treatment. He also reviewed the research that had shown that vision at time of diagnosis helps predict prognosis.[2]

Treatment

We discussed that anti-VEGF injections are the mainstay of wet AMD treatment, and that the worst part of the injection is the anxiety beforehand. I told her that most people will have a few loading dose injections, and then it is likely that with the new medications, specifically Vabysmo (faricimab, Genentech), injections will be able to be spread out to every 12 to 16 weeks. Also, just-released data from the PULSAR study has shown that 8mg Eylea (aflibercept, Regeneron) will allow up to 80% of wet AMD patients go 12 to 16 weeks between injections.[3] While Vabysmo will allow for longer intervals between injections, the previously approved drugs still work well and are sometimes what insurance will cover.

Prompt treatment of wet AMD is associated with better long-term visual outcomes, and many cases of severe vision loss can actually be prevented through early detection and referral.

Optometrists must stay current on the latest advancements in anti-VEGF therapies as well as how to manage and follow patients long-term. This includes recognizing potential adverse events and any safety concerns.

Dr. Gerson strongly encourages home self-screening, and patients should be educated on how to properly use these screening tools. "The percentage of retina doctors (and optometrists) using it is growing," he says. "No retina doc will say home screening is a bad idea; however, they should take the time to tell a patient about it."

Despite their utility, home self-screening tools do not replace the need for frequent in-office examinations. ■

Intermediate AMD with large drusen.

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About Dr. Ajamian **Dr. Ajamian** is board certified by the American Board of Optometry and serves as Center Director of Omni Eye Services of Atlanta. He is vice president of the Georgia State Board of Optometry and general CE chairman of SECO International. He has no financial interests to disclose.

Simplify Daily Decision-Making with Modern ERG

Michael Cymbor, OD, FAAO
Nittany Eye Associates

Dorothy L. Hitchmoth OD, FAAO
Hitchmoth Eye Care

J. James Thimons, OD, FAAO
Ophthalmic Consultants of Connecticut

What type of ERG device do you use in your practice?

Dr. Thimons: We use the RET*eval* Device (LKC Technologies) because it's powerful and fits in the palm of your hand. In fact, it's the only FDA-cleared, portable, non-mydriatic electroretinography (ERG) testing instrument on the market in the US. It is a major departure from traditional electroretinography and represents a technology that has evolved from large, expensive, and complicated to portable, affordable and easy-to-use and interpret.

How does ERG help you manage patients with diabetes?

Dr. Hitchmoth: Early detection of retinal abnormalities is a critical step in preventing vision loss. That's why the RET*eval* has been a game changer in how I care for my patients who have diabetes. In an assessment of RET*eval*'s ability to evaluate diabetic retinopathy, the advantages included earlier detection of retinal dysfunction, lower investment costs, and less required reading knowledge than traditionally-used imaging techniques.[1] The way we care for patients is advancing beyond fundus imaging.

How does the RET*eval*'s DR Assessment Protocol work?

Dr. Cymbor: The DR Assessment Protocol provides a superior risk assessment for progression. A score of 23.5 or higher indicates an 11-fold risk of requiring intervention within 3 years.[2] As diabetic patients worsen into moderate and severe nonproliferative disease, it may become challenging to determine the best time to refer to a retinal specialist. The DR Assessment Protocol helps me to clarify the correct time to refer, enhancing patient outcomes.

Is ERG needed if you have access to a good structural imaging device?

Dr. Thimons: Combining structural and functional information provides better results. Electroretinography objectively evaluates the functional abnormalities of the retina, while structural imaging shows the anatomy of the retinal tissue. While both functional and structural assessments have their benefits, functional changes generally appear well before structural changes. In studies comparing ERG and structural imaging's abilities to evaluate sight-threatening diabetic retinopathy, RET*eval* ERGs outperformed the traditional imaging techniques in predicting which patients would later need medical intervention.[2,3]

The RET*eval*® is a powerful aid in the diagnosis and management of retina and optic nerve diseases, such as diabetic retinopathy and glaucoma.

How does ERG help you manage glaucoma patients?

Dr. Cymbor: With the variability and length of subjective visual field testing, I appreciate having a quick and objective way to measure visual function. The RET*eval* PhNR test objectively measures the ganglion cells' function by evaluating the electrical activity of the cells to a light stimulus. Knowing about the function of the ganglion cells assists in the detection and tracking of glaucomatous changes. The test also has high repeatability, independent of media opacities.[4-6] PhNR testing with the RET*eval* Device provides sensitive, objective tracking of retinal changes for more informed follow-up, even where no changes in visual field or RNFL could be detected.[7]

> **"Catching retinal abnormalities quickly is critical for minimizing damage and maximizing vision retention. That's why the RET*eval* has been a game changer in how I care for my patients"**
>
> **— Dr. Hitchmoth**

How time-consuming is ERG testing?

Dr. Hitchmoth: Your technician can perform the test in 2-3 minutes, making it one of the most efficient tools available in our practice. Plus, it's completely objective and doesn't cause patient frustration. Patients tolerate the test and love the fact that they do not have to respond or provide the "right answer".

How do you bill for ERG?

Dr. Cymbor: There are more than 670 codes you can select from to bill for ERG. In our practice, we most commonly use CPT code 92273, billing the RET*eval* as a reimbursable test for DR. Some optometrists use the DR protocol to screen all patients with diabetes, whether or not they've been diagnosed with retinopathy, but this application is not reimbursable using this code.

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BY BISANT A. LABIB, OD

Color Clues

Iris hue can be a good starting point to determine the potential for disease development.

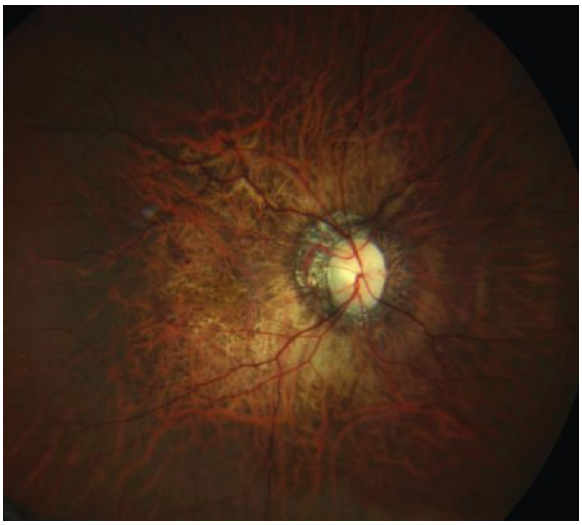
Eye color is a noticeable trait to anyone, but the impact it has for the eyecare provider is novel. While one may notice and appreciate variability in eye color and how it affects physical appearance, to the trained practitioner it may also serve as a harbinger of disease. The roles of both the structure and pigment of the iris have been well-studied features in many ocular disease processes. As such, it is important to take this into consideration when examining the iris, paying close attention to intricacies that may coincide with potentially harmful conditions.

Iris Pigment

A key component of color is the presence and function of melanocytes. These cells are present in the anterior border layer of the iris stroma.[1] A unique property of melanocytes is their ability to produce melanin in melanosomes. This function is generally influenced by the structural proteins of melanosomes, the enzymes responsible for synthesis of melanin and the proteins required for transportation and distribution of melanin. Melanocyte development and function is an integral part of many features and processes throughout the entire body, not just the eye. However, any disruptions to these functions can produce either hyper- or hypopigmentation, which is often a result of pathological factors.[2]

Normally, iris color starts out lighter during infancy as pigment develops. While it can darken initially, it is thought to be fully developed by then. Following the conclusion of infancy, additional pigment development is not normally observed. However, some studies do suggest that lightening or loss of iris color can arise with increased age due to changes in melanosome granule morphology.[1]

Iris color is determined by a combination of genetic components and ethnicity. However, there are studies suggesting influence from environmental factors as well. Similar to the protective mechanism in skin, people residing in regions with more sunlight may have increased melanin production and darker irises in order to prevent damage to the retina.[3] Along the same vein, the degree of pigment in the iris is relevant to its protective function, which explains why color is indicative of the development of some ocular diseases.



An example of degenerative myopia, which correlates more with light-colored irises.

Link to Diseases

The leading cause of blindness worldwide is cataracts. While the disease's development is multifactorial, iris color has been studied in association with age-related cases. In an epidemiologic study of white patients over age 49, those with darker brown-colored irises were found to have an increased incidence of both nuclear and posterior subcapsular cataracts. Further studies elucidated that there was also a higher degree of nuclear cataract acceleration over time.[1] Contrasting data in smaller studies have been documented that instead showed that lighter brown-, blue- or green-colored irises correlated more with cataract development.[3]

Given these conclusions, there are few hypotheses that would explain the role of iris color in cataracts and also affirm the dual genetic and environmental mechanism. One theory is explained by the function of melanin, in that it absorbs photon energy. Subsequently, in eyes with higher melanin content, heat transfer may occur from the iris to the nearby lens, resulting in more rapid radiation-associated damage and resultant acceleration of cataracts.[1,3]

Another well-documented association between iris color and ocular disease is in age-related macular degeneration (AMD). Studies suggest that blue- or lighter-colored irises are a risk factor for the development of

About Dr. Labib **Dr. Labib** graduated from Pennsylvania College of Optometry, where she now works as an associate professor. She completed her residency in primary care/ocular disease and is a fellow of the American Academy of Optometry and a diplomate in the Comprehensive Eye Care section. She has no financial interests to disclose.

TABLE 1. IRIS COLOR ASSOCIATION WITH OCULAR CONDITIONS

Iris Pigmentation	Disease Association
Brown (more melanin)	Nuclear cataract Posterior subcapsular cataract Cataract progression
Green, blue, hazel (less melanin)	AMD Myopia/astigmatism Uveal melanoma

AMD. Again, this can be explained by the function of melanin. The amount of melanin in the iris is directly proportional to the amount of light that is absorbed by the tissue, which is a protective mechanism to prevent oxidative damage from reaching the retina and contributing to AMD.[1]

While not solidified, there have been hypotheses regarding a link between refractive error and iris color. Myopia has been related to decreased use of sunglasses, which would suggest that dark-colored irises would be more protective against sunlight and, in turn, myopic refractive errors.[1] Increased levels of both myopia and astigmatism were observed in animal models studying light- vs. dark-colored irises and refractive errors.[3]

Another clear association occurs between ocular pigmentation and the incidence of uveal melanomas. These formations are dangerous, sight-threatening tumors that primarily arise in the choroid and, to a lesser degree, the ciliary body and iris. Regardless of the location, the role of iris color in the development of melanoma is well established. Several studies have agreed that the lighter the iris color, particularly green and hazel, the higher the risk of uveal melanoma. The type and amount of melanin are crucial in preventing harmful light from penetrating the eye and causing toxic damage.[4]

Takeaways

The iris carries out many functions and possesses many distinctive properties. It lies adjacent to the ciliary body and in close proximity to the cornea and lens, and it contains muscles that affect pupil constriction and dilation. In some way or another, it is implicated in countless ocular disease processes. Examining another unique feature, iris color, which is readily observable to any person, can offer unique insight in determining a patient's risk of developing certain diseases. ■

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CONQUER THESE OCT TECHNOLOGY CHOICES AND CHALLENGES

Experts explain device differences to help you determine which factors matter for your clinical purposes and offer advice for successful integration.

BY CATLIN NALLEY
CONTRIBUTING EDITOR

Optical coherence tomography (OCT) is a valuable tool in optometric practice that plays a key role in various aspects of care, including managing posterior segment disease, glaucoma and even some anterior segment conditions. However, as technology evolves and the number of available options continues to grow, determining the best device to invest in comes with a host of considerations.

"When first deciding how to proceed with purchasing an OCT, a good place to start is to consider your patient population and what you want to achieve," notes Jessica Haynes, OD, of the Charles Retina Institute in Memphis. "For example, some may want to integrate OCT imaging into their practice primarily as a screening tool, referring patients to a specialist when abnormalities are detected.

"Others may want to diagnose and manage a larger range of disease and send patients to specialists only when they require surgical interventions not within the optometrist's scope of practice," she continues. "These two physicians may have very different ideas about what instrument is necessary for their practices."

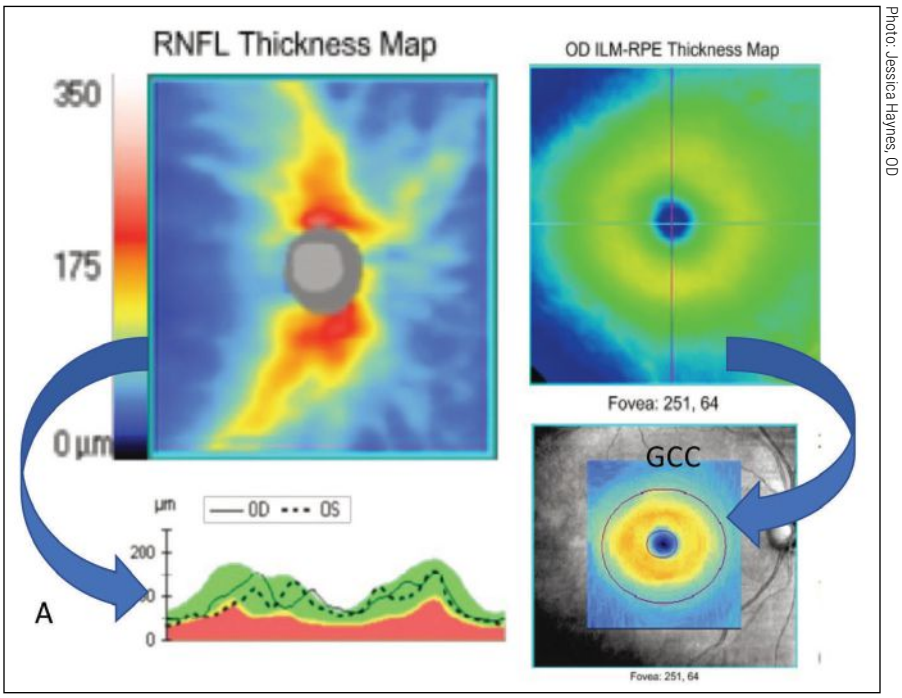


Photo: Jessica Haynes, OD

Fig. 1. Basic scanning patterns such as the optic disc cube (left) and macular cube (right) can be used to extrapolate a wealth of information such as RNFL thickness maps (top left) and RNFL TSNIT curves (bottom left), macular thickness maps (top right) and ganglion cell complex thickness maps (bottom right). These types of scans may also provide deviation plots to compare your patient to a normative database. Scans from a Zeiss Cirrus.

"What specialty are you in or what clinical interest would you like to explore?" asks Michael Cymbor, OD, a partner at Nittany Eye Associates in State College, PA. "Are you interested in retina (age-related macular degeneration and diabetic retinopathy), optic nerve, ganglion cell and angle (glaucoma) or cornea (keratoconus scleral fits and refractive surgery)? Each manufacturer has strengths and weaknesses—you want

Photo: Jessica Haynes, OD

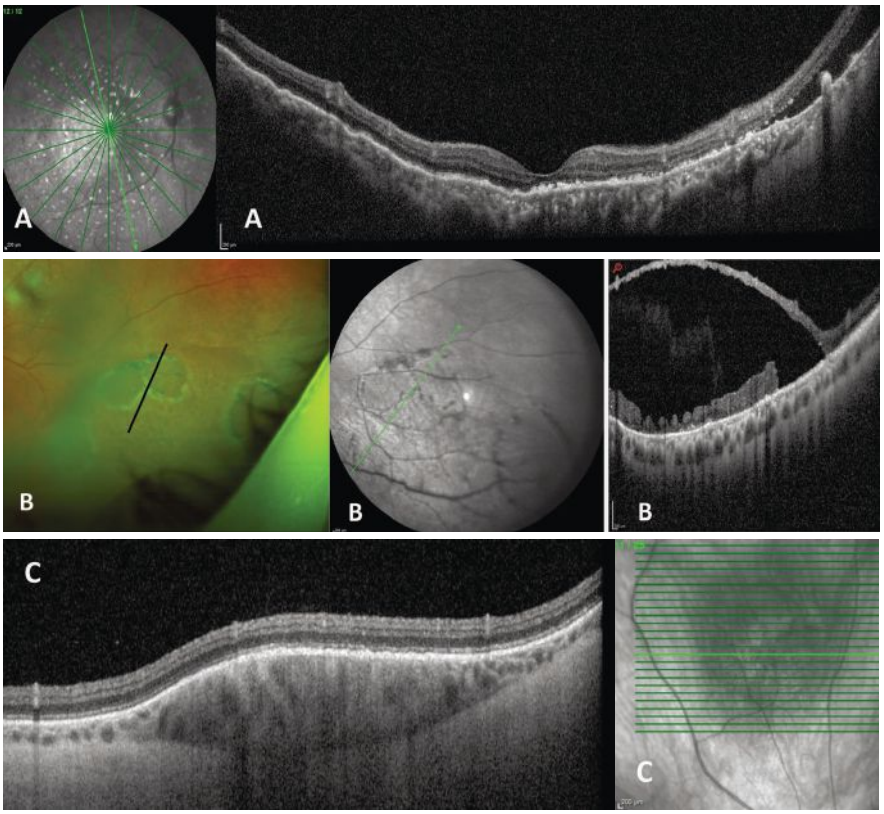


Fig. 2. Complex or customizable scanning patterns along with wider fields of view are available on some instruments. Examples are (A) 55° widefield radial scan through the macula of patient with uveal effusion syndrome. (B) Peripheral OCT of patient with large outer layer retinal breaks within a retinoschisis. (C) A customized cube scan through a midperipherally located choroidal nevus. All images from a Heidelberg Spectralis.

to choose the company that matches well with you and your interests."

Whether an OD is preparing to invest in their first OCT or is ready to upgrade their current device, it is important they have a clear picture of their needs and how the available options on the market can support their clinical practice. We spoke with several experts to gain their insights on best practices as well as challenges and missteps to avoid.

Tech Considerations

Since the first commercial OCT device came on the market in 1996, the technology has evolved exponentially. This has opened the door to a plethora of new uses; however, it has also made deciding which option is best for your clinical needs more complex.

Today, optometrists can use OCT to image a variety of structures in the eye, according to Dr. Haynes, who notes that OCT is classically thought about in terms of scans of the macula or scans of the optic nerve, with the typical macula scan being a "cube scan centered on the macula and the optic nerve scan showing the temporal superior nasal inferior temporal (TSNIT) or NITSN curve measurement of the retinal nerve fiber layer (RNFL)."

While these scan types are an important component of patient care, many devices can now go beyond basic scanning patterns. "They offer complex or even customizable scanning patterns of the macula, optic nerve, mid-peripheral and even peripheral retina," explains Dr. Haynes (*Figures 1 and 2*).

Scan resolution is another important consideration. "Some instruments offer more densely spaced scans with lower resolution alongside higher resolution raster type scans," says Dr. Haynes. "It is important to consider the resolution of scanning patterns that you will be using frequently (*Figure 3*)."

Spectral domain (SD) vs. swept source (SS). A key decision when selecting a device is whether you prefer SD-OCT or SS-OCT. While both offer high resolution retinal images, there are differences to consider.

"When evaluating different technologies, prioritize which features are most important to you and what best aligns with your practice's needs," says Julie Rodman, OD, a professor at Nova Southeastern University. "Current SD-OCT models provide outstanding resolution with a rapid acquisition time, capturing 26,000 to 80,000 axial scans per second."

SS-OCT operates at a speed of 100,000 to 200,000 axial scans per second, allowing for more precise imaging of the deeper retinal layers and choroid, she explains, noting that the faster scan time with SS-OCT also allows for wider B-scans to assist with widefield images.

The superiority of SS-OCT in imaging deeper structures such as the choroid can be impactful in patients with pachychoroidal disease or when identifying and more clearly defining the extent of an occult choroidal neovascular membrane on OCT angiography (OCT-A), according to Dr. Haynes. "How this may impact use in every day clinic would depend on the individual provider and which types of disease they want to manage."

She also noted that some SD instruments offer enhanced-depth

Photo: Jessica Haynes, OD

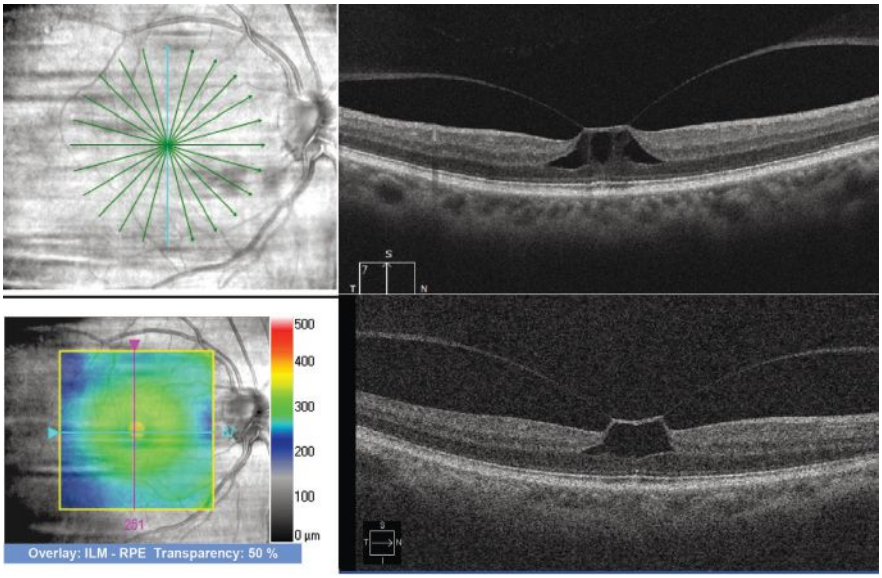


Fig. 3. Comparison of a high-resolution radial scan (top) and lower-resolution cube scan (bottom) performed on a patient with vitreomacular traction. Both are good quality scans, but they are different types, with the radial scan offering higher resolution and more slices through the fovea. Scans taken with Zeiss Cirrus OCT.

imaging (EDI)—a scanning strategy that can be selected for patients with suspected pathology that affects deeper tissue such as buried optic nerve head drusen. For optometrists purchasing an SD-OCT, Dr. Haynes recommends asking if the device comes with the EDI option.

So, how do you decide which option is best for your clinical practice? While every individual practice is unique and has its own specific needs, Danica Marrelli, OD, assistant dean of clinical education at the University of Houston College of Optometry, says that a SD-OCT without angiography is going to be sufficient for the vast majority of optometrists. However, that's not to say this is the case for everyone or that there isn't value in investing in newer technology when possible.

"There is an argument for purchasing the very latest technology you can afford because technology is only going to continue to move forward," Dr. Marrelli suggests. "And I do believe we are moving more and more towards SS-OCT. That being said, you're not going to go wrong with a good SD-OCT."

OCT-A. There are several technologies on the market that offer OCT-A—a non-invasive imaging modality that provides visualization of the retinal and choroidal vasculature. "When used alongside OCT, OCT-A rounds out the complete picture of structure and vascular integrity," notes Dr. Rodman.

"This technology is particularly useful in medically oriented practices, where common pathologies, including diabetic retinopathy and age-related macular retinopathy, are seen routinely," she says. "Capillary nonperfusion, microaneurysms, retinal ischemia and choroidal neovascularization can be seen without the use of intravenous dye."

Dr. Marrelli, who works in a glaucoma and retina heavy practice, has found that angiography is an interesting tool that, at this point, is not something they typically use to make clinical decisions. "It creates some really nice images, but it doesn't direct our management at this point that much more than an SD-OCT," she notes.

"Certainly in the glaucoma area, it's interesting to look at the angio of the blood vessels surrounding the optic nerve, but we really don't know what to do with it yet," she explains. "From a clinical standpoint, I don't know that we fully understand how to use the information we can gain from this tool yet in glaucoma patients. I think we'll get there, but I don't know if we are there right now."

Other clinicians do find this capability to be useful in glaucoma today. Dr. Cymbor says that "OCT-A is helpful to me in all stages of the glaucoma spectrum. The technology not only allows me to see RNFL defects easier, it helps me determine treatment efficacy. I would find it difficult to run our glaucoma clinic

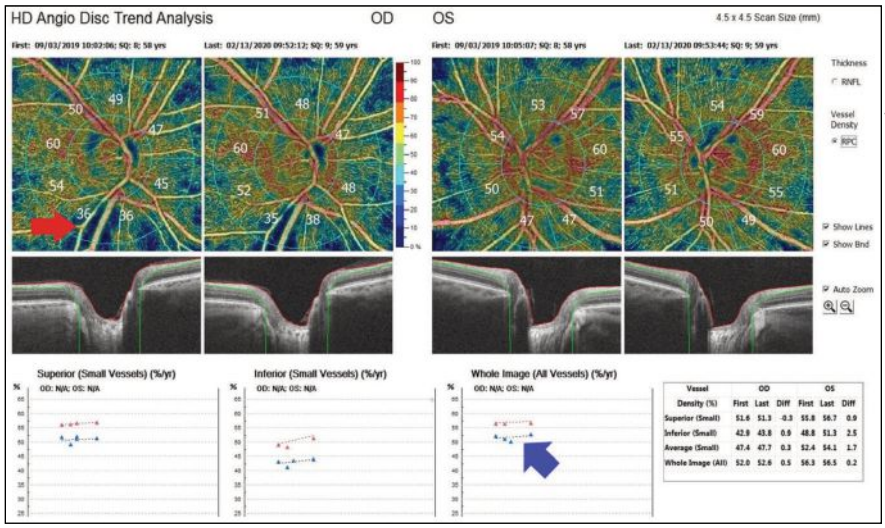


Photo: Michael Cymbor, OD

Fig. 4. This is an image of OCT-A glaucoma progression analysis from an Avanti from Visionix (Optovue). The top left red arrow shows a nerve fiber layer defect, and the bottom right blue arrow shows optic nerve perfusion improvement after treatment.

without OCT-A." (*Figure 4*)

Anterior segment. Most OCTs can also scan the anterior segment. This can be used to evaluate iris and angle anatomy in patients in the narrow angle spectrum, evaluate contact lens clearance when fitting scleral lenses, evaluate the penetrating thickness of a corneal foreign body and obtain optical pachymetry measurements, according to Dr. Haynes, who notes that consideration should be given if these are available with a particular instrument (*Figure 5*).

There are now some anterior-specific devices available that can offer improved imaging of this area than the instruments that are engineered for posterior segment use but have since added an anterior segment module to the software, says Sharon Keh, OD, assistant professor at SUNY College of Optometry. "Dedicated anterior segment OCT devices use deeper, higher wavelengths allowing for better imaging and resolution of the cornea and other anterior segment structures," she notes, while adding that most practices opt for posterior segment devices, which are more mainstream and have a longer list of billable diagnoses.

While a dedicated anterior segment OCT is probably not the right choice for most ODs, there are some that may find this option a good fit for their practice, Dr. Keh says. "Most practices who have this device are those with a very high volume of specialty contact lens fittings or OD/MD clinics that comanage complex corneal disease and glaucoma," she notes. "For the most part, anterior segment OCT allows for more precise imaging but has yet to replace an in-depth clinical evaluation, *e.g.*, gonioscopy."

"Some OCTs, such as our Heidelberg, have anterior-segment OCT capabilities and even fundus photo and fluorescein angiogram options but require that separate special lenses be attached to the machine in order to capture these," says Sara Weidmayer, OD, of the Kettles Medical Center VA in Ann Arbor, MI. "In our busy practice, whose OCT is in use non-stop all day—overwhelmingly for posterior segment OCTs—these lens swaps are actually fairly cumbersome, so it is prohibitive" when used at high volume.

"As scan speeds improve, it is only a matter of time until we have devices that are able to effectively image all layers of the eye from eyelid to cornea," notes Dr. Cymbor.

Viewing and analytic software. While the raw reflectivity data of OCT is often viewed as a cross-section OCT B-scan, explains Dr. Haynes, the instruments allow for visualization of the data in a variety of ways, including *en face* OCT imaging and thickness maps, among others. Thickness maps may involve the ability to view full retinal thickness or even that of individual retinal layers.

"Instruments are unique in the software analysis that they provide," Dr. Haynes notes. "For example, the Cirrus by Zeiss has a program that allows the visualization of drusen volume that can be tracked for change over time (*Figure 6*). Another example would be that while many instruments offer OCT-A, the Optovue Angiovue from Visionix provides a software package called AngioAnalytics that gives numerical data about the size of the foveal avascular zone and vascular density (*Figure 7*)."

Also consider tracking software, which is particularly valuable in glaucoma as well as retina care, where the ability to detect structural change over time is critical, according to Dr. Haynes. Some questions to ask include:

- How does the software allow the physician to look for change over time?
- Can progression analysis be viewed efficiently in a busy practice?
- What technology is used to ensure that scans are accurate and repeatable over time?

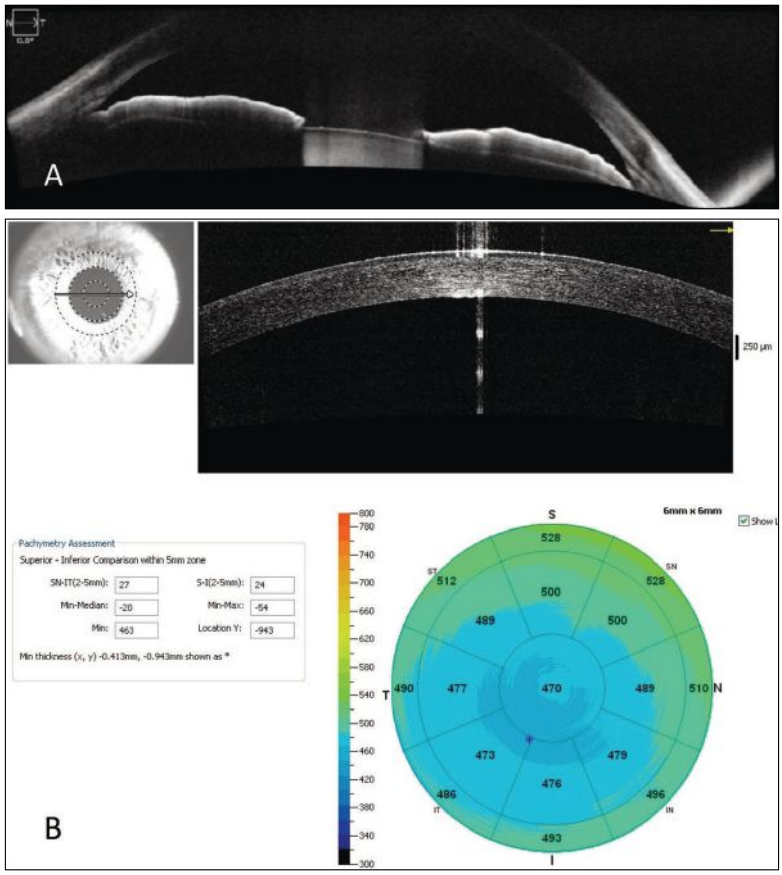


Photo: Jessica Haynes, OD

Fig. 5. An anterior segment OCT (A) used to evaluate a patient with anatomically narrow angles taken with Zeiss's Cirrus OCT, and (B) optical pachymetry measurements performed with the Optovue Avanti OCT from Visionix.

When making buying decisions, it can also be helpful to think about how you can view your OCT information, recommends Dr. Haynes. "Can you add a desktop viewer to each work station? Or will you have to rely on viewing a pre-selected printout sent to your EMR in your exam room?"

Dr. Weidmayer's VA clinic initially had a license for only so many viewing stations, which she says was terribly inconvenient for workflow, since they had many more doctors than available viewing stations. "This created a ruckus of having to track down doctors to log off so others could log on," she explains. "Especially for the retinal scans and OCT-A, the pre-selected printouts or reports are not adequate for decision-making, so the workstation viewing software—or using the machine itself—is critical."

Overcoming OCT Artifacts, *by Henrietta Wang, BOptom (Hons), BSC, MPH*

The guiding principle in all of healthcare is *primum non nocere*, or "first, do no harm." While we usually think of that in a treatment context, this can also be applied to the diagnostic techniques we employ on a day-to-day basis. If the device itself introduces errors, this can undermine the clinical care we provide by confounding accurate diagnosis.

OCT technology is extremely precise but not infallible. Artifacts—faulty data arising from one or more errors in scan acquisition—are surprisingly common in OCT use. They can arise from one of four sources:

(1) *the patient:* small pupils, media opacities, poor fixation or anomalous retinal planes

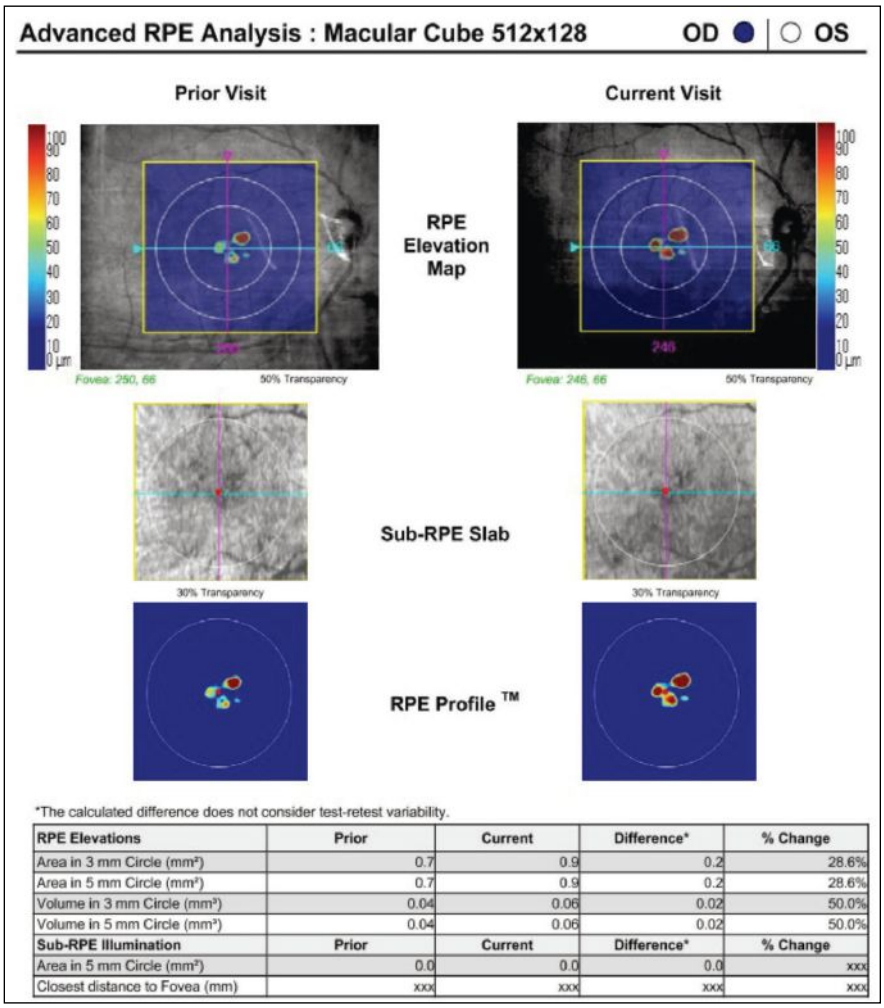
(2) *the instrument:* segmentation errors, inadequate depth of view, limited refractive range or posterior shadowing

(3) *the operator:* alignment or centration error, inappropriate scan selection or focusing mistakes

(4) *the disease:* the presence of concomitant pathology (*e.g.*, ERM or PPA)

Our team at the Centre for Eye Health (UNSW) has developed a set of guidelines that appears on the next two pages. They can help you minimize the confounding effects of OCT artifacts. Feel free to photocopy or download it for use in your practice. Best of luck!

Photo: Jessica Haynes, OD



RPE Elevations	Prior	Current	Difference*	% Change
Area in 3 mm Circle (mm²)	0.7	0.9	0.2	28.6%
Area in 5 mm Circle (mm²)	0.7	0.9	0.2	28.6%
Volume in 3 mm Circle (mm³)	0.04	0.06	0.02	50.0%
Volume in 5 mm Circle (mm³)	0.04	0.06	0.02	50.0%
Sub-RPE Illumination	**Prior**	**Current**	**Difference***	**% Change**
Area in 5 mm Circle (mm²)	0.0	0.0	0.0	xxx
Closest distance to Fovea (mm)	xxx	xxx	xxx	xxx

Fig. 6. A macular cube scan on the Cirrus can be used to extrapolate and track drusen volume using its Advanced RPE Analysis software.

"While many optometrists are fine with reviewing standard OCT reports, some will prefer using viewing software to access each scan slice to pick up pathologies like subtle paramacular drusen that is easily missed in current reports," adds Dr. Cymbor.

Value of Other Features

Many of the platforms available today offer a variety of multimodal imaging capabilities that go beyond the traditional OCT experience. This includes fundus autofluorescence (FAF), OCT-A, fluorescein angiography, indocyanine green angiography, fundus photography and multispectrum imaging. But how important are they for the optometrist? As with other buying decisions, this will depend on a practice's individual needs.

"It is extremely beneficial to have the capability of visualizing a fundus image simultaneously with the OCT scan. This way, pathology can easily be located on the fundus image and cross-referenced with the OCT B-scan," notes Dr. Rodman. "As previously mentioned, clinics that are seeing a high volume of medical retina would certainly benefit from the addition of OCT-A. This technology allows for pre-emptive diagnosis of disease in turn optimizing care of our patients."

Chairside Reference: How to Avoid OCT Optic Nerve Head & RNFL Artifacts

POINTERS FOR ASSESSING OPTIC NERVE PRINTOUTS

1 A signal strength (SS) of 6 or more is recommended by the manufacturer; however, SS of 7 or greater has been shown to have higher reproducibility.

2 The RNFL thickness maps should not have any areas of missing data (*i.e.*, black pixels).

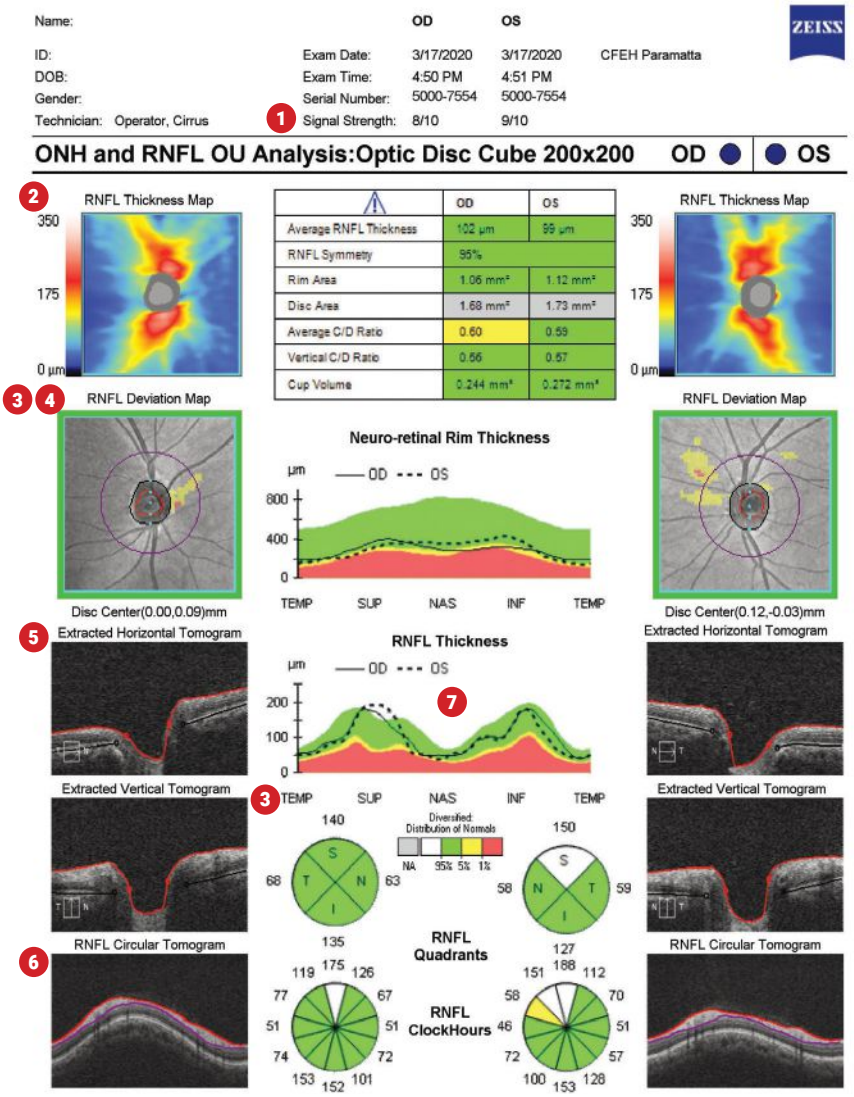
3 Disc and cup margins should be accurately delineated both in the *en face* (front on) image and on the horizontal/vertical tomograms (if applicable).

4 Vasculature in the scanning laser ophthalmoscope (SLO) image should be continuous and should not have any missing data. Note: OCT banding may masquerade as a motion artifact; however, this is simply the correction of saccadic movement during the scan.

5 The tomograms should be well-centered, with no opacity-related missing data, posterior shadowing or image truncation.

6 The RNFL should be segmented throughout the circular RNFL tomogram.

7 TSNIT curve values below 30µm are typically due to errors in segmentation.



FACTORS AFFECTING SCAN INTERPRETATION

Problem	*Potential Remedy*
• Presence of pathology • Inadequate pupil size, dry eye and media opacities • Poor signal strength • Motion and/or blinking during scans	• If a floater is present, get the patient to look to-and-fro prior to scan acquisition to displace it. • If a cataract is present, acquire the scan through the region of least opacity. • If poor signal strength is due to dry eye, the use of ocular lubricants may be warranted. • Advise the patient not to move or blink prior to scan acquisition. Check the fundus reference image immediately after acquisition and retake if necessary, • Ensure your gaze tracker is on, as this may help minimize the effect of motion or blink artifacts. • For patients with nystagmus, occluding the fellow eye and directing the patient's gaze towards the "null point" may be helpful.
• OCT lens opacities • Poor axial alignment of the image	• Check the OCT lens is clean with no smudges. • Ensure the patient is properly positioned during the test. • Check that alignment is correct and the entire scan is visible in the acquisition screens.
• Inaccurate delineation of the disc and/or cup margin • Incorrect segmentation of the RNFL	• To obtain accurate disc parameters, manual placement of the RNFL scan circle may be required. • It is important to ensure there are no other artifacts present as these may arise from other factors (*e.g.*, motion or media opacities)

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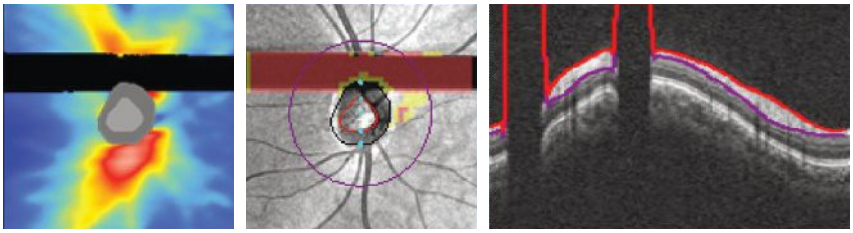
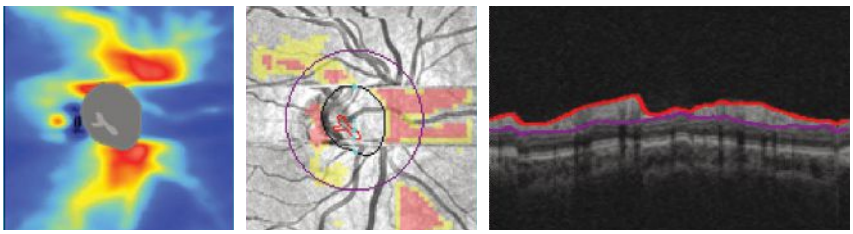
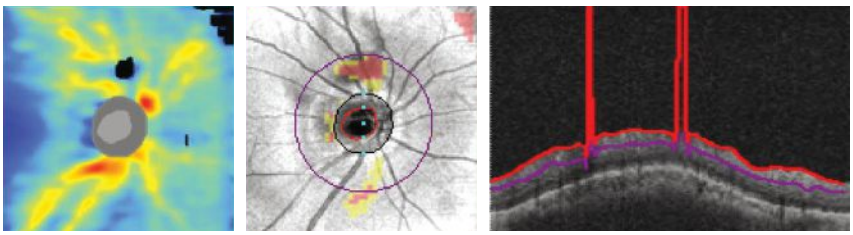
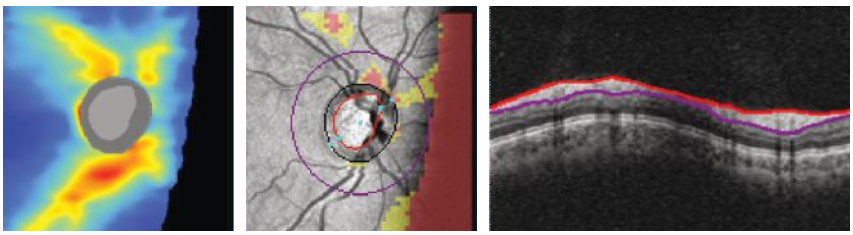
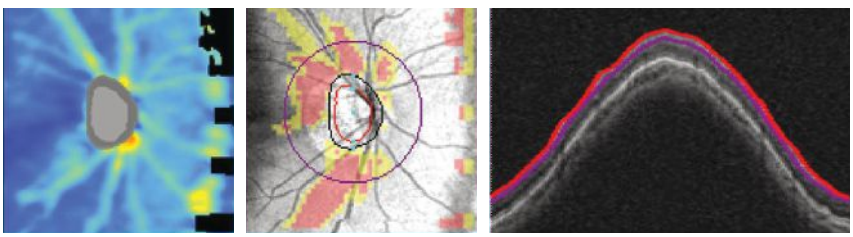
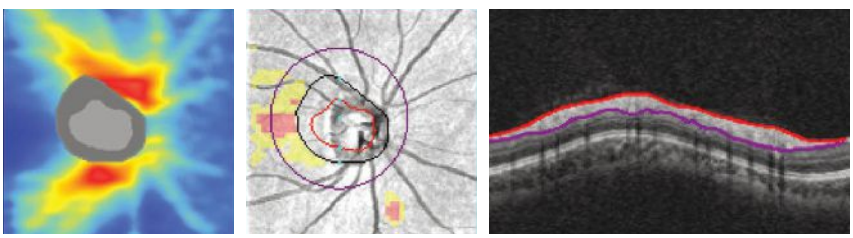
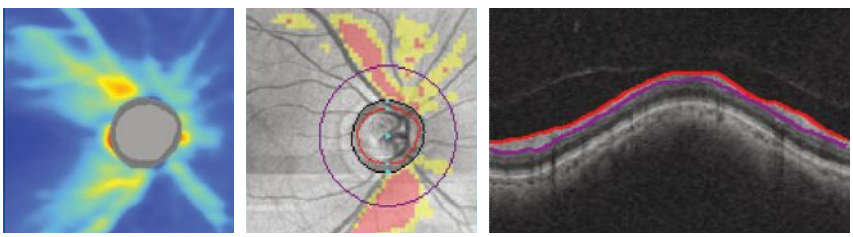
This guide was created by Dr. Henrietta Wang at the Centre for Eye Health, University of New South Wales. You can download this form at www.reviewofoptometry.com (look for the January 2023 issue) or by scanning the QR code at right.

Chairside Reference: How to Avoid OCT Optic Nerve Head & RNFL Artifacts

ATLAS OF OCT ARTIFACTS

Blink artifact. Black bands of missing data, caused by a patient blinking during acquisition, spanning the entire image. This may affect disc and/or cup delineation. Blinks affecting the scan circle will have vertical black rectangles of missing retinal profile on the TSNIT tomogram.	
Motion artifact. Manifests as a discontinuity in the thickness and (*en face*) deviation maps, most easily visualized as breaks in the retinal vasculature. The deviation map may consequently flag regions of apparent thinning. Retinal profile can be discontinuous if the scan circle is affected.	
Media opacities. Can cause data gaps (black areas) and apparent thinning (red pixels) corresponding to the opacity. Opacities affecting the scan circle manifest as vertical black shadows interrupting the retinal profile and RNFL segmentation.	
Vignetting/cut-edge. These arcuate black areas of missing data are typically due to shadowing from the pupil margin. The circular tomogram will show a degrading scan signal in the affected area (white arrow) of the scan.	
Image truncation. Data gaps corresponding to the areas of truncation show apparent thinning on the deviation map. The B-scan is vertically displaced, resulting in part of the retinal profile being cut off. This may cause RNFL segmentation errors.	
Inaccurate optic cup and disc margin delineation. Grayscale depiction of the cup and disc margins adopt an unusual appearance and do not match the funduscopic findings.	
OCT fundus banding. Correction of motion artifacts results in different gradations of individual B-scans. These appear as horizontal lines or bands in the deviation map (*en face* image) with no true discontinuity of the retinal vasculature.	

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Dr. Cymbor adds, "There is a substantial number of diabetes patients whose only structural change is diabetic macular ischemia. This ischemia can only be found on OCT-A or fluorescein angiography. Early diagnosis allows us to recommend more aggressive care at a more appropriate time point, leading to better patient outcomes."

The ability to view the retina with multiple imaging modalities can provide a more complete picture of disease, improving diagnostic and management capabilities, says Dr. Haynes. This may also, she notes, allow the instrument to be used for a wider patient range and billing codes.

"However, it is important to keep in mind that there are not currently separate CPT codes for each of these imaging strategies. For example, both OCT and OCT-A of the macula would use CPT code 92134 and both FAF and fundus photography would use CPT code 92250," advises Dr. Haynes. "While this is frustrating for physicians from a billing standpoint, the ability to properly and independently manage a wider variety of disease can ultimately create increased revenue for the practice, improve patient outcomes and increase job satisfaction."

In addition to the potential clinical benefits of being able to image the patient in numerous ways, devices with multiple modalities can be a good option when space is an issue.

"If an optometrist needs to minimize the footprint of their instruments, combination devices that offer fundus photography and OCT can be a good option," says Dr. Marrelli.

"Be mindful that some units that combine OCT and fundus photography offer poorer resolution than stand-alone camera systems," says Dr. Cymbor.

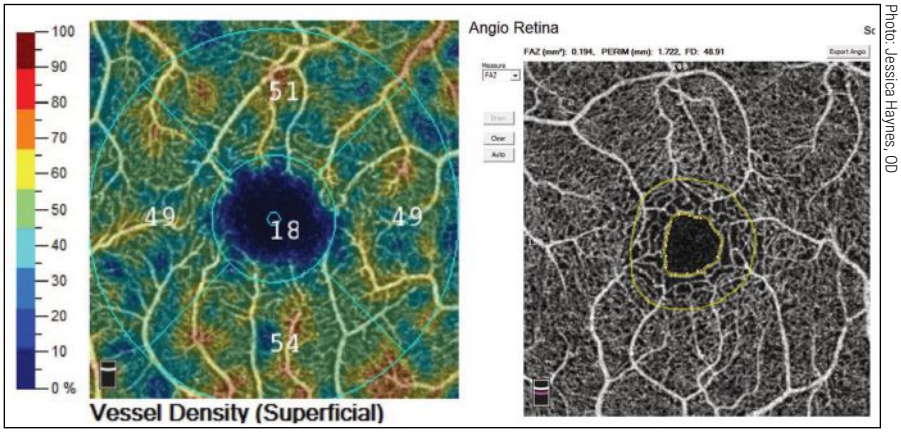

Photo: Jessica Haynes, OD

Fig. 7. The Optovue Angiovue OCT-A platform offers AngioAnalytics, a software package that computes vessel density and foveal avascular zone size that can be tracked over time.

Tech Support and Office Integration

Prior to purchasing an OCT, optometrists must take the time to time to have an in-depth discussion with the manufacturer of the device(s) they are considering. Don't hesitate to ask questions and raise any concerns you may have.

One of the most important questions to ask, according to Dr. Rodman, is what kind of support/service they will receive after the sale. "Support may come in the form of website access to informational portals or direct communication with a liaison that is familiar with the technology," she notes.

Understanding the level of support that will be available is critical, not just in terms of the technology itself, but also when incorporating these devices into clinical practice, notes Dr. Haynes. Several key questions to consider include:

- How will the instrument be maintained?
- How will it integrate into the EMR?
- Who is going to train your staff on how to use the instrument?
- What happens if there is a problem with the software or the hardware?

"Having a highly trained, easily accessible technical support team is crucial as these problems will undoubtedly come up," says Dr. Haynes, while advising ODs to inquire about training services, technical support and warranty plans before making a purchase.

Dr. Cymbor notes, "Sometimes these items are negotiable, so don't miss an opportunity to put yourself in a more attractive position."

Dr. Marrelli reiterates the importance of training and technical support. "This is something I feel very strongly about," she says. "Someone should come to your office, and they need to train you and your staff until you feel comfortable with the instrument."

This support should go beyond using the device, Dr. Marrelli explains. "Optometrists should have support not only with how to use the device and get the best quality scan but also how to really understand the report and analyze the images," she says. "Both types of training are important and need to be included in your purchase price."

This is also, in Dr. Marrelli's opinion, one of the drawbacks of buying a used OCT. "Buying an instrument from someone who, for instance, is looking to upgrade, may get you a great deal financially, but are you going to have the support and training you need? Training is critical for optimizing the impact the device has on your clinical practice."

Another key consideration is upgrade paths. If you want to initially purchase an OCT-only device right now, would you have the option to upgrade to obtain other platforms like FAF or OCT-A in the future?

It is critical that ODs have a clear understanding of the upgrades—and warranty—associated with

Key Questions for the Manufacturer

With numerous options on the market, it can feel daunting to wade through all of the information and data on each instrument. Below are key questions to ask that will give you confidence in your purchase and choice of manufacturer/device.

- What type of training does your company provide?
- Will a representative come to my office to train me and my team?
- What kind of support/service will I receive after purchasing the device?
- What is included in the service plans?
- How long is the warranty and what does it entail?
- Could you describe the upgrade paths/trade-in options?
- What does your product line offer? What distinguishes it from other instruments on the market?

the specific device they purchase, according to Dr. Marrelli. Are you going to have to pay for every software upgrade? Will you have to replace instruments? Will the upgrades be downloadable and easy to install? Or will someone have to come to your office for the installation?

"It is important ODs know what they are signing up for," she emphasizes. "Are you going to have to pay for every new feature or new upgrade? This can get expensive, so optometrists must keep this in mind in terms of their budget."

Another consideration is maintenance and service agreements, which can be very costly, Dr. Marrelli adds. "A smaller practice, or a practice that has a lower budget, may want to prioritize these considerations. An instrument that is going to be under warranty for a longer period of time and includes upgrades can be a very appealing option."

Potential Challenges and Missteps

Don't fall into the trap of thinking that all instruments do the same thing, urges Dr. Haynes. "There is such a variety of OCT instruments on the market," she says. "They all have their pros and cons, but they don't all do the same things. You really have to consider your patient population and clinic needs and find a device that best fits that need."

The second biggest mistake, in Dr. Haynes's opinion, is not using your OCT instrument to its fullest extent. If an OD has really done their research into the device they are pur-

TABLE 1. COMPARISON OF COMMERCIALLY AVAILABLE OCT MODELS

Model (Manufacturer)	Cirrus HD-OCT 5000 (Carl Zeiss Meditec)[1]	Plex Elite (Carl Zeiss Meditec)[1]	3D OCT-1 Maestro2 (Topcon)[2]	Triton (Topcon)[2]	Spectralis 2nd and 3rd Generation (Heidelberg)[3]	Spectralis OCT-A (Heidelberg)[3]	iVue80 Optovue (VIsionix)[4]	Optovue Avanti with Angiovue (Visionix)[4]
SD-OCT or SS-OCT?	SD-OCT	SS-OCT	SD-OCT	SS-OCT	SD-OCT	SD-OCT***	SD-OCT	SD-OCT
Scanning Speed (A-scans per second)	27,000-68,000*	100,000-200,000	50,000	100,000	85,000**	85,000	80,000	70,000
Axial Resolution (µm in tissue)	5	6.3	6	8	Optical: 7 Digital: 3.9	3.9	5	5
Imaging Modes	SD-OCT, cSLO	SS-OCT, OCT-A, LSO, CCD camera	SD-OCT widefield, color fundus, red-free fundus, IR fundus, enhanced IR fundus and external eye photography	SS-OCT, color fundus, red-free fundus, IR fundus	SD-OCT, cSLO	OCT-A	SD-OCT widefield	SD-OCT widefield, OCT-A, enhanced-depth imaging
SD-OCT Normative Database: *Number of subjects*	284 RNFL study 282 macula, ganglion cell, ONH study		399		201 (RNFL thickness)		480	
SD-OCT Normative Database: *Ethnicity*	43% Caucasian 24% Asian 18% African American 12% Hispanic 1% Indian 2% Mixed ethnicity		59% Caucasian 20% African American 18% Hispanic/Latino 3% Other		European descent		47% Caucasian 19% Asian 10% African 15% Hispanics 8% Indian 1% Other	

Adapted from tables by Drs. Lori Pennington and Brooke Smith, "OCT: What's Under the Hood?" Review of Optometry, Sept. 2022

** Cirrus OCT (5000 model) acquires 68,000 scans per second with OCT-A*

*** 3rd generation Spectralis offers Shift Technology: 20kHz, 125kHz not available in the United States*

**** Swept-source Anterion imaging app available*

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chasing, they should already be well versed in its capabilities.

"But in the event that you end up with an OCT instrument that you are not familiar with, be sure to take the time to learn all you can about the instrument," Dr. Haynes notes. "It may have scanning patterns, viewing software, progression analysis and other tools that enhance your ability to detect, accurately diagnose, monitor and manage disease, if only you learn how to use them."

A key issue ODs will have to contend with is staff training and avoiding scan accuracy issues. "In most situations, the doctor will not be the person obtaining scans," notes Dr. Haynes. "Does your practice have a highly trained ocular photographer or a skilled technician? If so, you may not have to worry about ease of use. If not, you may have to consider an instrument that is more 'point and click.'"

"This also goes hand-in-hand with support provided by the manufacturer," says Dr. Rodman. "It is very important that the doctor inquires about training. How is it done? Is there access to videos? Is there a liaison that could answer questions, if needed?"

Billing is also an important component. "For instance, a patient comes in for a vision exam, and you find something that needs to be medically evaluated. In that case, you need to separate that visit, re-appointment them and then do it under medical," says Dr. Marrelli. "So, optometrists who buy OCTs need to be on medical plans, they need to understand how to navigate vision versus medical and learn how to accurately bill."

Another question ODs may have is whether or not they need to stay with one brand for all of their devices. Is there a platform "lock-in" effect where you feel like you need to buy the Zeiss instrument because you have other Zeiss devices and years of patient data on their platform? According to Dr. Rodman, the answer is no.

"Most companies have platforms whereby you can merge different technologies and view on a common portal," she explains. "Oftentimes, these companies partner with other companies who can provide this service to their customers, so they don't feel locked into a particular brand."

However, an OD upgrading their existing OCT may find it easier to opt for the same brand when purchasing their new device, notes Dr. Marrelli.

"We're at a time now where instrument companies are pretty sensitive to the fact that we need to be able to have backward compatibility with former technology. However, to my knowledge, we don't really have anything that would allow you to, for example, transfer 10 years of data from a Zeiss instrument to a Topcon instrument," she says.

"When you change brands, you're not going to be able to directly compare and so there is some value in staying with the same brand in terms of bringing that data forward into the new instrument."

Staying with the same company does not always assure the use of previous patient data. Dr Cymbor says, "Sometimes new OCTs are unable to use previous data from older devices even within the same company, so you might start over for things like glaucoma progression analysis."

Ultimately, the optometrist must make their own choice and own that decision. The best way to do that is to be as informed as possible.

"Ask the vendor if they will let you try out the unit in your office before purchasing. Using the device and 'playing' with it in your setting is the most important thing to do," says Dr. Rodman.

"This will allow the doctor to assess the ease of use and the frequency of use of the various modalities offered on the device," she concludes. "What's good for one practice may not be for another. The needs of one practice may not match the needs of another." ■

SEEING GLAUCOMA THROUGH OCT'S EYE

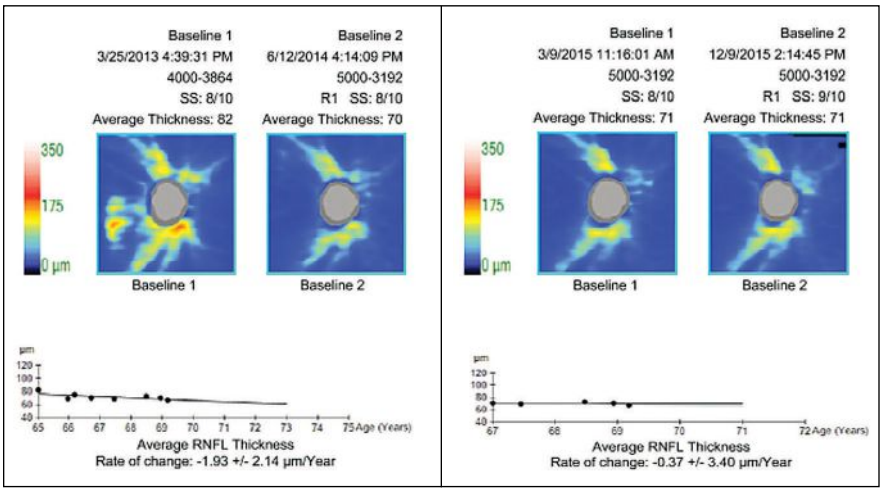
The answers to so many of our clinical quesions lie in the details; be sure to know what you're looking at.

BY ANDREW RIXON, OD, AND ABBEY KIRK, OD
MEMPHIS

Glaucoma is a progressive optic neuropathy characterized by the death of retinal ganglion cells and their axons.[1] Per this definition then, it behooves the optometric community to examine retinal structures in both qualitative and quantitative ways. OCT technology was designed for this very purpose and, when used properly, it can improve our virtuosity in the management of glaucoma.

The Breakdown

Understanding the anatomy of the OCT is just as important as understanding the information it generates, which can be broken into a few anatomical regions: the retinal nerve fiber layer (RNFL) or circumpapillary RNFL (cpRNFL), the ganglion cell and inner plexiform layers (GC-IPL) and Bruch's membrane opening minimum rim width (BMO-MRW). In addition to anatomical landmarks, reports and newer parameters exist that further assist physicians in diagnosing and managing glaucoma.



The pre-treatment rate of change (at left) is borderline fast. The re-baselined rate of change (at right) is less than age-related attrition. Re-baselining post-modification of treatment maintains an appropriate perspective and prevents over-management.

RNFL. This is a widely recognized and extensively researched parameter in the diagnosis and management of glaucoma, as it includes the optic nerve head (ONH) and the surrounding RNFL feeding into the neuroretinal rim. The cpRNFL acquisition and algorithm varies among SD-OCTs, but the data supplied is consistently cohesive, quantifying ganglion cell axonal loss. As the variety of SD-OCTs on the market is substantial, this article will briefly discuss facets of four platforms: the Cirrus HD-OCT (Carl Zeiss Meditec), Spectralis (Heidelberg Engineering), RTVue (Visionix) and Maestro2 (Topcon Medical Systems). Each OCT curates a ring scan involving the optic nerve: the Maestro2 (3.4mm), RTVue (3.45mm) and Cirrus (3.46mm), while

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TABLE 1. OCT SCAN INTERPRETATION

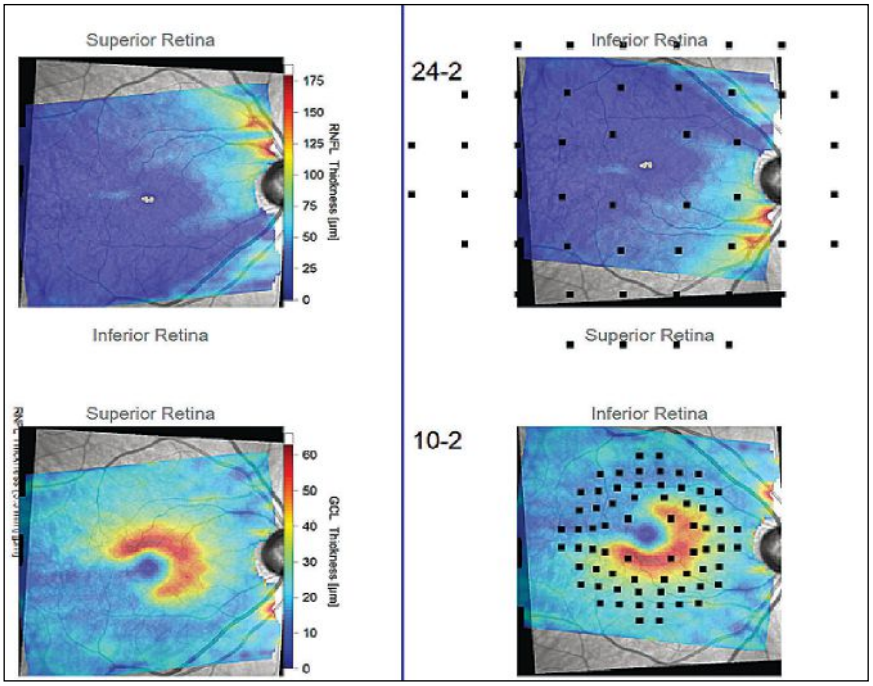
	Cirrus HD-OCT[14,15]	Spectralis[2,14]	RTVue[8,14]	Maestro2[7,16]
Macular Analysis[10]	*Ganglion Cell Analysis*	*Posterior Pole Asymmetry Analysis*	*Ganglion Cell Complex*	*Ganglion Cell Complex*
	Outer boundary of RNFL to outer boundary of IPL	Bruch's to ILM	RNFL + GC-IPL	ILM to IPL/INL
	200x200 or 512x128 line scans in a 6mm cube scan	8x8mm cube scan comprised of 61 line scans	7x7mm cube scan	6x6mm cube scan
Nerve Analysis	*ONH and RNFL Analysis*	*RNFL Basic Report*	*Nerve Head/RNFL Analysis*	*3D Disc Report*
	RNFL layer (axon thickness value from volume scan)	RNFL layer (single layer, no extrapolation)	RNFL layer (axon thickness value from volume scan)	RNFL layer
	RNFL values:[24] 98.7±10.9	RNFL values:[24] 106.6±12.8	RNFL values:[24] 112.8±13.2	RNFL values:[24] 101.9±8.4
	200x200 line scans in a 6mm cube, 3.46mm diameter ring around ONH	3.5mm, 4.1mm and 4.7mm circle and radial scans enveloping ONH	5mm diameter ring around ONH, 3.45mm cpRNFL circle scan for TSNIT graph	6x6mm ONH scan, 3.4mm cpRNFL circle scan for ONH and NSTIN graph
	*Reference plane-dependent (200µm above RPE)	*Reference plane-independent (BMO-MRW)	*Reference plane-dependent (150µm above RPE)	*Reference plane-dependent (120µm above RPE)
Glaucoma Analysis	*PanoMap*	*Hood Report*	*cpRNFL and Ganglion Cell Complex Analysis*	*3D Wide Report with VF test points*
	Combined macular and optic disc cube scans, other scans such as pachymetry and HD angle can be integrated for a more comprehensive view	Combined large B-scan image of ONH with TSNIT plots, cpRNFL and macular deviation maps and field maps correlating structural loss to functional loss	Not combined but are intended to be viewed simultaneously, providing thickness maps, deviation maps and large sector, TSNIT and significance maps	Combined B-scan cpRNFL maps, GC-IPL maps and correlation (VF + GC-IPL data) maps with both retinal views and field views for a complete structure and function layout
BMO-MRW Analysis	*Not available as separate printout*	*GMPE package, Garway-Heath sectors*	*Not available as separate printout*	*Not available as separate printout*
	Access points customized to disc size and ONH entry to retinal surface	24 HD radial scans of ONH (48 data points total)	Manual	Manually marked on radial image inspection

the Spectralis allows the user to choose their preferred diameter (3.46mm, 4.1mm or 4.7mm).

Color mapping helps the user visualize areas of thinning, denoted as cooler colors.[2] Many OCTs will section the RNFL into "sectors" and further color-match RNFL thickness to that of their respective database. It is important to ensure the scan data is complete and high quality and the sectors agree with the anatomy of the ONH and its emerging vasculature.

Two clinically relevant RNFL parameters captured in these scans are the superotemporal and inferotemporal quadrant RNFL thicknesses and the average/global RNFL thickness. The inferior RNFL is especially susceptible to glaucomatous damage and consequential focal visual field defects as there are fewer RNFL bundles derived from the inferior portion of the macula compared with the superior macula.[3] However, superotemporal RNFL bundles may be more often overlooked due to the larger density of the bundles that reside there, and RNFL loss can result in more shallow, widespread visual field depression; therefore, special attention should be given to both quadrants. Average RNFL thickness, when obtained reliably, can be a useful metric to use in assessing progression. A difference in 9µm between the average RNFL thickness of intra-eye measurements should be closely evaluated. Also, each OCT has its own criteria for "normal" RNFL thickness and should be considered when interpreting this data (*Table 1*).

GC-IPL. Scanning the macula is known to be very useful in glaucoma management. Various scan protocols across multiple instruments will capture tissue differently, but their universal goal is to assess retinal ganglion cell (RGC) thickness directly or indirectly. These cells are found most densely concentrated in the macula.[4] RGCs span the GC-IPL (bodies and



The Hood report of a patient with a wide-angle glaucomatous wedge that extends temporal past the vertical midline (black lines). These reports enhance the practitioner's ability to topographically analyze patterns of neural tissue loss that may or may not be consistent with glaucoma.

dendrites, respectively). RGCs are thought to be affected by glaucoma early in the disease process, thus evaluating this crucial area is considered to be the new standard of care.[5]

This genre of scan analysis employs color overlays (similar to that of RNFL maps) of a macular image, allowing the physician to use tissue thickness as a surrogate for RGC health. These scans can provide early insight into glaucomatous changes that may precede functional loss shown on visual fields.[6] The Ganglion Cell Analysis (Cirrus) uses the outer boundary of the RNFL, while the Ganglion Cell Complex (RTVue) report comprises the entire RNFL and the GC-IPL. In comparison, the Posterior Pole Asymmetry Analysis (Spectralis) spans Bruch's membrane to the internal limiting membrane (ILM), with a separate ganglion cell analysis also available. The Maestro2 spans the ILM to the inner plexiform/inner nuclear layers (IPL/INL) in its Ganglion Cell Complex report.[5,7]

Interpreting a macular scan can be uncomplicated. A loss in warmer colors in a typical arcuate glaucomatous pattern (often referred to as the nautilus or temporal raphe sign) quickly indicates a discrepancy in the anatomy of the RGCs. Macular scans also provide further complex parameters to detect early glaucomatous changes such as the focal loss volume and global loss volume available on the RTVue. The former measures the percentage of average focal loss over the entire map scan, while the latter measures the average ganglion cell complex loss over the entire map.[5,8] One study suggested that the focal loss volume is the most reliable single predictor in the conversion of functional loss.[9] These indices take less time to acquire, require less data processing and have a high correlation with the total RGC count.

BMO-MRW. Bruch's membrane is the innermost layer of the choroid and is a stable anatomical landmark of the optic nerve. The MRW marks the true anatomic disc margin denoted by the end of Bruch's membrane and provides a highly accurate measure of the neuroretinal rim. It measures the minimum distance between the termination of Bruch's and the nearest point of the ILM. This structure is reproducible throughout most of the disease spectrum and can be a stable reference point.[10] Additionally, it can provide an accurate correlation between structural and functional loss, especially in normal-tension glaucoma and patients with disc hemorrhages.[11,12] It is obtained via Spectralis 24 high-resolution ONH radial scans.[13]

The Process

In the past, studies on how to screen for and confirm glaucoma with OCT looked at the diagnostic sensitivities of the macula, RNFL and optic disc parameters in isolation, as well as global summary parameters, *i.e.*, whether the overall scan was normal, abnormal or borderline. It is now understood and accepted that combined analysis of at least both the GC-IPL and cpRNFL performs better than using each individual parameter on its own in cases of early glaucoma.[17]

To maximize outcomes in the initial screening process, it is recommended that all the tissues affected by glaucoma should be evaluated on OCT in combination.[18] Specifically, given that glaucomatous loss primarily behaves in an arcuate pattern, a topographical approach to assessing loss demonstrated on OCT is recommended.[19] This is the approach taken with many of the widefield glaucoma analysis reports, including the RNFL deviation map. As glaucoma-like artifacts can exist on healthy RNFL deviation maps, it is suggested to employ the vertical midline rule, wherein true glaucomatous arcuate damage will cross a vertical line through the fovea outside the macular-papillary bundle, while an artifact resembling glaucoma will not.[20] Although there is a push toward using OCT as a stand-alone, it should be used in concert with a combination of other diagnostic tools—such as funduscopy—to evaluate the presence of glaucomatous optic nerve abnormalities and confirm the diagnosis of glaucoma.

Misinterpretation. When reviewing the ONH and macular data provided

TABLE 2. OCT ERRORS AND ARTIFACTS

Patient Dependent	Operator Dependent	Machine Dependent
Age (relative to reference database)	Poor alignment of scan (axial, rotational, centration)	Inaccurate segmentation of RNFL tissue
Pupil size	Incorrect patient positioning	Inaccurate segmentation of disc margin
Tear film quality	Insufficient B-scans/low automated real time	Inaccurate segmentation of Bruch's membrane
Media opacities	Poor reflectivity	
Eye movement/blinking	Inadequate quality	
Epiretinal membrane	OCT lens opacities not mitigated	
Myopia/increased axial length		
Abnormal ONH insertion		
Peripapillary atrophy		
Cyclotorsion		
Past congenital or acquired ONH or macular abnormalities		

by the various OCT instruments, it is important to trust but verify that the information is usable. Proactively looking for sources of misinterpretation is a more realistic approach than being surprised by avoidable errors upon review. Research supports this approach, confirming that artifacts affecting the quality and therefore utility of images are common in clinical practice. A recent review of this issue noted that over 25% of patients are expected to have artifacts in their SD-OCT glaucoma imaging that could introduce either false positive or false negative interpretation.[21] Awareness of potential artifacts and errors is pertinent to maximize the quality and interpretability of the scans. Potential error generating factors can be broken down into three categories: patient dependent, operator dependent and machine dependent (*Table 2*).[21,22]

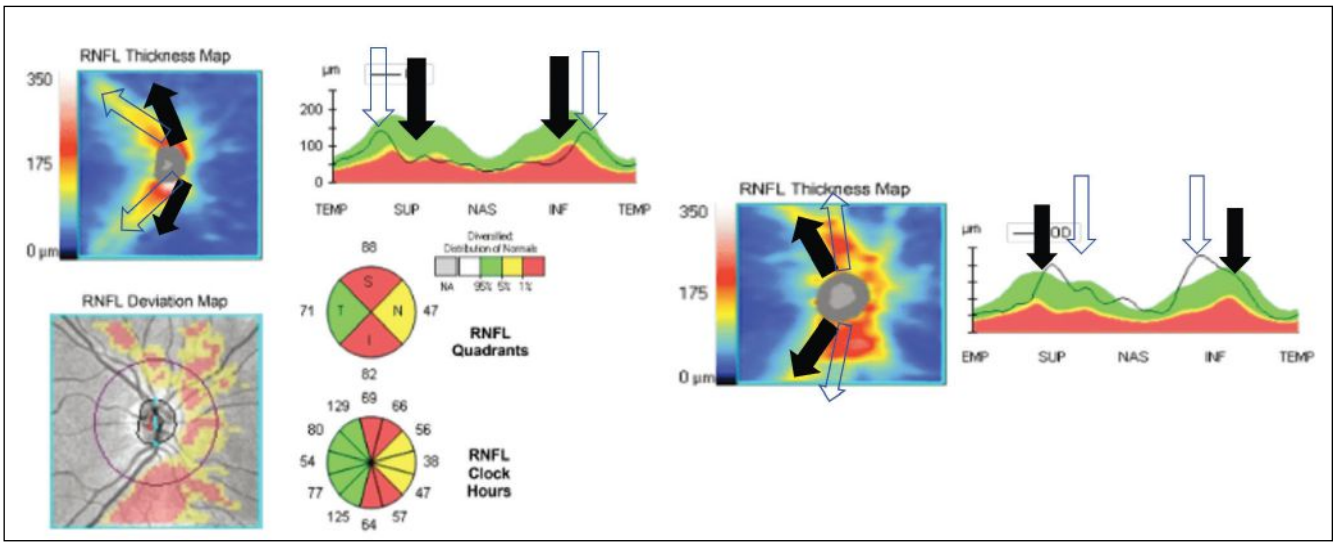
After accounting for artifacts that affect the utility of the data, it is necessary to be aware of how the various machines present normally and what deviation from that normal may look like, recognizing that green isn't always clean. Each instrument has its own normative database comprised of multiple individuals of various ages, races and genders unique to those platforms. Remarkably, the FDA has no standards or guidelines for the types or numbers of subjects who should be included in these databases or how that data should be presented or analyzed.[23] A more realistic way to classify these databases is to use the term "reference database," as they do not represent population norms but are comprised of averages of included patients. Those averages are based on proprietary segmentation, scanning protocols and thickness algorithms.[24]

Regardless of how the individual databases were compiled, tissue thicknesses are presented by all platforms as normal, borderline or outside normal limits/abnormal. These thicknesses are based on the percentile distribution of database thicknesses and employ color schemes to aid analysis. These color schemes use white and typical traffic light colors green, yellow and red. White includes the 95th to the 99th percentile (above normal), green includes the 5th to the 95th percentile (normal), yellow the 1st to the 5th (borderline) and red the 1st percentile (below normal).[24] Therein lies the trap: green accounts for 90% of the tissue thickness on these machines. A normal finding does not signify the patient is free of disease and/or from progression over time; conversely, an abnormal finding does not mean the patient has disease, and misinterpretation of results could lead to misdiagnosis and mistreatment.

So, what is the relevance of this broad range of tissue for clinical practice? Let's step back for a second and quickly review dynamic ranges, measurement floor and steps. For OCT, the dynamic range is the usable range between the highest and lowest capturable values of tissue, based on healthy eyes. The measurement floor is the lowest detectable value beyond which measurement has no clinical value. In any test run multiple times, there will be test-retest variability, or a measurement step. The test-retest variability for average RNFL thickness on most platforms is 4µm to 5µm and 2µm to 3µm for average GC-IPL thickness. The number of steps in the overall range determines how much significant change can be detected over time; the greater the number of measurement steps in a given range, the greater the ability to capture glaucomatous change over time.[25]

The relevance of this information is that within the green category there can be a considerable number of steps of RNFL progression, meaning there can be substantial, clinically meaningful glaucomatous loss even though the machine will categorize these changes as normal. As a point of reference, one study employing estimated RGC count at various stages of glaucoma found that a loss of 5µm was equivalent to a loss of 100,000 RGCs. There were six 5µm steps between 75µm and 105µm.[26] The take-home was that reliance on colors for affirmation of normalcy is a mistake.

Monitoring. OCT can be used to monitor progression and in doing so can confirm the success or failure of treatment. The only repeatable way to objectively confirm the success or failure of a glaucoma treatment is through tracking the progression of structure and function. As a result, there has been a recent effort to implement the use of target rate of progression as a more impactful, quantitative concept than attempting to use target intraocular pressure (IOP) as a monitor for success, given how the dynamics of IOP, among other factors, make this difficult



The left image shows congenitally temporalized vasculature. The machine expects the vessels to be where the black arrows are. The clear arrows show the actual distribution of the vessels. This results in false positive thinning in the superior and inferior quadrants in an actually healthy patient. On the right is a congenitally nasalized vasculature, which could create a false positive.

to confirm.[27] The most repeatable, researched and therefore usable metric for tracking progression is the average/global RNFL, and the least repeatable is clock hours.[28,29]

The value of the BMO-MRW and GC-IPL when tracking progression has also been studied but not as extensively as the global RNFL. However, it has been suggested that, although the BMO-MRW has good intra-visit repeatability in mild and moderate disease, it may be less useful in advanced disease where the location of the BMO may change over time and be less consistently trackable.[30,31]

In general, GC-IPL and RNFL rates of thinning in early glaucoma have been shown to be comparable, but not interchangeable.[32] Although not commercially available, combining the RNFL and GC-IPL has been shown to reveal eyes that are progressing prior to detection by the RNFL or GC-IPL alone and might be a useful integrated parameter in the future.[33] The GC-IPL may be most useful in advanced-stage disease when the RNFL has reached the floor effect.[6]

Prior to delving into what are considered slow or fast rates of RNFL progression it is critical to perform baselining/re-baselining. As discussed previously, it is an absolute necessity to have high-quality baseline scans from which to compare. Once the practitioner initiates or escalates treatment with the intent to modify the disease, re-baseline. Comparing a pre-treatment tissue thickness to a post-treatment tissue thickness may give the impression that treatment has been unsuccessful and result in additional treatments that may be unnecessary. Realistically, the goal of treatment is to blunt the disease, not halt it entirely. Fortunately, an unrealistic number of scans per year is not necessary to confirm success or failure by determining the rate of progression. A 2022 study discovered that performing two quality OCT scans per year is reasonable and sufficient to determine those patients who are fast progressors.[34]

For reference, normal age-related attrition of the RNFL is less than -1µm/year (an average of -0.54µm/year), normal age-related attrition of the BMO-MRW is -1.92µm/year and the GC-IPL is -0.31µm/year.[35,36] A faster rate of change in any of these parameters might be indicative of true pathological tissue loss.[12] In fact, average RNFL progression rates from the Duke Glaucoma Registry are delineated into 1µm/year as slow, between 1µm to 2µm/year as moderate, 2µm to 4µm/year as fast and >4µm/year as a catastrophic rate of progression.[37] It is particularly important to capture these rates, as research recently found that rapid initial RNFL deterioration is a good predictor of a faster disease course and rate of visual decline.[38]

In progression analysis, OCT software will supply event and trend analysis. These may be labeled differently by the various platforms, but the concept is the same. Event analysis compares each OCT taken to a minimum of two baseline OCTs. The various platforms will then present probabilities of whether the patient has progressed. Conversely, trend analysis takes an average of the tissue thickness over time and provides the rate at which the disease is progressing.

Specifically in regard to event analysis, the test-retest variability of OCT has led to the informal "Rule of Five," which states that if a glaucoma patient undergoes a repeatable 5µm or greater loss of average RNFL on consecutive scans/events, then that loss is considered quantitative evidence of progression and might justify escalating treatment.[15] Although this concept is simple and easy to implement, recent research recommends exercising caution with its application, noting that this rule is not specific for glaucoma progression between tests and actually performs

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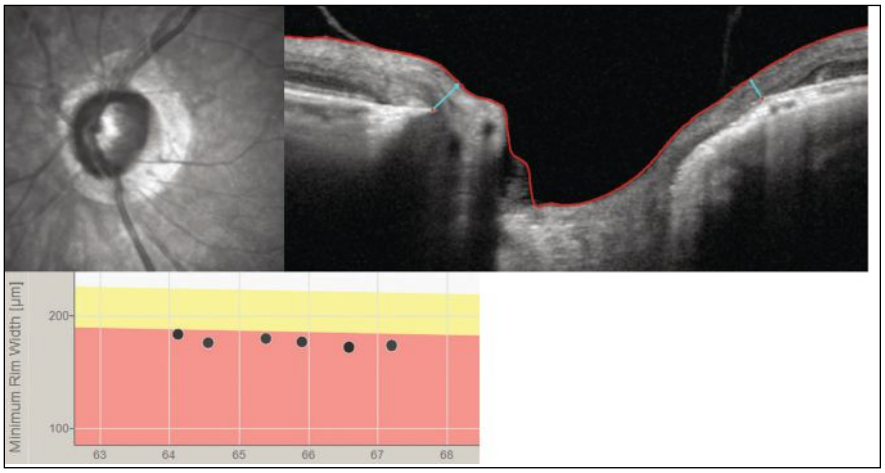
worse than OCT trend analysis when compared longitudinally.[39,40] For example, after five years of semiannual testing, use of the rule resulted in a false positive rate of 24.8%, signifying that it might result in unnecessary treatment in patients with stable disease, causing unnecessary burden.

Regardless of whether an informal rule or a more structured machine-based trend analysis is used, practitioners cannot over-rely on average RNFL trend or event analysis as a stand-alone; they need to be used in tandem to capture both local and global loss. Even then local defects may be missed, so grossly looking at how the tissue is captured and how it changes over time in deviation maps has value. Ultimately, don't get caught up in the minutiae of the data and make it impossible to comprehensively analyze all clinical information before deciding whether to intensify management.[41,42]



The BMO-MRW (light blue arrows) may be a better way to track progression than the RNFL in high myopes who are suspected of having glaucoma or in those with confirmed myopic glaucoma.

The Differentials

Glaucoma is a diagnosis of exclusion, albeit in most cases not an extremely difficult one. Among optic neuropathies, glaucoma is unique because it undergoes damage to both connective tissue and neural tissue, whereas other neuropathies result in only neural tissue damage. Glaucoma is a laminopathy.[43,44]

Accordingly, it is important to recognize typical patterns of tissue loss that might mimic glaucoma but are more consistent with non-glaucomatous neuropathies or retinopathies. The caveat is that there are confounding situations where discernment between glaucoma and non-glaucoma can be difficult. The list of potential differential diagnoses is substantial, so we will concentrate on a few major examples.

Non-arteritic anterior ischemic optic neuropathy (NAION). This is the most common cause of optic disc edema between the ages of 45 and 70 and may appear on the surface to share some features with glaucoma. Once ONH edema resolves, there are many potential presentations of neural tissue damage, most frequently pallor of the neuroretinal rim and axonal loss. NAION patients will have RNFL thinning as glaucoma patients do; however, the majority of RNFL thinning occurs in the superior and temporal quadrants in NAION and typically stabilizes six months post-inception on average.[45,46] To differentiate from OCT, patients with NAION will not have BMO-MRW thinning or lamina cribrosa thinning as they do in glaucoma.[47,48]

Diabetic retinopathy. The population-based Beijing Eye Study showed that localized RNFL defects occurred in 15% of screened patients on OCT, they are not uncommon and it is important to recognize that they are not always indicative of glaucoma. Non-glaucomatous causes of wedges were myopia/higher axial length, diabetes and other vascular conditions resulting in RNFL infarct and age.[49] Concentrating on diabetic retinopathy, specifically pre-capillary focal arterial occlusions within the RNFL, the angular width of the RNFL defect and its behavior over time can help differentiate from glaucoma. One study showed that the angular width of localized RNFL defects was substantially smaller in patients with diabetes when compared with their normal-tension glaucoma counterparts (7.7±6.5° vs. 43.5±19.2°). Furthermore, the defect enlarged over time in the glaucoma group.[50] So, initial assessment of the defect width—the wider it is, the more likely glaucoma is present—and longitudinal OCT analysis can help discern glaucomatous damage from diabetic damage if a localized infarct has not been witnessed or documented.

When distinguishing between larger caliber vessel occlusions and glaucoma, a study showed that comparing the superior and inferior hemispheres of the macula can be helpful. The authors found that greater global intra-eye asymmetry and tissue thickness, as well as total retinal thickness below 200µm in individual 3°x3° cubes, were diagnostic of previous artery occlusion and not glaucoma.[51]

Myopia. Patients with high myopia are at a two- to threefold increased risk of open-angle glaucoma, and myopic nerves should thus be assessed cautiously.[52] To complicate matters, commercially available SD-OCT reference databases do not incorporate axial length-related ocular magnification, meaning that the longer the axial length, the thinner the RNFL and the smaller the ONH.[53] Accordingly, many eyes will fall into a statistically abnormal range per the machine, resulting in a false diagnosis of glaucoma when they in fact have healthy nerves.[54] To avoid false positives, specifically in myopes between the range of -2.00D

to -5.00D, consider the temporal raphe sign in the GC-IPL or MRW scan, which has been shown to have much greater specificity (95% vs. 33%) when compared with the RNFL or GC-IPL. This is due to an improved anatomical and geometrical accuracy in these patients.[55-57] Although the aforementioned are better options than the RNFL in moderate and high myopia, it should be noted that there are some cases where none of these parameters can be meaningfully used as some eyes are just too atypical to allow consistent OCT structural capture.

Closing Thoughts

Remaining objective is a difficult skill when considering inter- and intrapersonal assessments of structural changes in the RNFL and ONH. OCT can maintain a level of reproducible objectivity that otherwise plagues even the most sagacious of clinicians. This consideration is important especially when data may not fit perfectly together. Remain objective, consider potential artifacts and differentials and remember that OCTs are devices that require good data acquisition to produce insightful information for the best outcomes. ■

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OCT: AN INDISPENSABLE TOOL IN RETINA CARE

Learn how to use the plethora of clinical data provided by this technology to help detect and assess dozens of posterior segment conditions.

BY JESSICA HAYNES, OD, AND MOHAMMAD RAFIEETARY, OD
GERMANTOWN, TN

Imaging with optical coherence tomography (OCT) has become standard in retina care. Its widespread use can be attributed to its ability to deliver a vast amount of high-resolution information regarding the structure of the retina both quickly and safely. A strength of OCT is its ability to segment various retina layers, which enables cross-sectional structural examination of the retina along with evaluation of various analytic data such as thickness maps extracted from OCT scans (*Figure 1*).[1] Using this advanced technology, we can diagnose posterior segment disease earlier and more accurately, identify biomarkers that indicate severity of disease and deepen our understanding of the pathological processes that affect the retina.

Here, we'll discuss how to spot and analyze OCT findings associated with a wide range of retinal pathologies. Keep in mind that this list only scratches the surface of the abundance of information the technology can offer.

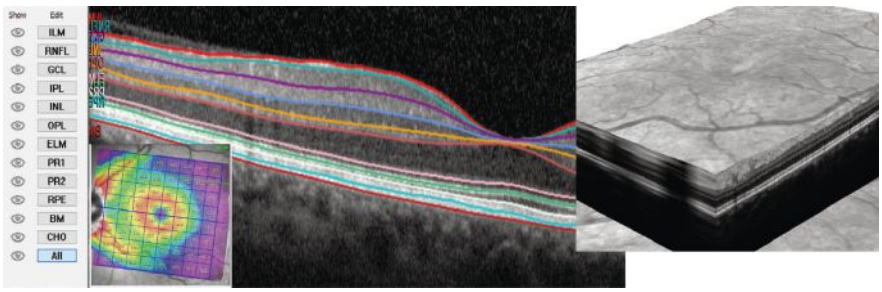

Fig. 1. OCT segmentation allows for various manipulation of images and algorithms for measurable analytics.

Age-related Macular Degeneration (AMD)

A wealth of data can be uncovered when evaluating the OCT cross-section in a patient with confirmed or suspected AMD to better stage the disease and consider the patient's risk of progression. While most clinicians are aware to look for the presence of drusen on OCT cross-section scans, a variety of additional biomarkers of disease severity can be spotted in regard to atrophic or nonexudative AMD.

Reticular pseudodrusen (RPD), also called subretinal drusenoid deposits, are a drusen phenotype associated with higher risk of conversion to advanced-stage AMD, particularly development of geographic atrophy (GA).[2,3] In addition, patients with RPD tend to have worse visual function. RPD present as hyperreflective deposits on OCT that sit above the retinal pigment epithelium (RPE), as opposed to typical drusen deposits, which are sub-RPE.[3] RPD may also be identified by their reticular pattern on imaging such as infrared

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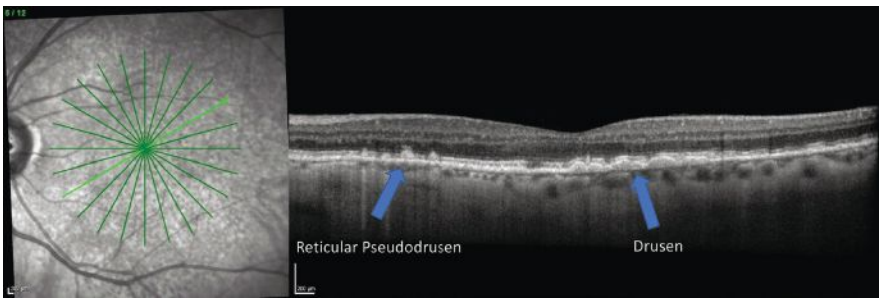


Fig. 2. A patient with both a typical drusen phenotype presenting on OCT as sub-RPE drusen deposits and RPD that present as hyperreflective deposits sitting on top of the RPE (right image). The infrared reflectance image (left) also demonstrates the reticular pattern consistent with RPD.

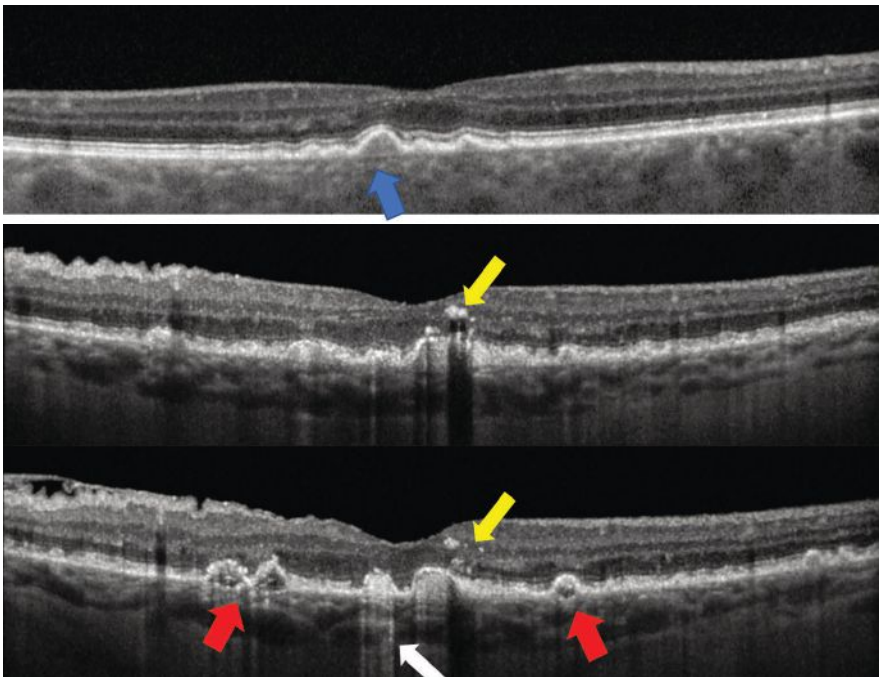


Fig. 3. The top image shows drusen with moderate, uniform internal reflectivity (blue arrow). The bottom two images show a patient with biomarkers of more advanced disease who presents with drusen of irregular, non-uniform internal reflectivity (red arrows), hyperreflective columns (white arrow) and hyperreflective foci (yellow arrows).

reflectance or fundus autofluorescence (FAF) (*Figure 2*).[2,3]

The internal structure of drusen as seen on OCT can also give clues to the severity of disease. Patients who have drusen with homogenous, uniform, moderate internal reflectivity have less risk to develop advanced-stage AMD than those with drusen that are internally hyperreflective, hyporeflective or those with non-uniform internal composition (*Figure 3*).[4-6]

AMD stages in the Beckman Scale rely on the size of the drusen. Using the line caliper on the OCT, you can make a more accurate assessment as to the stage of the AMD based on drusen size.

Hyperreflective foci are associated with higher risk of conversion to advanced-stage AMD.[7] They represent a more compromised RPE with anterior migration of pigment. Clinically, these may be observed as pigmentary abnormalities. On OCT, they present as hyperreflective points that are often found overlying large pigment epithelial detachments (PEDs) (*Figure 3*).[7]

Hyperreflective columns also represent RPE compromise. The RPE is highly reflective and creates an optical barrier for OCT light to penetrate into the choroid. As the RPE degenerates, this optical barrier is broken down and columns of light can cascade into the choroid. On OCT, this presents as hyperreflective light columns extending into the choroid. This may be a precursor to the development of GA (*Figure 3*).[7]

Incomplete RPE and outer retinal atrophy (iRORA) is a relatively new term defined by the Classification of Atrophy Meeting group (*Table 1*). This is a non-clinical finding that is defined purely by retinal imaging, primarily OCT. On OCT, iRORA presents as loss of outer retinal integrity that may include loss or thinning of the RPE, ellipsoid zone and outer nuclear layer; however, it doesn't yet meet the criteria of complete RPE and outer retinal atrophy (cRORA). iRORA is an important OCT finding, as it's a precursor to cRORA.[8,9]

OCT is also a valuable tool in the detection of GA, the presence of which constitutes advanced-stage nonexudative AMD. On OCT, GA

TABLE 1. DEFINITION OF IRORA[8]

iRORA is defined on OCT by meeting the following criteria:
1. A region of signal hypertransmission into the choroid **and**
2. A corresponding zone of attenuation or disruption of the RPE, with or without persistence of basal laminar deposits **and**
3. Evidence of overlying photoreceptor degeneration, i.e., subsidence of the inner nuclear and outer plexiform layers, presence of a hyporeflective wedge in the Henle fiber layer, thinning of the outer nuclear layer, disruption of the external limiting membrane or disintegrity of the ellipsoid zone, and when these criteria do not meet the definition of cRORA.

presents as a complete loss of the RPE, photoreceptor integrity line and atrophy of the outer nuclear layer. There is also increased light transmission into the choroid in regions of GA.

Historically, GA was defined as a clinical finding. More recently, the Classification of Atrophy Meeting group defined the term cRORA based on OCT, near infrared reflectance imaging and FAF (*Figure 4*). The criteria for a diagnosis of cRORA using OCT are described in *Table 2*.

While the terms "GA" and "cRORA" are similar, they are not entirely interchangeable.[9] For example, the term "GA" is suggested to be used only in the absence of macular neovascularization (MNV), whereas the term cRORA can be used whether there is or is not associated MNV.[9]

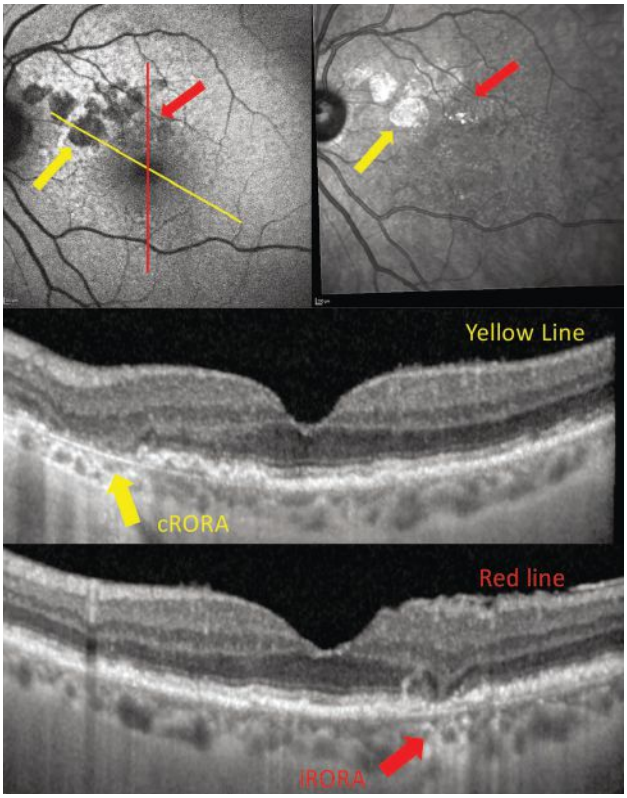


Fig. 4. OCT, FAF (top left) and infrared reflectance (top right) imaging of a patient with regions of both cRORA and iRORA.

OCT is highly sensitive in detecting signs of exudation that may constitute exudative AMD. These findings may be visualized on OCT prior to ophthalmoscopic detection or patient symptoms. When evaluating an OCT cross-section scan in a patient with AMD, be careful to note any presence of potential subretinal fluid (hyporeflective space between the RPE and neurosensory retina) and intraretinal fluid (hyporeflective cystic spaces in the neurosensory retina). These can be signs of exudation from MNV (*Figure 5*).

Another OCT-specific finding in MNV is subretinal hyperreflective material. This will present on the scan as moderately reflective, disheveled material between the neurosensory retina and the RPE. It can indicate exudative material from MNV (*Figure 5*). Presence of intraretinal fluid and/or subretinal hyperreflective material is related to poorer visual outcomes in patients with MNV.[10]

MNV occurs in four different types depending on the location and origin of the neovascular network. Type 1 is located beneath the RPE. Type 2 is located above the RPE. Type 3 or retinal angiomatous proliferation originates in the deep neurosensory retina, and type 4 is mixed type MNV. The variability of MNV presentation leads to a variety of presentations on OCT. For example, patients with type 1 MNV will present with a PED often with overlying subretinal fluid, while those with types 2 or 3 may not have a clearly visible PED and tend to have more subretinal hyperreflective material and intraretinal fluid. Due to these variations in presentation, it is

TABLE 2. DEFINITION OF CRORA[9]

cRORA is defined on OCT by meeting the following criteria using FAF, near infrared reflectance and color/multicolor imaging as confirmatory testing:

OCT	FAF	Near infrared reflectance	Color/Multicolor
Zone of hypertransmission of ≥250µm	Sharply demarcated borders	Sharply demarcated borders	Sharply demarcated borders
Zone of attenuation or disruption of RPE band of ≥250µm	Hypoautofluorescent	Hyperreflective	Hypopigmentation
Evidence of overlying photoreceptor degeneration whose features include outer nuclear layer thinning, external limiting membrane loss and ellipsoid zone or interdigitation zone loss	Diameter: ≥250µm; Area: 0.05mm^2	Diameter: ≥250µm; Area: 0.05mm^2	Diameter: ≥250µm; Area: 0.05mm^2
Exclude: scrolled RPE or other signs of RPE tear	Exclude: macular pigment or other artifact	Exclude: artifact	Increased visibility of choroidal vessels

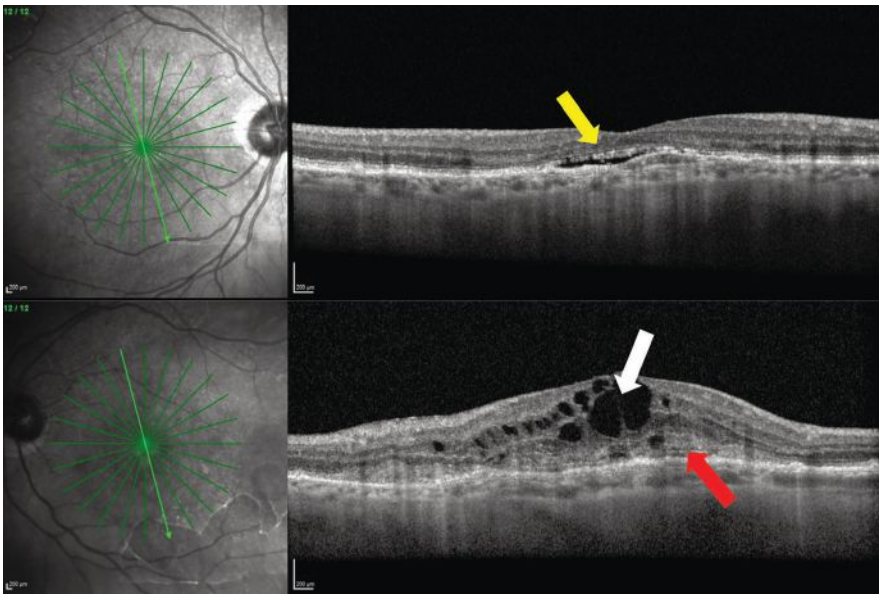
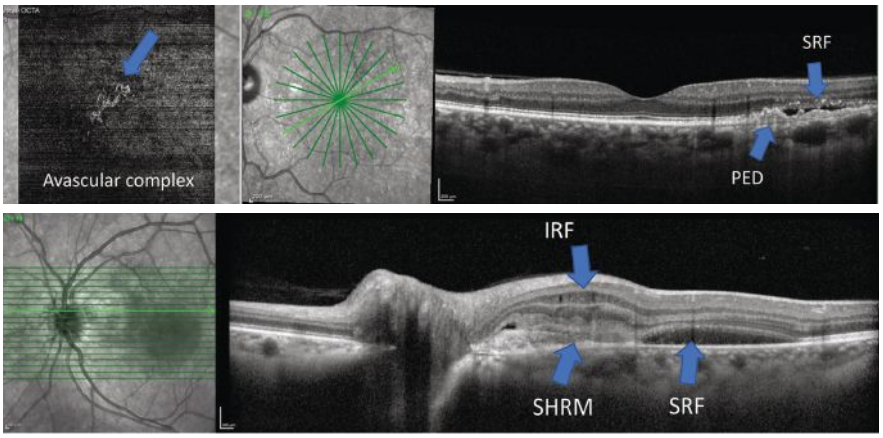


Fig. 5. A patient presents with bilateral MNV. The right eye (top images) has presence of subretinal fluid (yellow arrow). The left eye (bottom images) has intraretinal fluid (white arrow) and subretinal hyperreflective material (red arrow).



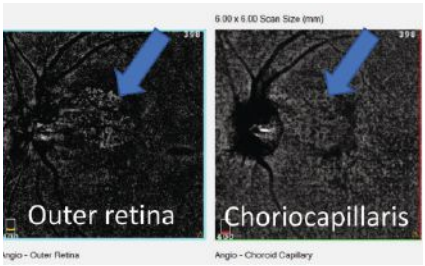


Fig. 6. Two non-AMD patients with MNV. Top image: A patient with pachychoroidal neovasculopathy with an elongated PED and subretinal fluid who has type 1 MNV. Bottom image: A patient with subretinal hyperreflective material, subretinal fluid and intraretinal fluid with type 2 peripapillary neovascularization.

important to consider any possible signs of exudation as a potential MNV—especially in those at risk (*i.e.*, patients with AMD) (*Figure 6*).[11]

OCT angiography (OCT-A) is also available on some OCT platforms and may aid in the diagnosis of MNV. On OCT-A, attention should be paid to the avascular complex and the choriocapillaris for presence of irregular vascular flow. OCT-A has brought about new categorization of MNV called subclinical or nonexudative MNV. This type of MNV presents as a PED that is often shallow and elongated. There is minimal or no sign of subretinal or intraretinal fluid on the OCT, but the PED is harboring MNV that is evident on OCT-A. These patients have a strong likelihood of developing exudation (*Figure 7*).[12]

Diabetic Retinopathy (DR)

In regards to DR, OCT has become the standard by which to diagnose and follow the course of diabetic macular edema (DME). Historically, DME was described as clinically significant or not based on criteria set forth in the Early Treatment DR Study. The term "clinically significant macular edema" has come to be outdated, with DME now being described as either center-involving (comprising the central 1000µm subfield) or non-center-involving based on OCT scans.[13]

Imaging with OCT can easily reveal cases of DME that aren't detectable on the clinical exam, and the topographical distribution of edema can be visualized through evaluation of retinal thickness maps (*Figure 8*). It's important to note that evaluating OCT cross-section scans is critical to arrive at an accurate DME diagnosis rather than relying on data from thickness, as DME is only one condition of many that may thicken the retina. It's important to look at the retinal thickness maps alongside the B-scans.

Findings such as exudate and cotton wool spots can also be identified on OCT. Exudates appear as hyperreflective deposits often within the neurosensory retina (specifically the outer plexiform layer) but can also accumulate in the subretinal space. Subretinal exudation in the fovea is an indicator of poor visual prognosis. Cotton wool spots appear as hyperreflective thickening in the retinal nerve fiber layer (RNFL).

Retinal hemorrhages may also be visible on OCT. Hemorrhage typically presents as a hyperreflective alteration on the OCT. Intraretinal hemorrhages that are common in DR may be difficult to distinguish on the OCT scan as they may blend into other hyperreflective retinal layers. However, one use for imaging over hemorrhages may be to determine their location. Patients with proliferative DR are prone to developing preretinal and vitreous

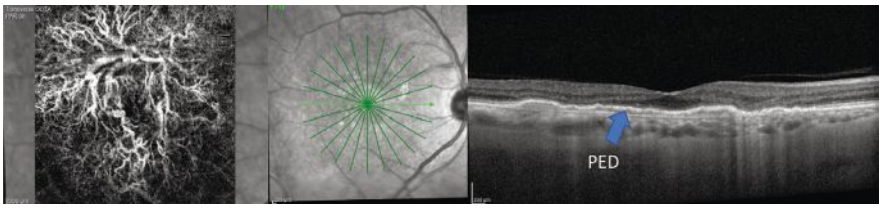


Fig. 7. AMD patient with a large, elongated PED and no sign of fluid on the OCT has an extensive MNV network present on the avascular complex of his OCT-A scan.

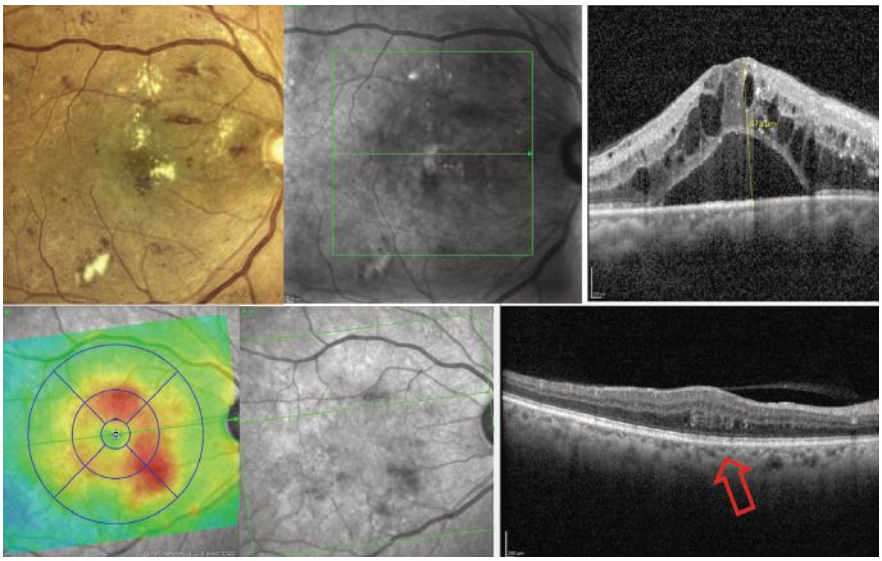


Fig. 8. OCT can be used to detect DME that is either clinically visible (top images) or not (bottom images). The bottom left image is a retinal thickness map that helps display the distribution of macular thickening from edema.

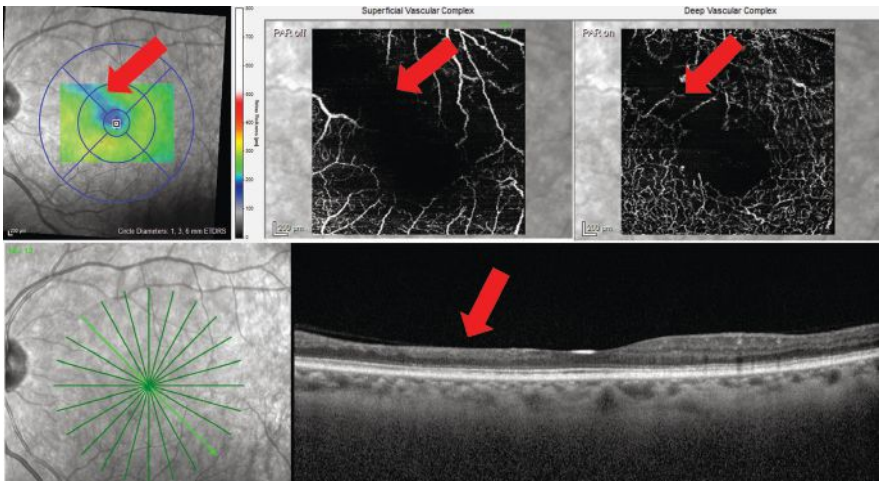


Fig. 9. A patient with DR presents with a localized area of inner retinal thinning on OCT cross-section scan. This corresponds to an area of macular thinning on the thickness map (top left image) as well as capillary non-perfusion in the superficial and deep capillary plexi (top middle and top right images).

hemorrhage as blood vessels from fragile retinal neovascular networks bleed on the surface of the retina and into the vitreous. Preretinal hemorrhages may present as hyperreflective deposits on the retinal surface between the RNFL and internal limiting membrane (ILM) or between the ILM and the posterior vitreous hyaloid. Vitreous hemorrhage may present as hyperreflective clumps in the vitreous, shadowing on the OCT image, or as small hyperreflective pinpoints within the vitreous (red blood cells).

In addition to the detection of DME, exudate and cotton wool spots, changes on the OCT cross-section scan can reveal clinically invisible data about your patient's disease severity. As we know, diabetes causes capillary damage that leads to a cascade of events with potential sight-threatening sequelae including DME and complications from neovascularization. Destruction of capillaries and capillary nonperfusion in the macula can also lead to decreased visual sensitivity and even severe central vision loss. As the retinal vasculature feeds the inner retina, its atrophy leads to OCT findings such as inner retinal atrophy and retinal thinning as well as disorganization of the retinal inner layers (DRIL).[14]

Macular thinning contributing to or found in DR is associated with macular non-perfusion on fluorescein angiography (FA) and OCT-A. This OCT biomarker is also associated with decreased visual function and more severe retinopathy (*Figure 9*). In addition to causing this frank thinning of the inner retinal layers, capillary non-perfusion can lead to DRIL.[15,16] This is another OCT-based finding where the regular and well-defined pattern of the inner retinal layers becomes disorganized with less regularity and indistinct retinal layers (*Figure 10*). Since DRIL is related to macular non-perfusion, its presence is also related to non-perfusion visualized on FA and OCT-A.

One unconventional use for OCT in diabetic retinopathy is identifying neovascularization of the disc or elsewhere. Traditionally, FA has been used to differentiate retinal neovascularization from other vascular abnormalities, such as intraretinal microvascular abnormalities (IRMA)

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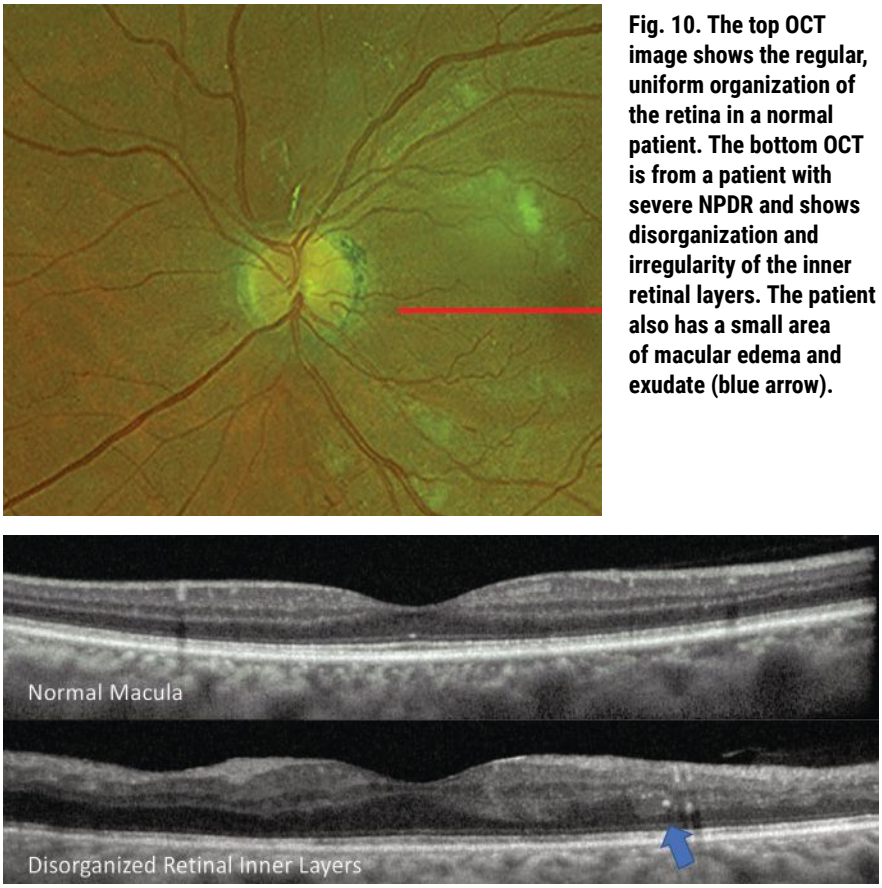

Fig. 10. The top OCT image shows the regular, uniform organization of the retina in a normal patient. The bottom OCT is from a patient with severe NPDR and shows disorganization and irregularity of the inner retinal layers. The patient also has a small area of macular edema and exudate (blue arrow).

and shunt vessels, by evaluating the vascular permeability of the vessels. Neovascularization leaks prominently on FA. More recently, OCT-A has been helpful in making this distinction by determining the location of the vasculature: IRMA is intraretinal vasculature while retinal neovascularization exists on the retinal surface and vitreoretinal interface. However, OCT itself may also be helpful. Retinal neovascularization grows in optically dense networks and presents as hyperreflective fibrotic opacities on the retinal surface or vitreous—often on the back surface of the posterior vitreous cortex.[17]

Central Serous Retinopathy (CSR)

Two improvements in OCT imaging that had a major impact on how we monitor and detect retinal disease include enhanced-depth imaging (EDI) and swept-source OCT (SS-OCT). EDI-OCT is a modified method of imaging acquisition with spectral-domain (SD)-OCT to provide deeper cuts, improving the structural details of the choroid. Using a different laser with a wavelength of 1050nm, which is 180nm longer than that used in SD-OCT, SS-OCT enables greater tissue penetration and better visibility of the choroid.[18]

These two innovations in OCT revealed information about the choroid that previously remained unattainable, leading to an entire spectrum of disease: the pachychoroidal disease spectrum. This includes pachychoroid pigment epitheliopathy; pachychoroid neovasculopathy, polypoidal choroidal vasculopathy and peripapillary pachychoroid syndrome, as well as CSR. Classic OCT findings in acute CSR include PED (not always present) with overlying or adjacent subretinal fluid. As the disease becomes chronic, OCT findings such as shaggy photoreceptors and intraretinal fluid may be present (*Figure 11*).[19]

Evaluation of the choroid with OCT is important in the diagnosis of CSR and other pachychoroidal diseases. Choroidal thickness varies based on many factors including age and refractive error, making it difficult to define a "typical" choroidal thickness. Reports of normal subfoveal choroidal thickness vary from 190µm to 350µm. In general, patients with CSR tend to have thicker than average subfoveal choroid or pachychoroid; however, they may have relatively normal subfoveal thickness and have localized regions of pachychoroid. In addition to the strict thickness of the choroid, the characteristics of choroidal vessels can also be a clue as to the disease process at hand. Pachyvessels, or vessels with abnormally large lumen, may be present, particularly in the deeper Haller's layer of the choroid. These vessels may displace smaller vessels in the more superficial Sattler's layer and choriocapillaris (*Figure 11*).[19]

Posterior Segment Tumors

The same advancements in technology that allow better visualization of the choroid with OCT have also improved our ability to evaluate choroidal lesions. OCT imaging of posterior segment lesions can help to confirm the location of a tumor which may affect the retina, RPE or choroid (*Figure 12*). In addition, OCT imaging adds value in the ever-important distinction between a benign choroidal nevus and a choroidal melanoma. In smaller lesions, OCT imaging allows for evaluation of anterior-posterior thickness and can help monitor thickness changes over time.

OCT scans also play a very important role in the detection of subretinal fluid, which is an important risk factor for choroidal melanoma. Chronic subretinal fluid can lead to the presence of shaggy photoreceptors, another negative prognostic factor.[20] OCT is more sensitive in the detection of subretinal fluid than clinical examination and ultrasonography (*Figure 13*). Multimodal

imaging of choroidal lesions is now advised with use of fundus photography, ultrasonography, OCT and FAF.[21]

Vitreomacular Interface Findings and Abnormalities

The next few sections discuss a variety of vitreomacular and vitreoretinal interface findings that can be revealed upon OCT examination. Note that when assessing this layer of the retina, it's best not to use EDI to maximize inner retinal resolution.

Vitreomacular Adhesion (VMA)

This complication has been defined as a partial separation of the posterior vitreous cortex from the macular surface in the perifoveal region without any alteration to the macular or foveal structure.[22] This could be considered a normal state of aging as there is forward prolapse of the posterior vitreous cortex during the development of posterior vitreous detachment (PVD). However, this process of normal aging can become anomalous, leading to a disease state such as vitreomacular traction (VMT), which we'll talk about next (*Figure 14*).[23] Once VMA is detected on OCT, it should be followed periodically to assess for further pathologic findings.

VMT

This common finding discovered by the advent of SD-OCT, is an abnormal PVD with persistent attachment resulting in an anatomic alteration or deformity over the area within 3mm of fovea.[23] VMT can spontaneously resolve without consequence (*Figure 15*). However, a focal (≤1500µm) VMT over the fovea can result in development of a partial-, lamellar- or full-thickness macular hole, while a board VMT (>1500µm) can progress to the epiretinal (or epimacular) membrane (*Figures 16 and 17*).[24]

Macular Hole

As previously mentioned, macular holes form as a result of abnormal vitreous traction as a consequence of PVD. The old staging of various macular holes has been somewhat replaced by the evaluation of their configuration as detected by SD-OCT. There are many different shapes which can be marked as partial- vs full-thickness forms (*Figure 16*).

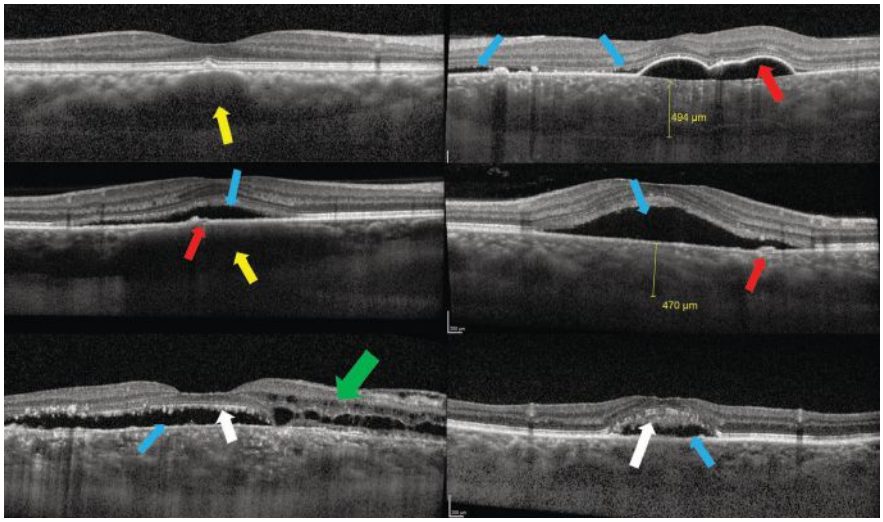

Fig. 11. Top left: Patient with pachydrusen overlying a pachyvessel (yellow arrow) with compression of Sattler's layer and choriocapillaris. Top right: Patient with chronic case of CSR presents with pachychoroid, large serous PED (red arrow) and subretinal fluid (blue arrows). Middle left: Acute case of CSR presents with small PED (red arrow) and subretinal fluid (blue arrow) with underlying pachyvessel (yellow arrow) with compression of Sattler's layer and choriocapillaris. Middle right: Acute case of CSR with pachychoroid, small PED (red arrow) and subretinal fluid (blue arrow). Bottom left and right: Chronic CSR presents with subretinal fluid (blue arrows) as well as intraretinal fluid (green arrow) and photoreceptor outer segment elongation or "shaggy photoreceptors" (white arrows).

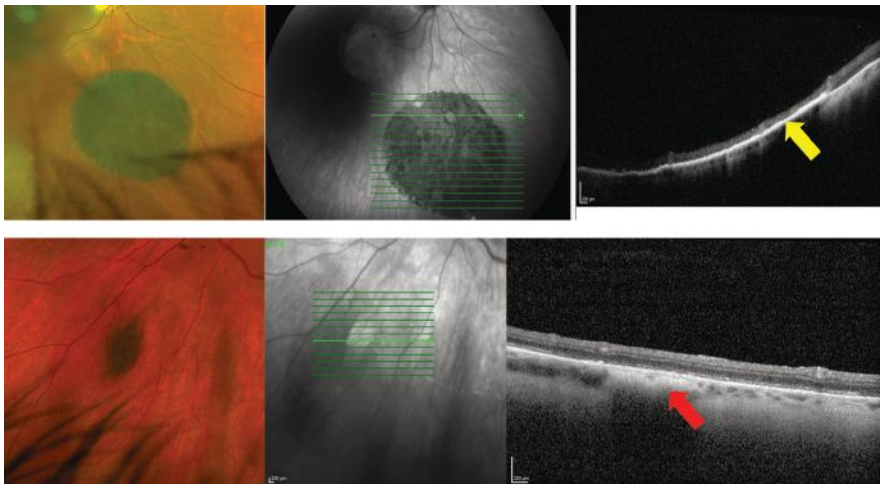

Fig. 12. OCT can be used to differentiate between pigmented lesions of the posterior segment, such as congenital hypertrophy of the RPE (CHRPE), which are lesions of the RPE (top images), and choroidal nevi (bottom images). This pigmented CHRPE shows hyperreflectivity and thickening of the RPE with posterior shadowing but no involvement of the choroid (yellow arrow). The small nevus shows hyperreflectivity and posterior shadowing within the choroid (red arrow).

The management of macular holes primarily depends upon the patient's visual symptoms plus hole configuration. While many partial-thickness holes are asymptomatic and can be clinically monitored, full-thickness macular holes usually require surgical intervention.[25]

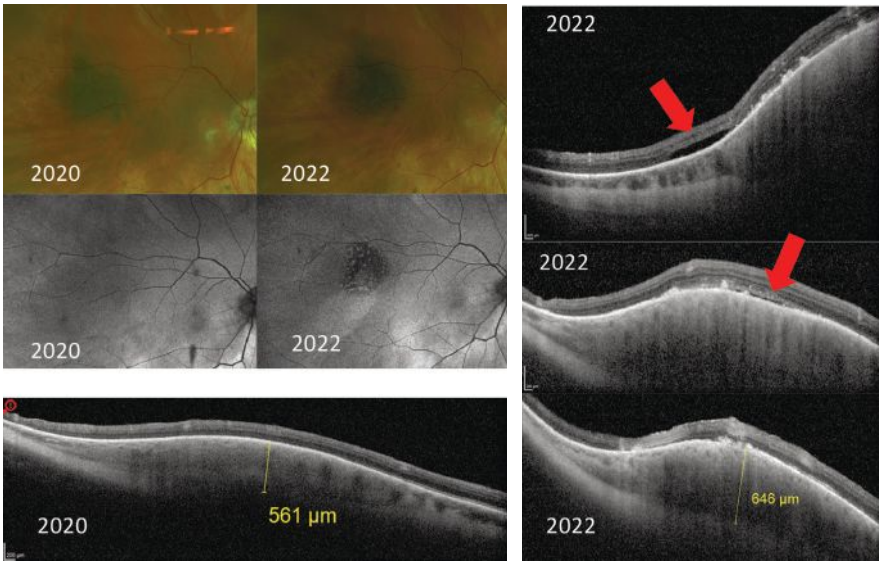

Fig. 13. A patient presents with a choroidal nevus in 2020 with normal overlying autofluorescence, total thickness on OCT of 561µm and no sign of subretinal fluid on OCT. In 2022, the lesion has mild growth in diameter, but there are concerning findings of hyper-autofluorescence on FAF, growth in thickness shown on OCT and new presence of subretinal fluid on OCT (red arrows). These findings are concerning due to the risk of conversion to ocular melanoma.

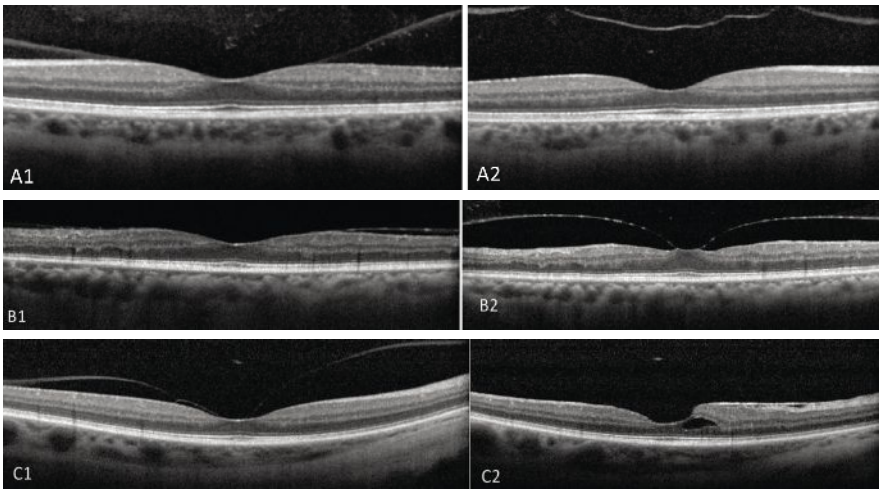

Fig. 14. In the first row of images, VMA (A1) leads to PVD (A2) without consequence. In another case, VMA (B1) progresses to focal VMT (B2). In the third case, VMA (C1) after release results in epiretinal membrane and small foveal schisis (C2).

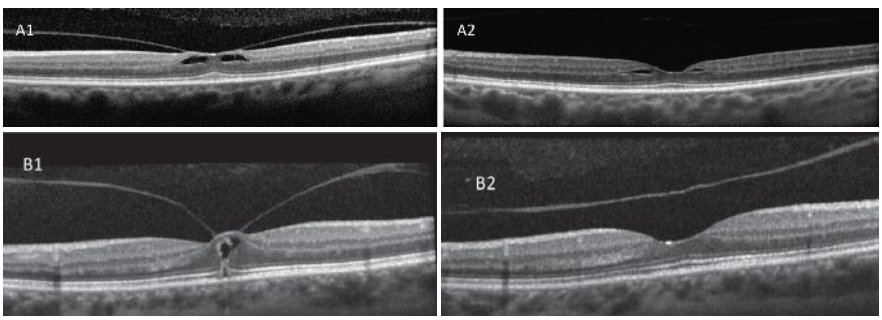

Fig. 15. Local VMT with foveoschisis (A1) spontaneously releases with improved macular contour (A2). Focal VMT with macular hole (B1) spontaneously released with normalized macular contour (B2).

Epiretinal and Epimacular Membranes

Epiretinal membranes are glial cell proliferations that develop at the vitreomacular interface. These are also typically post-PVD findings that can result in different visual symptoms like blurred vision and metamorphopsia with varying degrees based on the severity of the disease.

On SD-OCT, a hyperreflective band on the macular surface can be detected which results in tractional thickness of the retina, with surface folds or striae and often an inversion of the foveal contour can be seen (*Figure 17*). The radial traction by epimacular membranes can also result in separation of retinal layers leading to various degrees of schisis, often misinterpreted as macular edema (*Figure 17*). Partial-thickness macular holes are also not uncommon in the presence of epiretinal or epimacular membranes. Surgical intervention in these cases depends on patients' visual symptoms as well as the degree of dysmorphic macular changes.[26]

Takeaway

OCT imaging has provided us with a wealth of new information on a variety of retinal disease states. The findings described above represent only a small sampling of the abundance of data that OCT provides, which is what makes this clinical tool indispensable in retina care.

The ability to view and analyze the structure of the retina in this way has revolutionized how disease is detected and managed. When possible, clinicians should take advantage of the available technology to provide a higher level of care to their patients. ■

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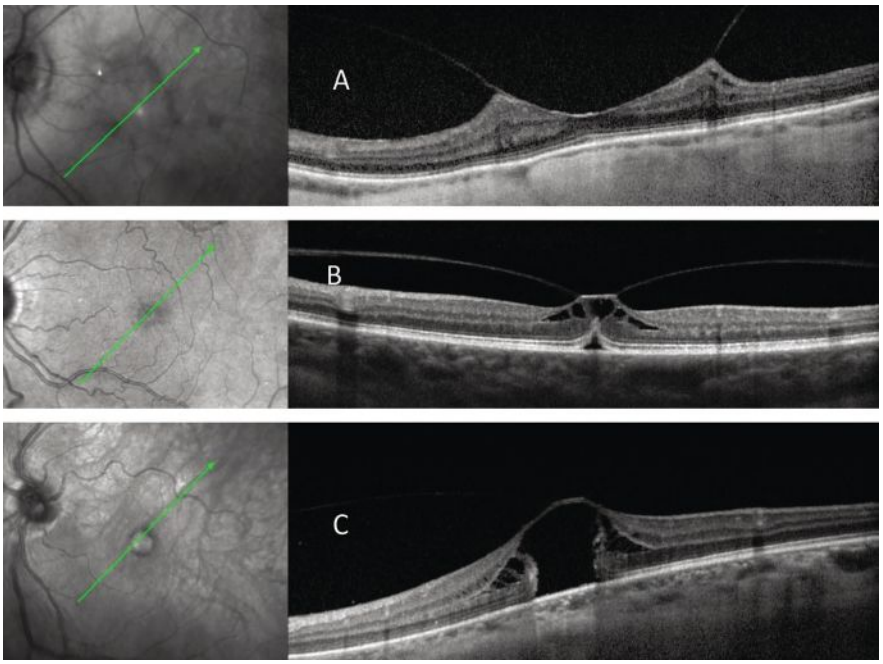

Fig. 16. Broad VMT with epiretinal membrane (A) vs. focal VMT (B and C) with two different macular hole configurations.

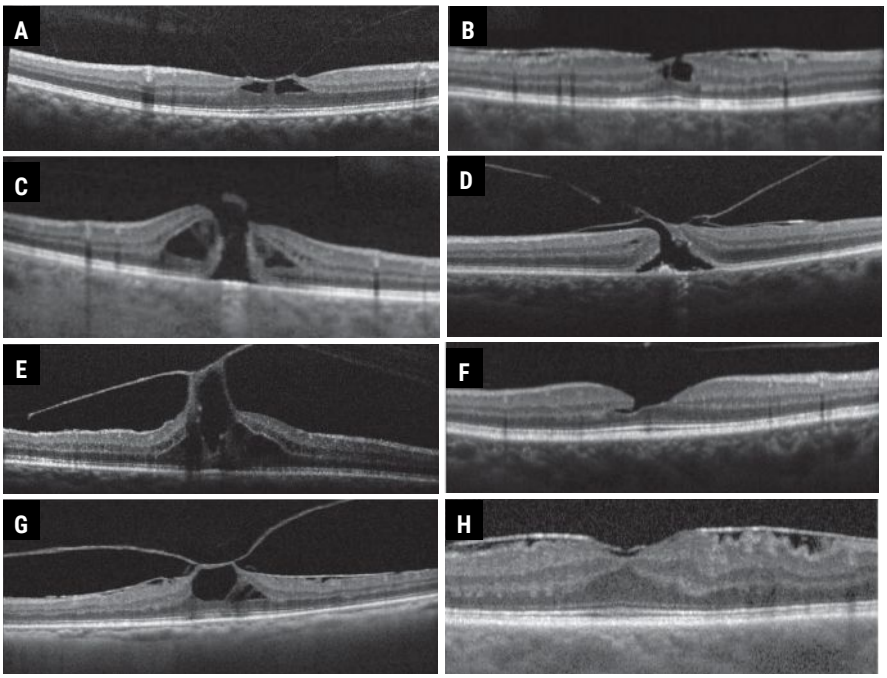

Fig. 17. VMT (A) taxonomy leading to one of the following potential clinical courses: epiretinal or epimacular membrane and lamellar macular hole (B), full-thickness macular hole (C), full-thickness macular hole with epimacular membrane (D), persistent VMT combined with macular hole (E), or released with lamellar macular hole (F), or persistent with epimacular membrane and macular hole (G) or epimacular membrane (H).

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SIX QUESTIONS ABOUT THE ROLE OF OCT IN NEURO EVALUATIONS

This technology plays a pivotal role in clinical practice.
With answers to these key points, learn how it can help you assess these complex conditions.

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Approaching neuro-ophthalmic disease may be daunting for many eyecare practitioners, given the wide array of possible differential diagnoses and systemic implications. With the ability to visualize structural changes from the retinal nerve fiber layer (RNFL) to the posterior aspects of the lamina cribrosa, optical coherence tomography (OCT) is an indispensable tool for disease screening, differential diagnosis, prognostication and progression analysis for patients with optic nerve disease. This article will answer six neuro-ophthalmic questions optometrists may face in clinical practice with a focus on the role of OCT in clinical decision-making.

1. Can OCT explain non-pathological field loss?

When a patient presents with bitemporal visual field loss, often the first thing that comes to mind is chiasmal syndrome—but is this always the case? Tilted disc syndrome is a benign physiological anomaly that presents with some characteristic clinical features. These include tilt or oblique insertion of the optic nerve, situs inversus of major blood vessels exiting the disc and posterior bowing of the retina in an area surrounding the optic disc.[1]

Tilted disc syndrome can also present with refractive visual field loss corresponding to the area of retinal ectasia when defocus between the retinal plane and corrective lens results in a refractive field defect.[2] As tilted disc syndrome is hypothesized to arise from a fault in embryonic fissure closure, retinal ectasia typically manifests in the inferotemporal aspect of the retina, and thus superotemporal visual field defects are most commonly observed in tilted disc syndrome.[3,4] While less common, other defects, such as altitudinal or hemianopic ones, have also been reported.[5] An example of tilted disc syndrome is shown in *Figure 1*.

To confirm the refractive nature of the defect, negative addition lens testing can be applied (usually a -3.00D negative addition lens is placed on top of the calculated testing refraction).[2,6] This results in an improvement of the sensitivity values and probability scores within the region of ectasia, as the new lens results in divergence of light rays to focus more precisely onto the ectatic retina.[2]

While negative addition lens testing is an effective method for differentiating between pathological and refractive visual field loss, imaging can also be used to guide the process of differentiation by characterizing the retinal profile matching the visual field defect location.[2,6] The primary goal of imaging in tilted disc syndrome is to characterize the region of posterior bowing corresponding to the visual field defect. This can be achieved using B-scan ultrasound, which provides an extensive view of the globe and its morphology, whereby the posterior staphyloma corresponds with an outpouching of the retinal profile.[7]

As many optometric practices may not readily have access to B-scan

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ultrasounds, OCT can be a suitable alternative.[6] On OCT, the staphyloma will manifest as posterior sloping of the retinal profile. A wide, qualitative line scan placed through the region of suspected ectasia is the ideal scan type (*e.g.*, scan the inferonasal retina for superotemporal field defect). Thus, qualitative assessment of the retinal profile on posterior pole OCT line scans can identify variations in the retinal slope to explain visual field loss due to dioptric defocus, rather than true pathology.

2. How can OCT assess for complications associated with congenital optic disc pits?

Similar to tilted disc syndrome, optic disc pits arise from defective embryonic fissure closure.[9] They are often encountered incidentally upon routine examination, presenting as focal gray or white excavations of the optic nerve.[9,10] While they are most commonly located in the inferotemporal quadrant of the disc, they can also present in other locations.[10] Radial OCT line scans through the disc can be helpful in characterizing the profile and extent of the pit.[11] On OCT, optic disc pits present as focal loss of the lamina cribrosa with associated tissue herniation (*Figure 2A*).[9] The prognosis of optic disc pits can vary based on the accompanying clinical features, ranging from associated maculopathy to RNFL.

While the pathogenesis of optic disc pit maculopathy is still not well-understood, it is characterized by the accumulation of intraretinal or subretinal fluid as well as retinal pigmentary changes (*Figure 2B*).[13,14] It can present in 25% to 75% of patients with optic disc pits and has the potential to result in progressive visual impairment if left untreated.[15] Patients with optic disc pits presenting with reduced visual acuity or symptoms of metamorphopsia would benefit from a macular OCT scan to exclude the presence of associated maculopathy.

Another potential complication associated with optic disc pits are papillomacular RNFL defects.[16,17] These are thought to arise from incomplete fusion of the temporal retinal nerve fibers, resulting in the absence of RNFL in the papillomacular region (*Figure 2C*).[16]

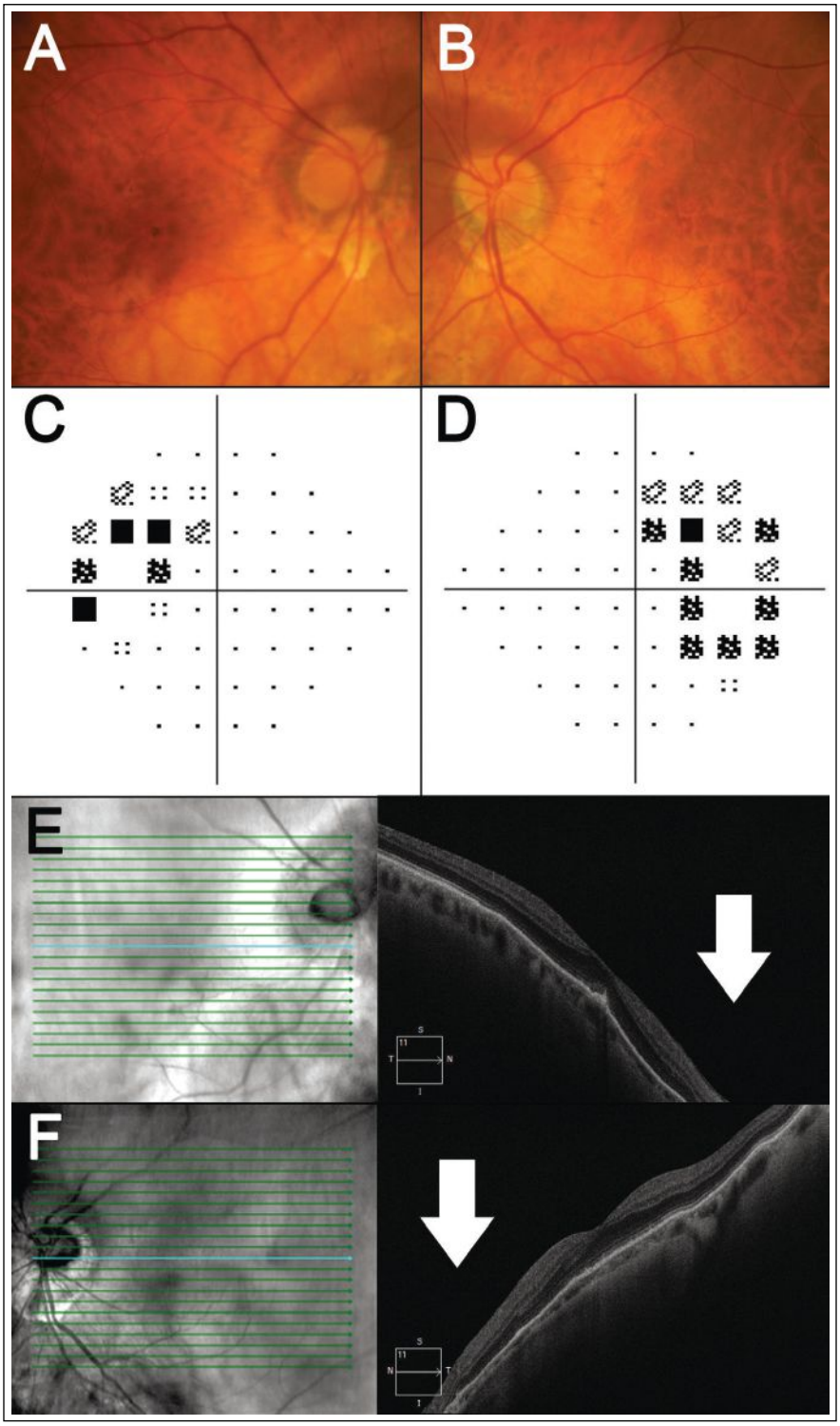

Fig. 1. A patient with bilateral tilted disc syndrome. (A-B) Fundus photography shows tilt and/or oblique insertion of the discs in both eyes with situs invertus of the blood vessels. (C-D) Here, 24-2 visual field testing shows a bitemporal visual field defect. (E-F) Horizontal OCT line scans through the fovea show posterior bowing of the nasal retina relative to the fovea (white arrows), corresponding to the temporal visual field defects.

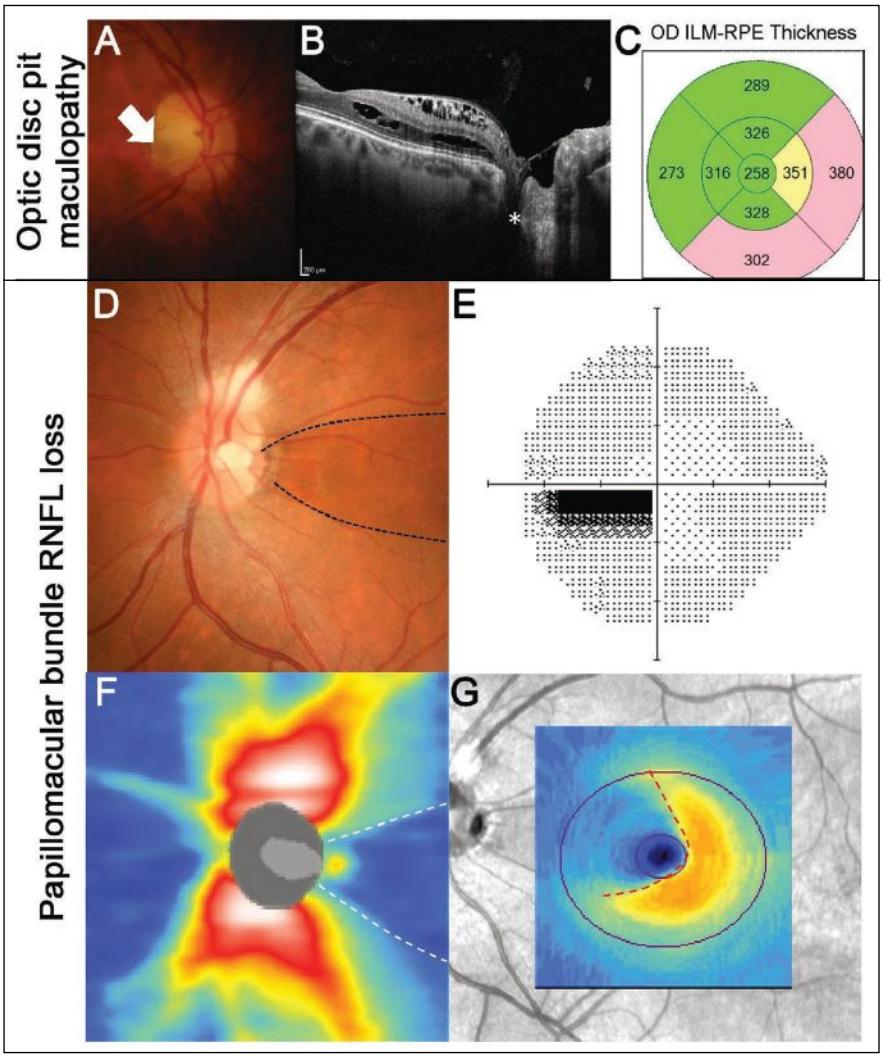

Fig. 2. Example of maculopathy associated with an optic disc pit. (A) Fundus photography shows an optic disc pit temporally (white arrow) in the right eye. (B) OCT line scans show the disc pit (white asterisk) with associated intraretinal fluid accumulation extending from the disc to the fovea. (C) EDTRS macular thickness grid shows thickening of the nasal and inferior subfields corresponding to the regions of fluid. (D-G) An example of papillomacular bundle loss associated with an optic disc pit (black dashed lines). (D) Fundus photography shows a temporal disc pit. Note there is also RNFL myelination superiorly. (E) A 24-2 visual field test showed a centrocecal defect in the left eye, corresponding to the RNFL defect. (F) OCT RNFL thickness heat map shows reduced RNFL thickness temporally (white dashed lines). (G) Macular ganglion cell analysis shows nasal loss (red dashed lines).

Visual function in these patients is dependent on the extent of RNFL loss. This highlights the importance of quantitative OCT, such as the thickness heat maps and deviation maps, as well as peripapillary ring scans (also called scan circle) to evaluate the depth and extent of associated loss. Unlike glaucoma, RNFL loss associated with optic disc pits is not progressive, and thus observation alone is sufficient for these patients.[16]

Remember: when examining patients with optic disc pits, both qualitative and quantitative OCT are crucial to assess for associated macular or RNFL complications, particularly for patients with visual symptoms.

3. How can OCT differentiate between various types of optic atrophy?

RNFL loss is a common feature across several forms of optic atrophy, including congenital conditions, ischemic optic nerve disease and hereditary optic neuropathies.[18] Although these conditions share several overlapping clinical features, careful inspection of quantitative OCT outputs can provide clinicians with a means of differentiating between them.

Localized retinal ischemic events such as cotton wool spots or branch retinal occlusions can result in colocalized deep and focal loss of the RNFL and ganglion cell layer.[19] In these instances, the pattern of loss follows the RNFL bundle trajectories and corresponds to location of ischemic insult (*Figure 3A-C*).

Qualitative OCT line scans can also highlight inner retinal thinning and disorganisation associated with ischemic retinal damage.[20] As expected from a localized insult, the corresponding RNFL loss is deep, focal and well-defined.

In contrast with localized ischemic RNFL loss, the pattern of loss in congenital conditions, such as superior segmental optic nerve hypoplasia (SSONH), present with a more widespread and deep pattern of loss, often with a nasal predilection.[21,22] SSONH, also known as 'topless disc syndrome,' presents with four distinct features in the superior aspect of the disc: displacement of the central retinal artery, disc pallor, a scleral halo and RNFL loss (*Figure 3D-F*).[23]

The location of RNFL loss can also be used in the process of differential diagnosis. For example, Leber's hereditary optic neuropathy is an autosomal dominant optic atrophy that presents with focal temporal disc pallor and corresponding temporal focal wedge-like RNFL loss (*Figure 3G-I*).[24,25]

OCT thickness heat and deviation maps are useful for characterizing the depth and pattern of loss, while peripapillary RNFL scan circle provide quantitative information for progression analyses. Unlike hereditary optic neuropathies, congenital and ischemic RNFL loss are typically non-progressive when monitored longitudinally.[16,19] Overall, it is important to relate the

expected pattern of RNFL loss with the understanding of underlying pathological process for accurate differentiation.

4. Can OCT differentiate between different causes of disc elevation?

While not diagnostic, OCT can be a useful tool to guide differentiation of papilledema, optic disc drusen and RNFL protrusion secondary to congenitally crowded optic discs. With careful evaluation of OCT line scans, key features differentiating causes of elevated optic discs can be observed. As these findings can be quite subtle, high definition and high density scans with enhanced depth imaging protocols or swept-source OCT may be required to improve visualization.

With papilledema, Bruch's membrane—just adjacent to the Bruch's membrane opening—can protrude anteriorly towards the vitreous due to the increased intracranial pressure causing forward distension.[26-28] There may also be intraretinal cystic spaces, due to either fluid leakage or mechanical retinal stretching over the elevated disc.[26-28]

In contrast, superficial optic disc drusen can be observed as oval, hyporeflective lesions with hyperreflective margins and are located within the optic nerve.[26,29] Horizontal hyperreflective lines observed within the optic nerve may indicate an earlier variant consistent with buried drusen.[29] Each of these features is absent in congenitally crowded discs.[30]

An OCT feature that may be observed in cases of elevated optic discs is peripapillary hyperreflective ovoid mass-like structures (PHOMS), which are thought to represent herniated retinal ganglion cell axons.[31] As PHOMS are a nonspecific finding, they are not particularly useful for differential diagnosis of the various causes of elevated optic discs (*Figure 4*).

While quantitative peripapillary RNFL data is less useful in differentiating these conditions in a cross-sectional nature, this data proves to be much more useful in longitudinal assessments because of their varying natural histories. Peripapillary increases in RNFL thickness, particularly along the superior, nasal or inferior margins, may be a warning sign of progressive papilloedema, and conversely, reductions in RNFL thickness at these locations can indicate a prior instance of papilledema. Peripapillary increases in RNFL thickness can also occur as optic disc drusen become more superficial; however, this would be accompanied by the OCT line scan findings outlined above.

Further longitudinal changes associated with optic disc drusen include development and/or progression in arcuate RNFL defects that resemble those observed in cases of glaucoma. In contrast, peripapillary RNFL scans from congenitally crowded optic discs should remain stable over time. Overall, it is important to use qualitative OCT line scans and peripapillary RNFL measurements in cases of disc elevation. This is because there are distinct advantages of each technique in various stages of assessment.

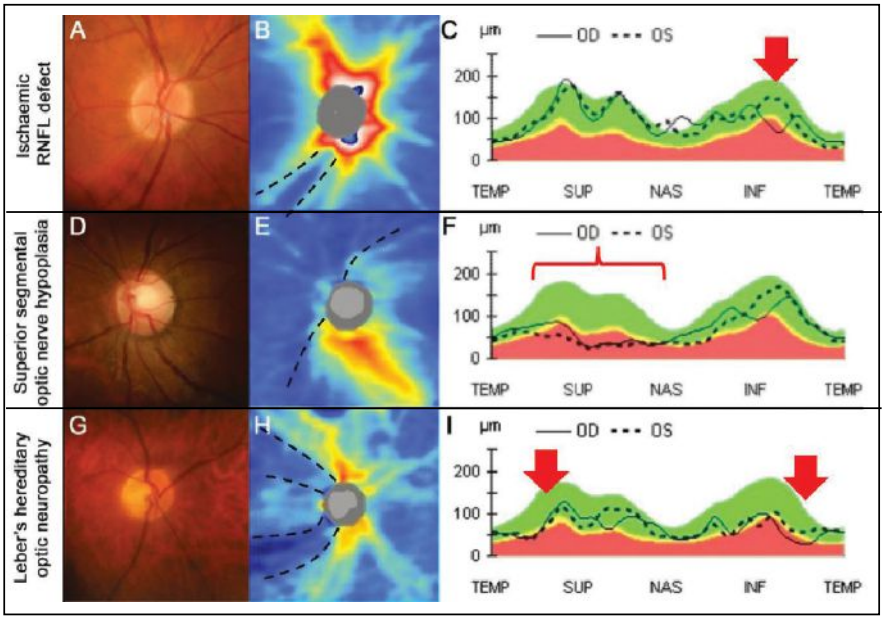

Fig. 3. An example of an ischemic RNFL defect. (A) The right disc has an intact neuroretinal rim with a deep inferior RNFL defect inferiorly. (B) OCT thickness heat map shows deep and focal RNFL thinning that is not contiguous with the disc inferotemporal (black dashed lines). (C) RNFL TSNIT graph shows focal and steep loss of the RNFL inferotemporally in the left eye (red arrow). (D-F) An example of superior segmental optic nerve hypoplasia (SSONH). (D) Classic funduscopic features of SSONH (superior displacement of the central retinal artery with a superior scleral halo. (E) OCT thickness heat map shows diffuse and deep RNFL thinning extending nasally to superiorly in an anti-clockwise manner (black dashed lines). (F) RNFL TSNIT graph shows generalized thinning in the nasal and superior aspects (red bracket). (G-I) An example of Leber's hereditary optic neuropathy. (G) The right disc shows temporal pallor with an otherwise intact neuroretinal rim. (H) OCT thickness heat map shows deep superotemporal and inferotemporal thinning of the RNFL (black dashed lines). (I) RNFL TSNIT graph shows RNFL thinning, more marked inferotemporally than superotemporally (red arrows).

5. How can OCT be used to localize lesions along the visual pathway?

Cerebral cortical lesions of at least four weeks' duration can often be observed on OCT imaging due to retrograde degeneration, eventually affecting the retinal ganglion cells.[32] If the cortical lesion is located posterior to the lateral geniculate nucleus, resulting degeneration would cross the synapses connecting the retinal ganglion cell axons and the optic tracts. Hence, this process is sometimes referred to as trans-synaptic retrograde generation.

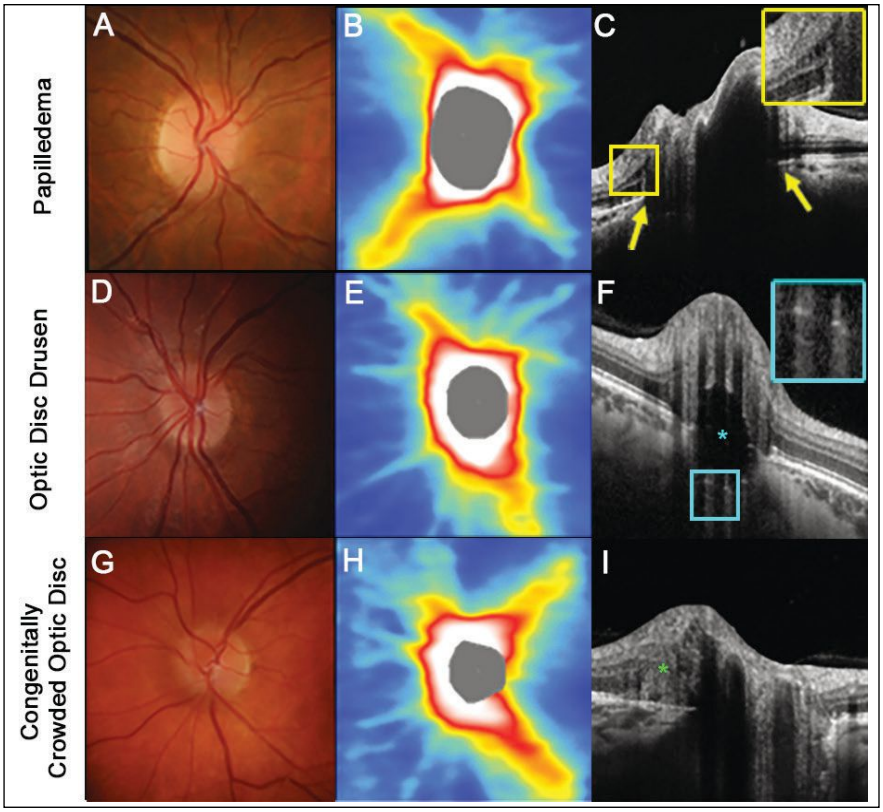

Fig. 4. An example of papilledema. (A) The right disc has minimal visible cup with some subtle blurring of the superonasal disc margin. (B) OCT thickness heat map shows thick superior and inferior RNFL. (C) A high-density OCT line scan shows anterior protrusion of Bruch's membrane at its opening (yellow arrows) and intraretinal cystic spaces temporal to the disc margin (inset). (D-F) An example of optic disc seen more posteriorly within the optic nerve space (inset). (G-I). An example of a congenitally crowded optic disc. While the fundus photograph and OCT thickness heat map are similar to the other two conditions, there are no other features specific to these conditions visible on a high-density OCT line scan. Note that while a peripapillary hyper-reflective ovoid mass-like structure (PHOMS) can be noted at the nasal neuroretinal rim (green asterisk), this feature is non-specific to various causes of elevated optic discs (I).

Assessment of OCT thickness heat and deviation maps from macular inner retinal layer analyses from both eyes are the most useful in these cases, as the location-specific information aids visualization of patterns of change. These analyses include the macular ganglion cell layer (GCL), ganglion cell-inner plexiform layer and ganglion cell complex, which are variably available across different OCT instrument software.

Due to the poor contrast between the inner plexiform and inner nuclear layers and potential for more variable segmentation, complexes including layers other than the GCL are advantageous and more commonly available across OCT software. While concordant RNFL damage will also be observed, interpretation of peripapillary OCT volume scans is more complex, especially in postchiasmal disease, due to differences in temporal vs. nasal retinal involvement between eyes and resultant different RNFL projections to the optic discs.

Patterns of structural damage reflect the paths of affected retinal ganglion cell axons and/or optic tracts and mirror concurrent visual field defects. Lesions anterior to the optic chiasm typically present with unilateral, global inner retinal reduction, while those affecting the optic chiasm tend to present with binasal inner retinal loss respecting the vertical midline. This is because of decussation of the nasal retinal ganglion cell axons.[28,32,33] Meanwhile, defects posterior to the optic chiasm generally respect the vertical midline and are homonymous, either affecting the left or right hemifield in both eyes and with the lesion located on the same side as the eye with temporal retinal loss. Such defects may be incongruous or not entirely symmetrical, particularly with more anterior involvement at the temporal or parietal lobes, with increasing symmetry observed as lesions involve more posterior locations towards the primary visual cortex.[32] A schematic depicting the patterns of inner retinal loss on OCT vs. lesion location is shown (*Figure 5*).

It is important to note that large, compressive lesions may not follow these general trends due to extensive involvement. For example, a large prechiasmal lesion may affect both optic nerves and subsequently present bilaterally, while a large chiasmal lesion may extend anteriorly such that temporal inner retinal involvement may be observed. Moreover, as retrograde degeneration is a gradual process, acute cortical injuries with associated visual field changes may present with normal OCT findings. However, changes may begin to occur weeks to months after the original insult and could demonstrate progression with time.

6. Can OCT diagnose neurodegenerative diseases?

In recent years, there has been rich, growing evidence to suggest that OCT can be used in documentation of pathological retinal changes associated with neurodegenerative diseases. Currently included in this list are multiple sclerosis, Parkinson's disease and Alzheimer's disease.[34-37] Since the retina is often viewed as an extension of the central nervous system and can be visualized non-invasively, it may help aid in providing evidence of diseases like these.

The RNFL has been proposed as a biomarker in neurodegenerative disease that is reflective of axonal

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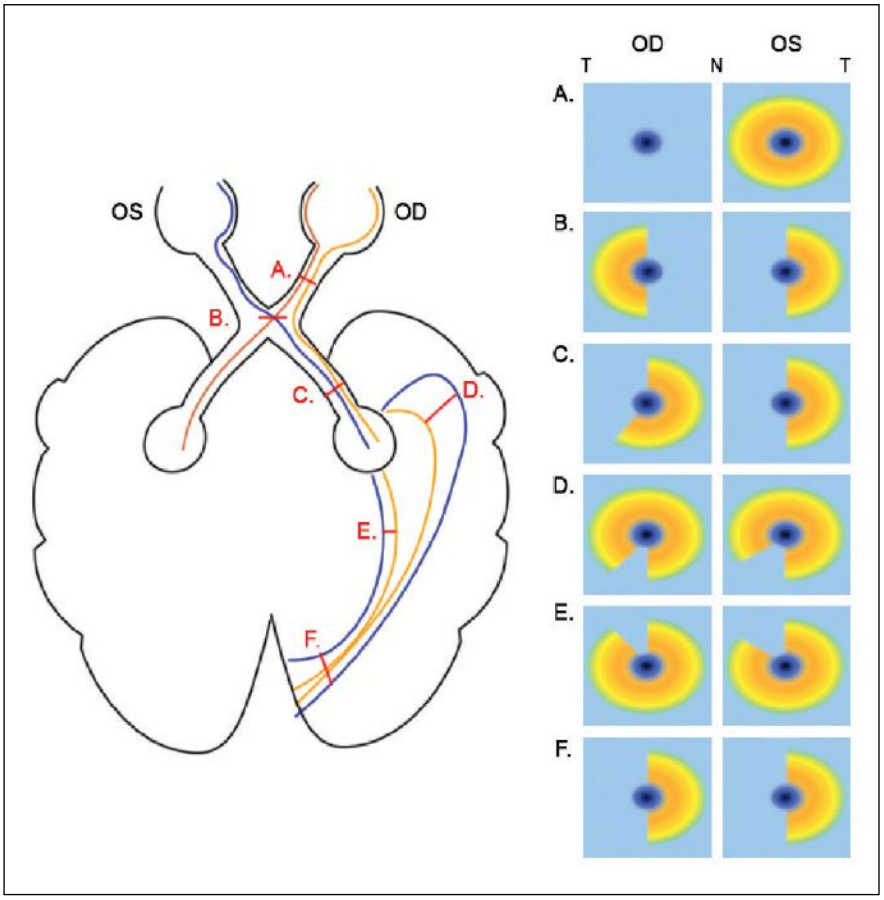

Fig. 5. A schematic of various lesions in the visual pathway and the corresponding changes in macular inner retinal OCT results. Only lesions affecting the right hemisphere are shown for brevity; for the left hemisphere, inner retinal results would be flipped horizontally. T and N denote temporal and nasal OCT locations, respectively. (A) For prechiasmal lesions affecting the right optic nerve only, global reduction in right inner retinal thickness on OCT are observed, and the left eye is unaffected. (B) For chiasmal lesions, due to decussation of nasal retinal ganglion cell axons, binasal loss in inner retinal OCTs are observed. (C) For postchiasmal lesions affecting the right hemisphere, homonymous left losses in inner retinal OCTs are noted; these may not be entirely congruent, given the relatively anterior location. (D) For lesions in the temporal lobe, homonymous superior-left defects are expected, with greater reduction in the eye ipsilateral to the lesion. (E) For lesions in the parietal lobe, homonymous inferior-left defects are expected, with greater reduction in the eye ipsilateral to the lesion. (F) For lesions of the occipital lobe that are at the primary visual cortex, homonymous left losses in inner retinal OCTs are noted.

and neuronal loss associated with progression of neurodegenerative diseases. While several studies have shown RNFL thickness is reduced in patients with Alzheimer's disease, multiple sclerosis and Parkinson's disease, compared to healthy control subjects, there is little agreement in the pattern of RNFL loss to differentiate it from other optic nerve diseases, such as glaucoma.[37-39] Interestingly, the relationship between the severity of disease and extent of RNFL thinning in neurodegenerative conditions remains equivocal.[35,36,40]

Other structural markers that have been examined, but with clinical implications that remain unclear, include the contour and shape of the foveal pit, asymmetry of foveal thickness and alterations in other retinal layer thicknesses.[37-39] Before these findings have clear clinically translatable outcomes, more work is necessary to better understand retinal changes associated with neurodegenerative conditions and to differentiate these from changes associated with other ocular pathologies.

Takeaways

OCT is a highly valuable clinical tool complementing the neuro-ophthalmic assessment in optometric practice. With careful choice of scan type, evaluation of both quantitative and qualitative data and holistic interpretation with other aspects of the clinical examination, OCT can provide key additional information for conditions. These range from tilted disc syndrome to cerebral cortical lesions, aiding the diagnostic process and guiding resultant management strategies. ■

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AN OVERVIEW OF ANTERIOR SEGMENT OCT

We present the virtues and shortcomings of its clinical applications in angle assessment, corneal disease and contact lens fitting.

BY SHARON KEH, OD, IRENE FRANTZIS, OD, AND YANA SEVIARYN
NEW YORK CITY

While not as popular or widely used as its posterior segment counterpart, AS-OCT has earned its rightful place as a valuable, adjunctive tool to diagnose and monitor corneal and anterior segment abnormalities. It uses low-coherence interferometry to enable non-contact, *in vivo* imaging of ocular structures. This technology is available to practitioners as a dedicated stand-alone device or as part of technology that also images the posterior segment.[1,2] This feature will highlight AS-OCT's most common applications in optometric practice.

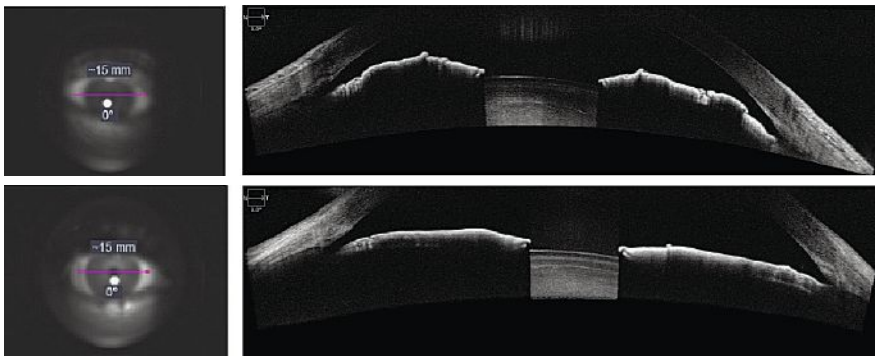

Fig. 1. AS-OCT evaluation of narrow angle (top) and open angle (bottom) using anterior segment angle-to-angle scans.

Indications

AS-OCT technology has evolved since its commercial availability in the early 2000s. Today, these instruments have higher wavelengths to improve penetration depth (especially through sclera and iris), higher resolution, faster imaging acquisition time and a wider field of view. They can image structures as anterior as the tear film and meibomian glands and as posterior as the lens, with certain units even providing 360-degree views of the anterior segment.

Clinically, it has major roles in corneal and anterior segment disease, contact lens fitting and anterior chamber analysis. AS-OCT images are reimbursable by using the Current Procedural Terminology for AS-OCT (92132, scanning computerized ophthalmic diagnostic imaging, anterior segment with interpretation and report, unilateral or bilateral). Payment is bilateral, so no laterality modifier is required for billing. Practitioners should familiarize themselves with the ICD-10-CM diagnosis codes that can be used in conjunction with 92132 (anterior segment).

Corneal and Anterior Segment Disease

When assessing an AS-OCT corneal image, the first, thin, hyperreflective layer is the tear film. Using the caliper tool, the tear menisci can be

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measured. Directly underneath the tear film is the hyporeflective corneal epithelium. AS-OCT accurately maps clear and opacified corneas, allowing practitioners to precisely delineate the individual corneal layers: epithelium, Bowman's, stroma, Descemet's membrane and endothelium. This can be instrumental when confirming the layer(s) involved for various corneal opacities and dystrophies, such as epithelial basement membrane dystrophy and lattice dystrophy (*Figure 3*).

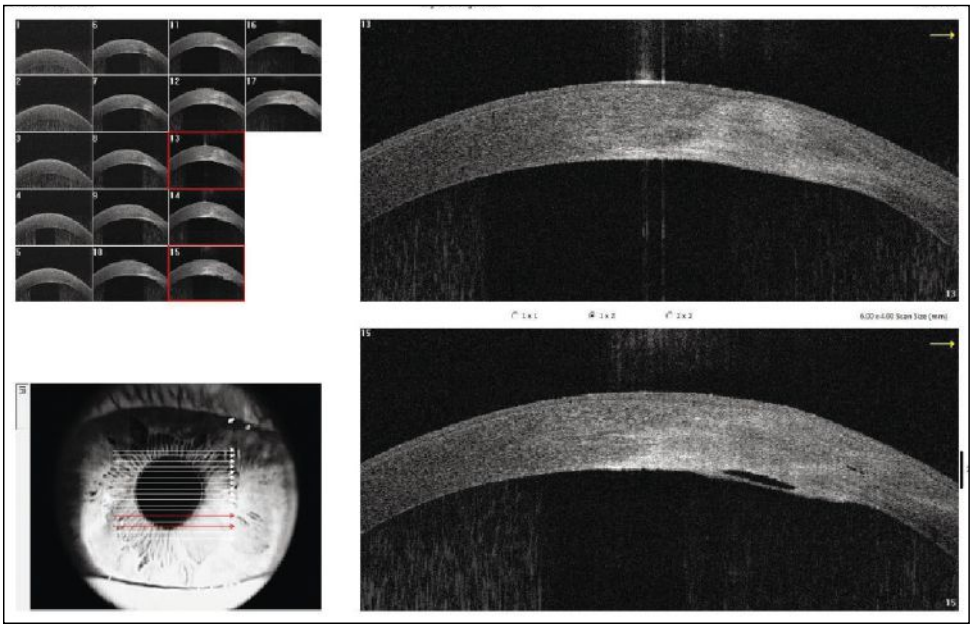

Fig. 2. Imaging of a resolving corneal hydrops in a patient with keratoconus, highlighting corneal edema and the break in Descemet's membrane.

Because the cornea is transparent and avascular, borders and landmarks between corneal layers using slit lamp are best appreciated qualitatively with optic section, but AS-OCT has the distinct advantage of quantification (*i.e.*, can measure to the nearest micron). A clinical example where AS-OCT is useful is monitoring epithelial healing from a superficial defect underneath a soft, bandage lens. The defect can be measured and assessed without needing to remove the lens earlier than warranted.[3]

Its technology can precisely map the depth of corneal and conjunctival abnormalities alike. For example, ocular surface squamous neoplasia, which was historically diagnosed by biopsy alone, has unique diagnostic characteristics visible with AS-OCT (*i.e.*, a thickened, strongly hyperreflective epithelium with abrupt transitions from normal to abnormal epithelium). In contrast, pterygia and pinguecula demonstrate a thin or normal epithelium overlying a fibrous subepithelial lesion.[4,5]

AS-OCT technology can provide practitioners with important data to help differentiate benign from malignant lesions, including lesion size, internal structures (*e.g.*, cysts), vascularity, layers involved and shadowing and its anterior and poster surfaces. While pathology specimens may still definitively differentiate ocular surface lesions, AS-OCT can still be used to guide appropriate management and confirm adequate treatment depth if excision is necessary.

AS-OCT similarly has applications in the management and diagnosis of infectious, inflammatory and ectatic corneal disease. The thickness of subepithelial infiltrates or corneal scars can be measured with the caliper tool on the AS-OCT and monitored over time as they improve and resolve with treatment.[6] In infectious cases, it can distinguish confounding variables such as significant epithelial defects, focal edema and anterior chamber reactions. Microscopic cells in the anterior chamber appear on AS-OCT as discrete, hyperreflective suspended foci. Obtained images can be used to objectively quantify measurements of inflammatory cells and aqueous flare, even when masked behind cloudy corneas.

In ectatic disease, pathologies such as keratoconus were traditionally monitored with corneal topography, pachymetry and eventually Scheimpflug tomography. With AS-OCT, specialists can visualize and track distinguishing features of keratoconus within individual corneal layers. Bowman's membrane and epithelial thickness maps over the apex of the cone have been highlighted in recent studies as important tools to distinguish non-diseased eyes from manifest, subclinical and forme fruste keratoconus.[7] This has important implications for refractive surgery screening, especially in uncertain cases.

For advanced keratoconus, patients with increased epithelial thickness, anterior hyperreflection at Bowman's and stromal thinning at the cone are considered to be at-risk for developing corneal hydrops. If corneal hydrops does occur, AS-OCT scans can pinpoint the location of the break in Descemet's and display the extent of edema present (*Figure 2*).[8] While hydrops is a rare complication, when it occurs the sudden visual loss is distressing and the corneal edema is often diffused, making it difficult to visualize posterior structures. AS-OCT is an invaluable patient education and monitoring tool to follow the resolution of edema over time.

AS-OCT has significant functionality when used preoperatively or intraoperatively for corneal procedures targeted at specific corneal layers, including epithelial debridement,

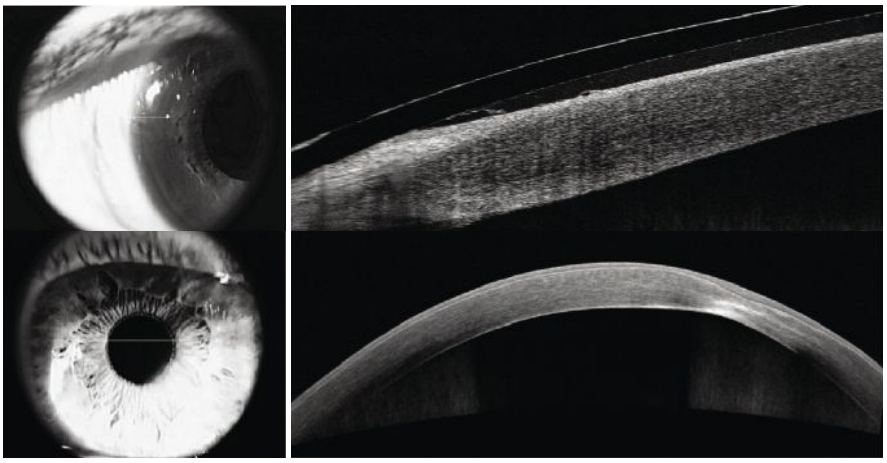

Fig. 3. Limbal microcysts under a scleral contact lens exhibiting limbal vault (left). Corneal scarring and opacification identifying the corneal layers involved (right).

superficial keratectomy and phototherapeutic keratectomy. It can help dictate if a partial corneal transplant, such as deep anterior lamellar keratoplasty, can be performed in lieu of a full-thickness penetrating keratoplasty by confirming that the lesion is located in the stroma alone. It can even precisely locate the LASIK flap interface of previously performed procedures or monitor graft adherence following DSAEK transplantation. By understanding the exact location of the corneal pathology with the help of AS-OCT, clinicians can easily choose and monitor complications of the surgical intervention to maximize outcomes.

Contact Lens Fitting

One of the most exciting applications for AS-OCT is its role in contact lens fitting for all lens types. The chord and sagittal height measurements provide vital information about peripheral corneoscleral profilometry and sagittal depth.[9,10] This allows fitters to measure the sagittal height at specific diameters, allowing for greater precision when empirically designing specialty soft lenses. AS-OCT can assess made-to-order lens fit on eye, guiding modifications to base curve, diameter, prism, optic zone sizes and decentered optics.

A recent study revealed that novel contact lens fitting software built into the AS-OCT may be used in the future to empirically design rigid gas permeable lenses for keratoconus.[11] AS-OCT is likewise used routinely to assess hybrid contact lenses without the need for sodium fluorescein by identifying the appropriate amount of vault under the gas permeable component of the lens. It can additionally provide insight into the fit at the junction of the lens and alignment of the soft skirt.

Recently, AS-OCT has been briskly gaining popularity in the diagnostic and empirical fitting of scleral lenses. Even for experienced fitters, the process of fitting scleral lenses can be time-consuming and laborious. Clinicians often need significant pre-fit data (including imaging) and the lens fitting may involve the insertion and removal of numerous scleral lenses, all of which require settling.

AS-OCT has modernized fitters' abilities to quickly measure clearance over the corneal apex, mid-peripheral corneal clearance, limbal vault and scleral edge alignment accurately and precisely. In fact, technicians and medical assistants can be trained to acquire these images over different trial lenses to further maximize exam efficiency. This is especially crucial for follow-up visits, where patients present wearing scleral lenses with a clear tear reservoir.

While scleral lenses can be evaluated with biomicroscopic viewing alone, a recent study found that novice, intermediate and advanced fitters all tend to overestimate the central corneal clearance using this method.[12] With the aid of AS-OCT, fitters can confirm and expedite their clinical assessments to quantify the exact vault in microns. AS-OCT also demonstrates the fitting relationship at the limbus, edges and within the tear reservoir (*Figures 3-5*). When speaking with lab consultants about these in-office fits, images can be shared directly aiding in the proficiency of modification recommendations. Many optometrists use their devices to assess specialty contact lenses, allowing for more timely, precise and clinically meaningful modifications to complicated fittings.

Anterior Chamber Angle Analysis

AS-OCT can be extremely useful in assessing the anterior chamber. It is an important complement to gonioscopy and can confirm that minimally invasive glaucoma surgeries have targeted the correct anatomy. Practitioners agree that it does not replace gonioscopy, but rather complements it, as AS-OCT is more objective and can be performed under different lighting conditions, which may supplement a practitioner's understanding of angle closure. It is non-contact, unlike gonioscopy, and requires corneal interaction (which not all patients tolerate well). Gonioscopy also necessitates subjective judgment and substantial training by the observer.

One study found that AS-OCT scans were more reliable in predicting the success of laser peripheral iridotomy than clinical examination by glaucoma specialists.[13] While the ZAP trial was focused on determining longitudinal changes in angle configuration in the eyes of primary angle-closure suspects treated by laser peripheral iridotomy and in untreated fellow eyes, there was significant interest in better understanding the anterior chamber and angle

anatomy, which the newest AS-OCT technologies may help to do.[14]

Another major advantage is that AS-OCT allows for quantification of angle parameters, *e.g.*, anterior chamber depth, angle-to-angle distance, angle-to-angle width, pupil diameter, crystalline lens rise and iris thickness. In fact, when quantitative iris parameters were investigated in angle closure disease, increased iris thickness as measured by AS-OCT was associated with a higher risk of angle closure.[15] However, while these parameters and quantifications exist, there are not yet strict guidelines of how to manage patients based on these values.

Moreover, AS-OCT has a growing role in imaging iris and uveal lesions in the anterior chamber. While iris nevi are relatively common and require no treatment, malignant melanomas have the potential to metastasize and often require treatment. Ultrasonography plays an important role when differentiating between these diagnoses, but AS-OCT can also reveal the location, size and extent of a mass's boundaries.

Limitations

While AS-OCT is an important tool for many clinicians, it's important to note some limitations that exist. The operator must be familiar with the specific device they are using, as scan protocols and instrumentation differ amongst different manufacturers. For example, the Cirrus 6000 (Zeiss) device has a separate cornea and an angle attachment and its own proprietary software to expand the capabilities of its posterior segment device. The Anterion (Heidelberg) is the newest, dedicated AS-OCT device and unique in that it combines tomography and anterior segment imaging in one instrument.

To capture a successful image, the operator must be familiar with the purpose of the image and its proper acquisition techniques. For instance, when acquiring images of the anterior chamber angle during a glaucoma evaluation, the operator should take into consideration the lighting conditions, which affect pupil size. As pupil size changes, the angle either narrows or widens, making it difficult to compare images over multiple visits.[16,17]

> **With AS-OCT, specialists can visualize and track distinguishing features of keratoconus within individual corneal layers.**

Unlike posterior segment OCT, which has built-in progression analysis, AS-OCT has a lack of tracking between visits. Once the lighting conditions are determined, the patient must be appropriately positioned. Images that are not acquired perpendicular to the ocular surface run the risk of leading to a diffraction phenomenon, giving rise to distorted images.[16] Common types of imaging artifacts include an inability to identify Schwalbe's line or anterior iris surface, poorly aligned scan or poor penetrance when there is opacification. Even after controlling for the factors mentioned above, the quality of the image can be difficult to assess, as certain AS-OCT devices do not come equipped with an objective scoring system like signal strength quality found in posterior segment OCT.[19] The operator, therefore, is left to subjectively determine the quality of the images.

After the image is found to be of good quality, different examiners may have their own interpretation of the position of the landmarks that they are looking for. This is another source of error that can lead to issues when comparing images over multiple visits or amongst different practitioners.[19,20] Previous work has shown only a moderate level of agreement in grading AS-OCT photos.[21] It is also important to mention that in the case of the anterior chamber angle evaluation, the light rays of the AS-OCT cannot penetrate the iris pigment epithelium; thus, the ciliary sulcus and posterior ciliary body cannot be imaged.[22] This can greatly impact interpretation of the scans.

Finally, no matter what the purpose of the image is, there are measurement differences between different AS-OCT imaging software. For instance, a practitioner who wants to compare central corneal thickness between other AS-OCT devices will not be able to do so, as it will differ.

AS-OCT vs. Corneal Tomography

When it comes to corneal shape, thickness and power, corneal tomography provides a great deal of information. Like AS-OCT, it captures cross-sectional images and provides deeper three-dimensional understanding of the cornea and its underlying angle. The Pentacam (Oculus) and AS-OCT have displayed good agreement in corneal thickness and anterior chamber depth measurements. Most AS-OCT devices do not have topography or tomography capabilities, with the Anterion being a notable exception.

AS-OCT vs. Gonioscopy

This eye test is frequently used to evaluate and monitor the angle

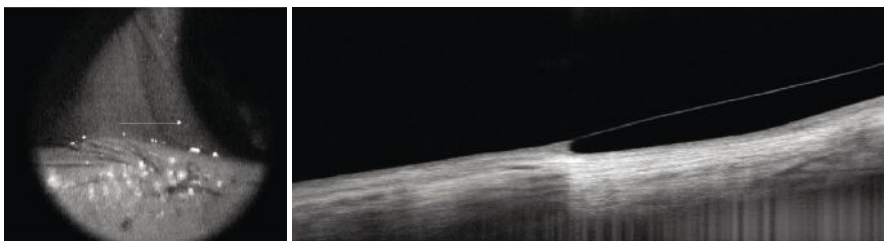

Fig. 4. Good scleral alignment of a scleral lens without impingement or lift-off.

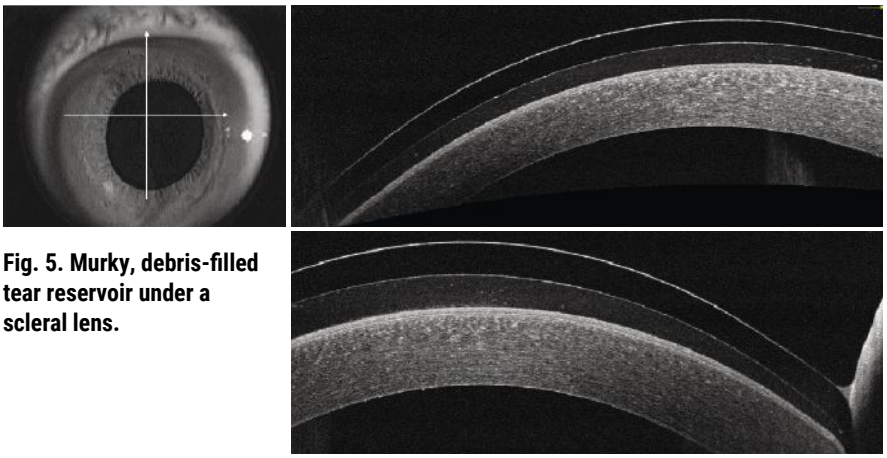

Fig. 5. Murky, debris-filled tear reservoir under a scleral lens.

of glaucoma patients and narrow angle suspects. One disadvantage of gonioscopy compared to AS-OCT is that it requires a skilled practitioner to perform and interpret the clinical appearance of the angle. The gonioscopy lens also contacts the patient's cornea and requires patient cooperation. However, AS-OCT does not provide a dynamic view of the angle as gonioscopy cannot yet image neovascularization, pigment and/or peripheral anterior synechiae. Similarly, the superior and inferior quadrants are difficult to scan with AS-OCT without manipulating the angle configuration as only gonioscopy scan. Both procedures are useful to understand angle anatomy iris approach with AS-OCT preferred for patients unable to tolerate gonioscopy.

AS-OCT vs.Ultrasound Biomicroscopy (UBM)

This ultrasound technique is a useful tool to image much of the anterior segment anatomy, specifically by providing visualization of ciliary body and structures posterior to the iris, which AS-OCT does not perform as well.

Compared with AS-OCT, this technology has several limitations. The UBM probe contacts the ocular surface and requires patient cooperation and a skilled examiner. UBM also produces lower resolution images than AS-OCT. However, UBM is preferred when imaging certain conditions such as iris cysts and melanomas because it is better able to penetrate the iris pigment epithelium.

Takeaways

There is no doubt that AS-OCT is a powerful imaging tool that allows us to measure, visualize and image cross-sectionally the anterior segment from tear film to lens. It is an amazing patient education tool when describing or illustrating the depth of a corneal scar or teaching patients about their iridocorneal anatomy. It is well-tolerated by patients, as it is non-contact and image acquisition times are short. Practitioners agree that training operators is straightforward, and that the interpretation of images is easier as the basic structures are the same as those visualized with slit lamp.

Given AS-OCT's unique advantages and the possibility of future visibility of anterior segment vasculature, growing in popularity much like its posterior segment counterpart is highly anticipated. ■

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MECHANISMS OF MYOPIA: WHAT WE KNOW & WHAT WE WONDER

With an increase in prevalence and severity, there is a growing interest in the pathophysiology of this condition.

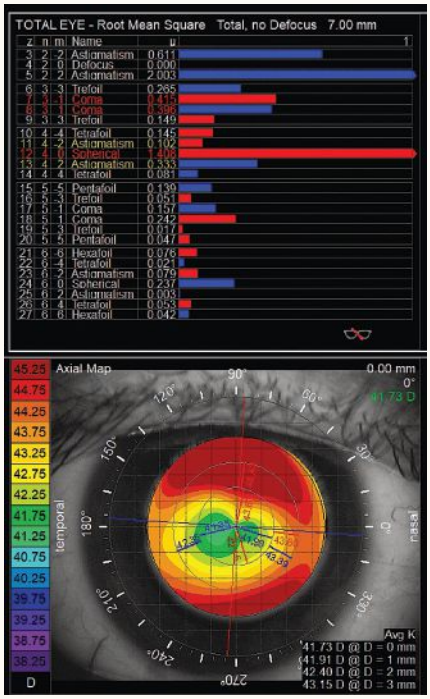
BY ERIN S. TOMIYAMA, OD, PhD
FULLERTON, CA

Myopia is a result of a mismatch between the refractive power of the eye and its axial length, most commonly from the eye growing too long.[1] In the United States, the prevalence of myopia increased from 25% in 1971 to 1972 to 42% in 1999 to 2004, and worldwide, the condition's prevalence is expected to reach 50% by 2050.[2,3] With an increase in prevalence and severity, the interest in the pathophysiology and mechanisms of myopia follows. Treatments such as atropine, orthokeratology and peripheral defocus contact lenses are used to slow the progression of myopia after it develops, but a better understanding of the causes of myopia could delay or prevent onset altogether.[4-9]

While there has been an exponential increase in myopia literature over the last few decades, the main focus has been on slowing myopia progression. And so, there are still several unknowns regarding the pathophysiology of this condition and the roles both genetics and the environment play in myopia development and progression. In this article, we will delve deeper into the mechanisms of myopia and explore several key areas that require further investigation as to how they might impact optometric practices.

Role of HOAs in Myopia Development

Higher-order aberrations (HOAs) can degrade retinal image quality and may interact with lower-order aberrations to change the optics of the eye. These changes may play a role in refractive development and the emmetropization process.[10] Some studies suggest an increase in HOAs, specifically spherical aberration and coma, is associated with myopia, while others suggest there is no change in HOAs with myopia.[11-15]



Higher-order aberrations measured after orthokeratology lens wear. Primary coma and spherical aberration are elevated to outside normal limits. Corneal topography shows central flattening with an incomplete treatment ring.

About the author

Dr. Tomiyama is an assistant professor of optometry at Marshall B. Ketchum University, where she offers a contact lens curriculum and serves as a clinical attending in the Stein Family Cornea & Contact Lens Center. She recently established the Myopia Management Service at the university. She receives consulting fees from Vyluma and conducts research with CooperVision.

Mixed results have also been observed in longitudinal studies evaluating the relationship between HOAs and axial elongation. However, age, degree of myopia and method of measurement were confounding variables.[16-18] More recently, one study found that increased levels of HOAs were associated with slower axial elongation.[19] This is perhaps due to the HOAs altering the retinal image quality and serving as a directional cue to slow eye growth. Further longitudinal studies are needed to fully understand the role of HOAs in the emmetropization process.

There is strong evidence to support the notion that current optical treatments for myopia management also induce more HOAs.[20] This increase in HOAs could play a part in how these optical devices, specifically orthokeratology and multifocal soft lenses, slow myopia progression.

Pathophysiology Differences Between Myopia Types

Most forms of myopia occur because the axial length is too long relative to the refractive power of the eye, otherwise known as axial myopia.[21] Conversely, there are conditions, such as keratoconus, where myopia results from corneal changes rather than axial elongation.[22] There are several hundred genetic conditions that feature myopia and involve various molecular pathways, but these rare conditions only account for less than 1% of the myopic population.[23] The most common form of myopia—school-age myopia—is impacted by both the environment and gene expression.[21] However, adult-onset myopia may have a different pathophysiology given that it develops later in life. Adult-onset myopia is commonly associated with near work and usually limited to low or moderate levels.[24] Therefore, the degree to which genetics play a role in myopia development may vary by type, but school-age myopia has been found to have both genetic and environmental components.

Disease Development and Genetics

There is an ongoing debate as to whether nature or nurture prevails in the development of myopia. Initial thoughts of a genetic basis for myopia come from a greater relationship of refractive error in monozygotic compared with dizygotic twins.[25] Overall, several twin studies continued to demonstrate high heritability, but this could be confounded with shared environmental factors such as location and education. However, one twin study investigated refractive error development in twin pairs reared separately and found greater similarity among monozygotic twins compared with dizygotic, indicating a genetic influence.[26]

Parental myopia is a risk factor for child myopia. One myopic parent increases the risk by threefold and two myopic parents by sixfold.[27] While genetics could be the primary driver of this increased risk, there are also other factors at play, such as lifestyle and upbringing. Further studies showed that there was a greater correlation between refractive errors of siblings than of children and their parents, implying that environment has a predominant role. Even correlations between cousins were almost as high as between siblings, highlighting contemporary environmental factors.[28]

Genome-wide association studies compare genetic information across large groups of people to identify single nucleotide polymorphisms that are associated with a particular trait. The combined efforts of 23andMe and the Consortium for Refractive Error and Myopia (CREAM) have identified 161 potential gene loci involved in myopia.[29] These studies have advanced our understanding of genetic mapping and potential loci associated with myopia, but ultimately these genetic variations only account for about 8% of the variation in refractive error.[30]

Other evidence to support a strong genetic component is the variation

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

- Recognize the pathophysiology and mechanisms of myopia.
- Explain the role genetics plays in the development and progression of this condition.
- Identify the environmental factors that contribute to myopia.
- Educate patients and their parents on what they can do.

Target Audience: This activity is intended for optometrists engaged in myopia management.

Accreditation Statement: In support of improving patient care, this activity has been planned and implemented by PIM and the Review Education Group. PIM 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 CE for the healthcare team. PIM is accredited by COPE to provide CE to optometrists.

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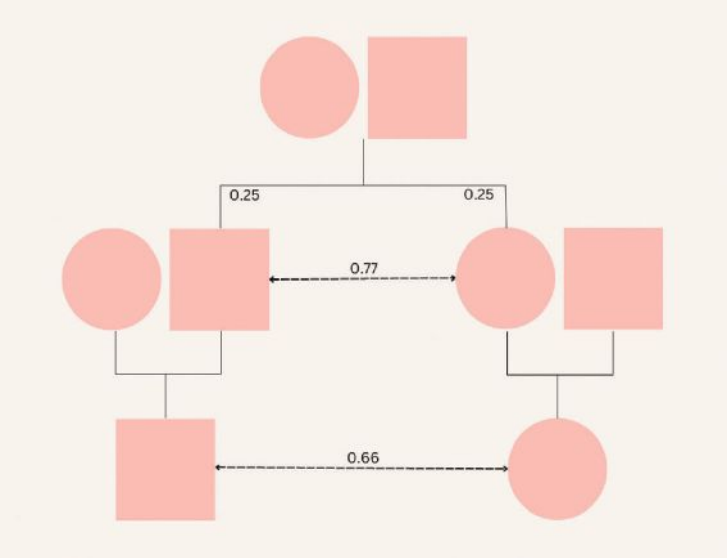
in the prevalence of myopia between different racial and ethnic groups. However, there can be large genetic variation between, and even within, populations.[31] Differences between populations are usually a result of rare alleles that would more readily disappear due to genetic drift, so it is unlikely this contributes to the high prevalence of myopia we see today.[32] Additionally, this genetic variation could also be confounded by location and environment.



A family tree shows the lowest heritability of myopia exists between parent and daughter/son. There is a greater heritability between siblings and cousins, which points to an environmental rather than a genetic cause.

While it is clear that there is a genetic component to myopia development, genetics cannot single-handedly be responsible for the dramatic rise in the condition.[21] The lack of causal evidence from specific genes and the fact that it is a locally regulated process both support this argument. Myopia is a multifactorial disease that also has an environmental component.

Impact of Environmental Factors

Emmetropization is a local, visually regulated process, thereby suggesting that myopia development is also modulated by visual signals perceived by the retina.[33] Several potential environmental elements could contribute, but study limitations and confounding factors prevent us from fully understanding these factors.[1] The two prevailing environmental factors are near work and outdoor time, which are discussed in more detail later.

Additional environmental and behavioral factors that have been explored include intelligence, physical activity, socioeconomic status and lived environment (rural vs. urban).[34] It could be hypothesized that intelligence is a surrogate for education or near work and physical activity for outdoor time. It has long been reported that members of higher socioeconomic status are more likely to develop myopia, but this could be associated with family income, parental myopia, parental education or near work demands.[35] It is unlikely that socioeconomic status itself is the direct cause of myopia, but perhaps it serves as a covariate for education and near work.[34]

Related to all these environmental factors is difference in lived environment. A greater prevalence of myopia has been found in more urban environments, but this could be related to education and outdoor time.[36] Those of higher socioeconomic status may live in more urban environments with greater population densities.[37] Urban environments also tend to have less access to green space, which could be a proxy for outdoor time.[38] In considering differences in the amount of outdoor time in urban vs. rural settings, other factors could include safety, weather, pollution and cultural attitudes.[34]

Education and Associated Near Work

The theory that myopia is related to education is based on the finding that the prevalence of myopia is higher in areas where there is an emphasis on education and more children complete more years of schooling.[23] Similarly, children who achieve higher grades and adults who complete higher educational levels tend to be more myopic.[27,39] The relationship between myopia and educational level is wrapped up in the confounding factors of ethnicity and increased near work.

It has been thought that there are academic pressures and more intense schooling undertaken by East Asian populations that could contribute to the higher prevalence of myopia among this group.[40] One study explored near work differences among East Asian and Caucasian groups and found that East Asian participants reported longer periods of near work, specifically with homework and computer use, that could be related to academic demands (differences in schools and participation in after-school programs, such as tutoring).[41] A meta-analysis found that there was a larger genetic influence on Asian participants with higher vs. lower levels of education but no significant interaction with European participants.[42] Additionally, a Mendelian randomization study showed that there is a causal relationship between educational attainment and refractive error.[43] It is clear that ethnicity is a risk factor for myopia development but also possible that differences in near work behavior and genetics among different ethnic groups augment this risk.

While there is a relationship between myopia and near work, it is potentially the intensity rather than the total duration of near work that is the primary driver.[41] Both closer working distances and longer time spent doing continuous reading were associated with a greater risk of myopia.[41]

Accommodation was also thought to play a role in near work, as near targets require a higher accommodative demand, which leads to increased accommodative lag that causes hyperopic defocus on the retina, stimulating axial elongation. Studies evaluating accommodative lag have shown that the increase in lag or under-accommodation occurs after the onset of myopia and is likely not causal.[44] Support for this accommodation theory grew when atropine, a topical agent that blocks accommodation, was shown to prevent myopia development. However, animal studies with chicks showed that atropine prevents myopia even though their accommodative system is mediated by nicotinic receptors; therefore, accommodation was shown to have no role in myopia onset or progression.[45,46]

Relationship Between Digital Screen Time and Myopia

In today's world, the type of near work has changed to increased use of computers, digital devices, video games and virtual reality. While parents are quick to blame excessive screen time for their child's myopia, the prevalence of the condition was increasing before the widespread use of digital devices.[47] However, with advances in technology and increasing use in schools, children are becoming digital users at a younger age. The World Health Organization points out that screen time may promote sedentary behavior that negatively impacts overall health, and some countries have imposed limitations of screen time in children.[48]

A systematic review of 15 studies included 11 on myopia prevalence and four on myopia progression.[47] Six of the 11 studies revealed a relationship between computer use and the prevalence of myopia, while the other five did not. The pooled odds ratio suggests there is no relationship between screen time and myopia, though there are some confounding variables such as education intensity, time spent on schoolwork/homework, recall bias and near work. It is also likely that screen time is a substitution for paper and pen reading/writing, not outdoor time.[47]

Outdoor environments provide higher light intensity, varying spectral composition of light and flatter dioptric demands, all of which may contribute to delayed myopia onset.

The COVID-19 lockdown provided a unique opportunity to study the effects of home confinement on myopia. During this period, there was less outdoor time, more indoor time, more screen time and more near work.[49] One study evaluated over 120,00 children in China who underwent noncycloplegic photorefraction.[50] The team found a myopic shift of -0.30D and an increase in myopia prevalence among six- to eight-year-olds compared with the years leading up to the epidemic (2015 to 2019); however, it is important to note that the measurements were noncycloplegic and taken with a photoscreener device.[50]

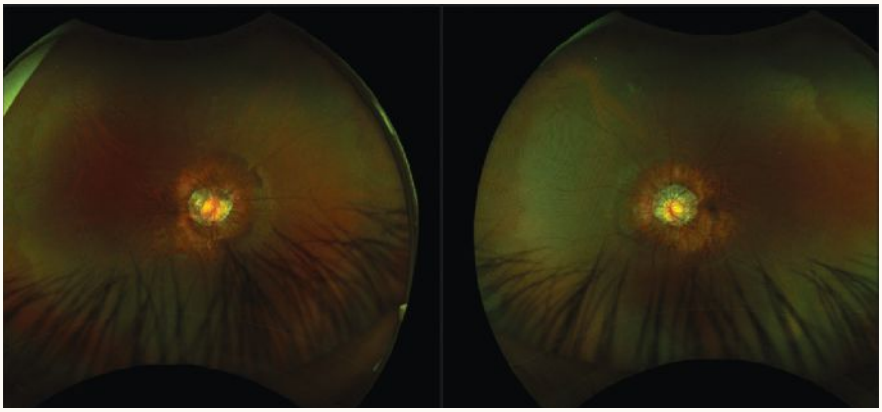
Understanding the Role of Outdoor Time

While this is one of the main modifiable risk factors for myopia development, the specific components of outdoor time remain unknown.

Protecting against myopia. Parents often ask how much time outdoors is needed. The Sydney Myopia Study showed that exposure of at least two hours per day was associated with a decreased risk of myopia development.[51] In 2012, a meta-analysis revealed that each additional hour spent outdoors per week reduced the odds of myopia by 2%.[52]

It is clear there is a dose-dependent response between increased time outdoors and the risk of myopia onset. One study found that an increase of 76 minutes per day is needed to reduce the incidence of myopia by 50%.[53] The main limitation to all of these studies is recall bias from the use of questionnaires where patients/parents report the amount of time spent outdoors. More recently, the development of wearable technology has allowed for more objective measures of outdoor time, such as light exposure and activity.[54,55] However, wearable devices are still subject to proper use (*i.e.*, remembering to wear and charge the device), and the location of the device on the body may not be truly representative of the visual input.

In addition to the total time spent outdoors, taking an outdoor break directly after prolonged near work may be more effective. Researchers showed that, in chicks, a brief period of bright light exposure, simulating



Fundus photos of the right and left eyes show posterior staphylomas, common among high myopes, with peripapillary atrophy around the optic nerve heads. White without pressure can be seen in the periphery of both eyes.

outdoor time, after minus defocus can help negate the signals for developing myopia.[56] Animal research has also shown that there may be a role in the timing and intervals of bright light exposure, rather than the cumulative daily exposure, that could disrupt normal circadian rhythm.[57]

Ultimately, a recommendation of two hours per day of outdoor time should be made to pre-myopic children. Specifics as to the timing or intervals of time outdoors will be elucidated with further research.

Delaying disease onset. There are several proposed reasons why increased outdoor time is linked with a lower incidence of myopia: higher light intensity, spectral composition of light and dioptric demand. Outdoor ambient illumination varies from 1,000 to 150,000 lux.[55] Rearing animals in a high-light environment has a protective effect against form-deprivation myopia but less so against lens-induced myopia.[58,59]

The primary theory behind high-light intensity is that brighter light triggers increased dopamine release from the retina, which slows axial elongation.[60-62] Dopamine also modulates retinal circadian rhythm, and other diurnal rhythms have been found in components of eye growth (*i.e.*, axial length and choroidal thickness).[57] It is possible that both light-regulated dopamine release and/or disruptions of circadian pathways could be involved in axial elongation.[34] Melatonin is another modulator of circadian rhythm with a reciprocal relationship to dopamine. Myopes have been shown to have higher levels of melatonin in the morning and poorer sleep quality.[63] Light exposure can promote dopamine release and suppress melatonin, both involved in modulating circadian rhythm, which may be required for the emmetropization process.

Another possible theory of high-light intensity is that increased ultraviolet light stimulates the production of vitamin D, which protects against myopia development. Though it has been shown that myopes have lower levels of vitamin D, there is no evidence of a causal relationship.[64,65]

The spectral composition of light varies between indoor and outdoor environments, time of day and season.[66] Longitudinal chromatic aberrations cause different wavelengths of light to come into focus at different positions. Short blue wavelengths come into focus in front of long red wavelengths. Rearing animals in monochromatic conditions results in a disruption of the emmetropization process. Conflicting results are presented, where long-wavelength red light causes myopia in chicks and guinea pigs but hyperopia in tree shrews and monkeys.[67-70] The exact opposite was found with chicks, guinea pigs and tree shrews in relation to short-wavelength blue light.

In the last year, several studies have looked at the use of low-intensity red light therapy as a treatment for myopia management in children in East Asia. Most used a protocol of three minutes, twice per day, of 635nm to 650nm red light administered via desktop light with at least four hours between each session.[71-75] The studies are all in agreement that children who are treated with low-intensity red light have less spherical equivalent refractive error changes and less axial elongation than children not receiving treatment. There are conflicting results regarding choroidal thickness changes, but most found an increase in choroidal thickness after red light therapy.[71,76,77] The one study that did not compared two eyes within a participant, with only one eye receiving red light treatment.[78] Though most data are presented for 12 months or less, two studies have already evaluated efficacy after treatment cessation and shown there is a moderate rebound effect.[76,79] Though promising, the use of low-intensity red light therapy is not approved in the United States, as there are insufficient data on its long-term safety. More longitudinal data are needed to fully understand the risks and benefits of low-intensity red light therapy.

While the focus has been on long-wavelength red light, one study evaluated the use of short-wavelength blue light and found that it decreased axial length with no significant change in choroidal thickness.[80] Participants were exposed to blue light for one hour, and hyperopic defocus was induced by a minus lens over one eye. The change in axial length was small (less than 10µm), so more studies are needed to show the efficacy of short-wavelength blue light.

Lastly, the dioptric demand of an outdoor environment is often much flatter than an indoor setting. Near stimuli in an indoor environment cause hyperopic defocus on the retina.[81] A recent study evaluated over 800 images of different indoor and outdoor settings and found that man-made outdoor

and indoor environments lacked high spatial frequencies and created spatial frequency profiles that were similar to Bangerter filters used to induce form deprivation myopia in animals.[82]

Preventing disease progression. A summary of four randomized controlled trials done in East Asia shows that increased outdoor time reduces myopia incidence by 5% to 10% but has little effect on slowing progression.[1,83-86] The reported reduction in myopia progression in myopic participants was 0.17D to 0.23D with a 0.03mm to 0.15mm reduction in axial elongation over the one- to three-year study period. A meta-analysis of 25 studies confirmed that there is a protective effect of increased outdoor time in delaying myopia onset of non-myopic children, but no effect on myopia progression of children who are already myopic was noted.[53] However, there is evidence that the rate of myopia progression can be regulated by environmental factors. Specifically, there are seasonal differences in myopia progression showing slower rates in summer than in winter.[87,88] A few studies have reported that more outdoor time does indeed slow myopia progression.[89,90]

Myopic children spend less time outdoors compared with non-myopic children.[91,92] While the evidence may be inconclusive, increased outdoor time is still an important public health recommendation for children that aligns with initiatives to promote a healthier lifestyle.[93] Additionally, delaying the onset of myopia will slow myopia progression since it is largely age-dependent and will also decrease the level of final myopia.[93]

Navigating the Unknown

Despite several advances in the understanding of myopia development and progression, there are still many unknowns. It is imperative that practitioners continue to stay on top of education in this area so they can practice evidence-based optometry. As this public health issue continues to grow, optometrists should aim to first delay or prevent the onset of myopia. By delaying onset, we can reduce the severity of an individual's myopia and decrease the prevalence of high myopia and the risk of ocular consequences that follow. Practitioners can educate parents and patients about myopia development and recommend behavioral modifications such as increased outdoor time and decreased near work, with breaks and increased working distances. Once children develop myopia, treatments alone or in combination should be implemented to slow the progression.

To answer the age-old question of nature vs. nurture, there are aspects of both genetics and environment that play a role in myopia development. The increase in myopia prevalence has dramatically exceeded the increase that would be expected from genetic variability. So, while there may be genetic susceptibility, it cannot be the only cause of myopia. There is resounding evidence that environmental factors influence the development of myopia. With environmental modifications, there is hope that we can win the battle against myopia. ■

With myopia on the rise, practitioners must implement myopia management therapies and recommend behavioral modifications to lower the risk of high myopia in the future.

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OPTOMETRIC STUDY CENTER QUIZ

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1. Which treatment is used to slow myopia progression?
a. Undercorrection.
b. Single-vision spectacles.
c. Peripheral defocus contact lenses.
d. Tropicamide.

2. Which is the primary accepted theory as to how optical treatments (i.e., contact lenses) slow myopia progression?
a. Peripheral myopic defocus.
b. Peripheral hyperopic defocus.
c. Decreased HOAs.
d. Peripheral retinal contrast.

3. Which of the following statements is true regarding HOAs?
a. Greater HOAs improve retinal image quality.
b. Current myopia management treatments induce fewer HOAs.
c. Trefoil is the most important term for myopia management.
d. Increased HOAs may slow axial elongation.

4. Which of the following is the most common form of myopia?
a. Pathological myopia.
b. Late-onset myopia.
c. Genetic myopia.
d. School-age myopia.

5. Most forms of myopia occur due to which of the following?
a. Axial length is too long.
b. Corneal power is too high.
c. Lenticular power is too low.
d. All of the above.

6. Myopia with genetic etiology accounts for what percentage of all myopia?
a. 1%.
b. 2%.
c. 8%.
d. 12%.

7. Which piece of evidence is the strongest argument for genetics causing myopia?
a. Parental myopia increases the risk by three- to six-times.
b. There is a high correlation of refractive error among cousins and siblings.
c. Monozygotic twins have greater heritability than dizygotic twins.
d. Genome-wide association studies have identified several loci associated with myopia.

8. Which of the following statements is true about genome-wide association studies?
a. They compare genetic information across large groups.
b. They can identify one specific gene of interest.
c. They are only done by commercial companies with funding.
d. They have identified numerous loci that account for more than half of the variation in refractive error.

9. Which piece of evidence is the strongest argument against genetics being the sole cause of myopia?
a. Variation in myopia prevalence among different ethnicities.
b. Rapid increase in myopia prevalence over a short period.
c. No one genetic loci has been identified to be the cause of variation in refractive error.
d. Heritability between parents and children is low.

10. Which of the following statements is true about the emmetropization process?
a. Emmetropization is a chemically mediated process.
b. Emmetropization occurs at the neural level.
c. The failure of emmetropization to occur leads to myopia.
d. Emmetropization is modulated by signals to the retina.

11. Which of the following factors could be a covariate of outdoor time?
a. Intelligence.
b. Physical activity.
c. Socioeconomic status.
d. Smoking.

12. Which of the following statements is true about lived environments?
a. There is a higher prevalence of myopia in rural environments.
b. Urban environments have less green space and higher population densities.
c. Lower socioeconomic status is associated with urban environments.
d. Related factors to lived environments are safety, weather and diet.

13. Which of the following statements is true about education as a cause for myopia?
a. There is a higher prevalence of myopia among children who complete more years of schooling.
b. Those with greater academic achievements tend to be more myopic.
c. Education could be confounded by near work and ethnicity.
d. All of the above.

14. Which component is likely the primary driver for near work contributing to myopia?
a. Longer periods of prolonged work.
b. Lower blinking rate during near work.
c. Underaccommodation with optical treatments.
d. Use of different digital devices.

15. Which recommendation should be made to patients who are not yet myopic?
a. Increase outdoor time to two hours/day.
b. Increase physical activity to one hour/day.
c. Decrease near work to two hours/day.
d. All of the above.

16. Why might higher intensity of outdoor light delay myopia onset?
a. Ambient outdoor light is always greater than 100,000 lux.
b. Dopamine is released and modulates axial elongation.
c. Circadian rhythms are reversed.
d. Lower levels of vitamin D are produced.

17. Which of the following is true about low-intensity red light therapy?
a. Red light prevents myopia development in animals and humans.
b. Unlike atropine, there is no rebound effect with red light therapy.
c. Longitudinal chromatic aberrations can disrupt the emmetropization process.
d. There is a systemic crossover effect of red light therapy where treatment in one eye has a residual effect in the non-treated eye.

18. Which recommendation would be most effective for a myopic patient?
a. Increase outdoor time to two hours/day.
b. Increase physical activity to two hours/day.
c. Increase breaks in near work.
d. Wear sunglasses with UV protection.

19. Which of the following patients has the highest risk of developing myopia?
a. A child who has one myopic parent.
b. A child who spends <30 minutes/day outdoors.
c. A child who lives in a rural setting.
d. A child whose academic performance is below average.

20. Which of the following statements about outdoor time is true?
a. Current data on outdoor time are confounded by recall bias.
b. New wearable device technology can objectively measure outdoor time and light exposure.
c. Increased outdoor time can delay myopia onset and but may have no effect on myopia progression.
d. All of the above.

Examination Answer Sheet

Mechanisms of Myopia: What We Know & What We Wonder

Valid for credit through January 15, 2026

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

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Answers to CE exam:

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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. Recognize the pathophysiology and mechanisms of myopia. ① ② ③ ④ ⑤

22. Explain the role genetics plays in the development and progression of this condition. ① ② ③ ④ ⑤

23. Identify the environmental factors that contribute to myopia. ① ② ③ ④ ⑤

24. Educate patients and their parents on what they can do. ① ② ③ ④ ⑤

25. Based upon your participation in this activity, do you intend to change your practice behavior? (Choose only one of the following options.)

Ⓐ I do plan to implement changes in my practice based on the information presented.

Ⓑ My current practice has been reinforced by the information presented.

Ⓒ I need more information before I will change my practice.

26. Thinking about how your participation in this activity will influence your patient care, how many of your patients are likely to benefit? (please use a number): ☐

27. If you plan to change your practice behavior, what type of changes do you plan to implement? (Check all that apply.)

Ⓐ Apply latest guidelines
Ⓑ Change in diagnostic methods
Ⓒ Choice of management approach
Ⓓ Change in current practice for referral
Ⓔ Change in vision correction offerings
Ⓕ Change in differential diagnosis
Ⓖ More active monitoring and counseling
Ⓗ Other, please specify: _____

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

Ⓐ Very confident Ⓑ Somewhat confident Ⓒ Unsure Ⓓ Not confident

29. Which of the following do you anticipate will be the primary barrier to implementing these changes?

Ⓐ Formulary restrictions
Ⓑ Time constraints
Ⓒ System constraints
Ⓓ Insurance/financial issues
Ⓔ Lack of interprofessional team support
Ⓕ Treatment related adverse events
Ⓖ Patient adherence/compliance
Ⓗ Other, please specify: _____

30. Additional comments on this course: _____

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Rate the quality of the material provided:

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

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

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

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

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

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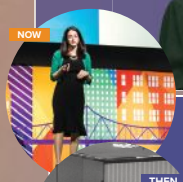

KEYNOTE SESSION

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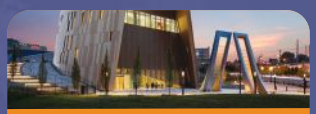
902 3.5 Things You Can Start Doing Today to Keep Burnout Away...
Dr. Ted McElroy
3/2 7:30 PM - 9:30 PM



CIVIL RIGHTS MUSEUM TOUR AND CE
901 Cool Corneal Dystrophies
Dr. Ashley Wallace - Tucker
3/4 3:30 PM - 6:00 PM

SPECIAL SESSIONS

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63 Neuro Nuggets!
Andrew Lee, MD
3/3 4:30 PM - 6:30 PM

64 Retina Revealed!
Jayanth Sridhar, MD; Ajay Kuriyan, MD
3/4 8:00 AM - 10:00 AM

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The Cheesier, the Better

Chalazion incision and curettage is effective and safe for patients. Here's how to get the best results.

BY NATE LIGHTHIZER, OD, AND KOMAL PATEL, OD
TAHLEQUAH, OK

A chalazion is a benign, rigid, non-painful, non-infectious, granulomatous lesion caused by obstruction of a meibomian gland or gland of Zeis. These lipogranulomatous inflammatory lesions are filled with lipid deposits made up of epithelioid cells, multinucleated giant cells and lymphocytes. A pseudocapsule made of connective tissue usually forms around the lesion. They present as round, enlarging nodules on the upper or lower tarsal plate (*Figure 1*).

Chalazia typically occur after internal or external hordeola, which are infections of the meibomian gland and gland of Zeis, respectively. There is no age or sex predilection, although they commonly occur in patients with chronic blepharitis and ocular rosacea. Clinical signs seen in association include eyelid margin telangiectasia, erythema and eyelash debris. A recurrent chalazion should be biopsied to rule out malignancies such as sebaceous gland carcinoma.

Treatment options for chalazia range from conservative to more aggressive approaches. These include warm compresses with or without oral antibiotics, intense pulsed light therapy, steroid injection, and incision and curettage.

When more conservative therapies or steroid injection fail to resolve a chalazion, surgical intervention with incision and curettage can be considered. This is a procedure that drains the contents of the chalazion and more likely avoids recurrence, as the entire lesion and capsule are removed. Cosmesis, discomfort and ptosis are all possible reasons why patients opt to have a chalazion removed. Practitioners may consider incision and curettage as initial therapy for larger chalazia, or after ineffectiveness of all other therapies. Patient preference may also dictate performing an incision and curettage earlier in the treatment paradigm.

Procedural Technique

Let's review how to perform an incision and curettage.

1. Place the patient in a supine position and set up the surgical microscope or loupes.
2. Instill topical anesthetic (proparacaine, tetracaine) into both eyes to minimize reflex lacrimation and the blink reflex, as well as keep the patient comfortable during the procedure.

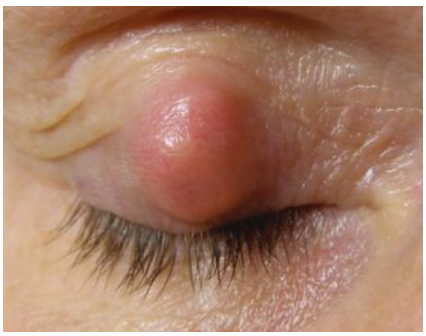

Fig. 1. Large chalazion.

3. Clean around the lesion and skin—where the anesthetic will be injected—with an alcohol prep pad.
4. Use a 30-gauge needle with bevel up to inject anesthesia (0.5%, 1% or 2% lidocaine with 1:100,000 or 1:200,000 epinephrine) subcutaneously at a 10° to 15° angle around the chalazion. A bolus should be seen where the anesthesia was injected. Continue to inject as the needle is withdrawn. If additional anesthesia is needed, the patient will feel less pain if the needle is inserted in an area that was already anesthetized.
5. Massage the anesthesia with a gauze pad or cotton-tipped applicator.
6. Use a 10% povidone-iodine (betadine) swab stick to clean the lesion surface, ocular adnexal area and eyelashes to maintain asepsis. Povidone-iodine should remain on the skin for one to three minutes.
7. Use a piece of gauze to remove any excess povidone-iodine if needed, with caution not to wipe uncleaned skin onto clean skin.
8. Apply two to three drops of ophthalmic 5% povidone-iodine into the eye being treated. Let sit for 30 to 60 seconds before rinsing the eye with sterile saline.
9. Use forceps to test the area to ensure it is anesthetized. If not, inject more anesthesia.
10. Use a correctly sized chalazion clamp and insert around the lesion, with the open side toward the inner eyelid. The clamp should be the smallest clamp possible while still fitting around the chalazion.
11. Slightly tighten the clamp, evert the eyelid and fasten the clamp tightly.

About Dr. Lighthizer

Dr. Lighthizer is the associate dean, director of continuing education and chief of specialty care clinics at the NSU Oklahoma College of Optometry. He is a founding member, and currently serves as president, of the Intrepid Eye Society. Dr. Lighthizer's full disclosure list can be found in the online version of this article at www.reviewofoptometry.com.

"I was only seeing light flashes early on, but light FLASHES when you've not seen anything for so many years—it was wonderful"

—Keith H, retinal prosthesis recipient

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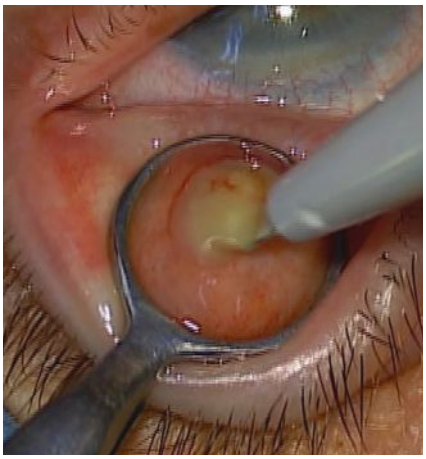

Fig. 2. Scalpel blade making a vertical incision (parallel to the meibomian glands) through the palpebral conjunctiva and into the meibomian glands.

12. Use forceps to test the exposed side of the chalazion (palpebral conjunctiva) to ensure it is anesthetized.
13. If inadequate anesthesia is present on the palpebral conjunctiva, which commonly occurs, apply one to two Weck-Cel sponges soaked in 4% topical lidocaine directly on the chalazion. Hold each sponge on the lesion for approximately 30 to 60 seconds.
14. Use the forceps to test the chalazion to ensure it is anesthetized. Ask the patient if they feel any pain or pinching. Pressure sensation is expected. Always ensure the lesion is fully anesthetized before performing the procedure.
15. Using a blade scalpel, make a vertical incision into the chalazion. The incision should not be closer than 2mm to 3mm from the eyelid margin.
16. Upon opening of the capsule, the chalazion contents may begin to express out of the lesion. (*Figure 2*). Use a cotton-tipped applicator to remove this content.
17. Use a curette to remove the remainder of chalazion contents. A cotton-tipped applicator can also be used to apply lateral pressure to aid in this removal.
18. Once the chalazion is drained, use the curette to traumatize the capsule. This helps avoid recurrence.
19. A horizontal incision parallel to the eyelid margin to make a cross-shaped or "x-shaped" incision may also be needed depending on the size and chronicity of the chalazion and surgeon preference. If an "x-shaped" incision is performed, often times then a Westcott scissors will be used to excise fibrotic tarsal plate in the four "flaps" that are created from the two incisions.
20. An optional injection of 0.1mL to 0.2mL of steroid (triamcinolone acetonide suspension) may be given to aid resolution.
21. Remove the chalazion clamp. No sutures or patches are typically needed. There may be bleeding at the conclusion of the procedure once the clamp is removed as it was aiding in hemostasis. Rinse the eye with sterile saline and hold direct pressure with gauze to the closed eye to provide hemostasis. This may take five to 10 minutes.
22. Apply ophthalmic tobramycin 0.3%/dexamethasone 0.1% ointment to the eye.
23. Measure blood pressure and pulse.

Follow-up

An operative report should be completed after every procedure that describes the steps taken to perform the procedure. Blood pressure and pulse is measured at the conclusion of the procedure due to the injection of epinephrine. An antibiotic/steroid combination ointment or antibiotic ointment is used three times a day for seven to 14 days to prevent infection and control inflammation. A one- to four-week follow-up appointment is typically scheduled to assess healing and chalazion resolution. All patients should be educated to return to the clinic sooner if signs and symptoms of infection arise.

Postoperative complications can occur, but fortunately, are fairly rare. Redness and mild pain are temporary during the post-op phase and will self-resolve once the area has healed. Bruising may occur from injection of anesthesia and typically resolves within two to three weeks. Eyelid notching results from excessive excision of the tarsus close to the eyelid margin, which again should be performed 2mm to 3mm away from the eyelid margin. A rare postoperative infection can be treated with oral antibiotics. Recurrence or a non-resolving chalazion may be re-treated with steroid injection or incision and curettage; however, take caution if malignancy, such as a sebaceous gland carcinoma, is suspected.

Any recurrent chalazion in the exact same spot needs to be considered for possible sebaceous gland carcinoma, in which case, consider a biopsy. In rare cases, intravascular injections may lead to retrograde infiltration that could lead to a central retinal artery

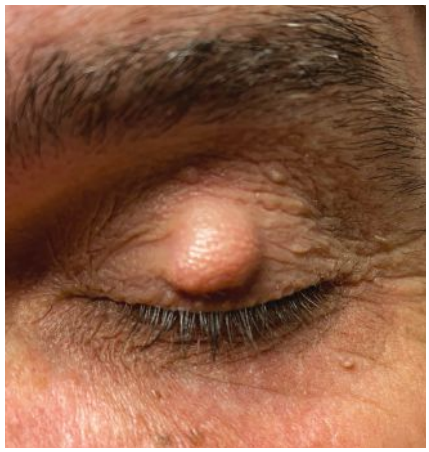

Fig. 3a. Pre-op appearance of a patient prior to a chalazion incision and currettage.

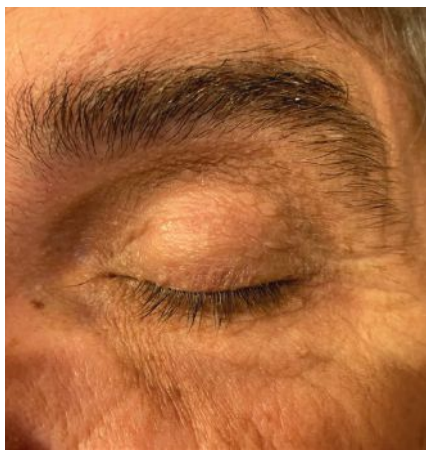

Fig. 3b. One month post-op appearance of the same patient showing complete resolution of the chalazion.

THE NEXT FRONTIER

Welcome to *Review of Optometry*'s newest column, "Advanced Procedures." This will be a bimonthly column intended for the optometric physician as a thorough review of an in-office procedure from as simple as punctal plug insertion to as complex as ptosis repair and everything in between. Indications, contraindications, preoperative considerations, postoperative management and potential risks and complications will be discussed for each procedure, with a special emphasis on procedural techniques.

It is an exciting time in optometry and eye care. Technology continues to emerge and advance at every corner and in every space, procedures are becoming more efficient, more precise and more available to patients. We have seen exciting legislative expansion in optometry across the decades that has only increased access to high quality eye care by well-trained providers. Now with 10 states allowing optometrists to perform certain anterior segment laser procedures, and with more than 15 states allowing optometrists to perform certain types of injections, it is become increasingly evident that optometry will be at the forefront of office-based procedures over the next few decades.

or vein occlusion. Two case reports have outlined individual patients who had a retinal and choroidal vascular occlusion after periocular injection of corticosteroid.[1,2] This can be avoided by injecting with low pressure to minimize retrograde arterial flow.

Takeaways

Chalazion incision and curettage is a very rewarding procedure to perform, as patients do quite well with the procedure and usually have significant preoperative to postoperative improvement in the appearance of their eyelid (*Figures 3a and 3b*). A word of advice for the optometric physician that is just starting out doing in-office procedures: get a number of other procedures under your belt first (*e.g.*, skin tag removals, seborrheic keratosis removals, sebaceous cyst removals, steroid injections for chalazion) to gain confidence in performing procedures on easier removals prior to starting chalazion incision and curettage.

Chalazia are fairly common eyelid lesions that most optometric physicians manage on a regular basis. A number of treatment options are available, with incision and curettage being a nice in-office surgical treatment option that typically delivers optimal results with high patient satisfaction. ■

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ABOUT THE CO-AUTHOR

Dr. Patel is an assistant professor at the Oklahoma College of Optometry (NSUOCO). Dr. Patel is a diplomate of the American Board of Optometry and a fellow in the American Academy of Optometry.

BY ALISON BOZUNG, OD

A Stroke of Luck

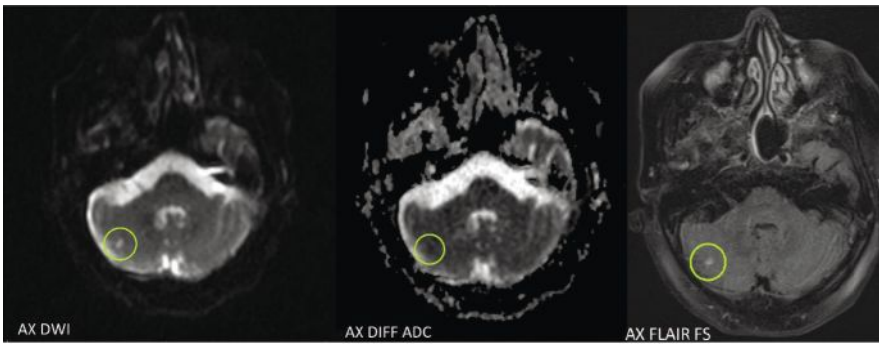
This elderly woman was fortunate to have family members by her side when ocular symptoms developed.

An 82-year-old Hispanic female presented for an evaluation of new visual symptoms. She reported that the day prior, she had seen colorful horizontal lines in the left eye for several hours. The visual phenomenon eventually subsided, and her vision returned to baseline. She came to the emergency department with her son-in-law, who helped provide additional history. He attested that during his mother-in-law's visual episode, he had noticed her left eye was temporarily "stuck" and not moving in conjunction with her right eye. He had asked her to try and track his finger, and although the right eye's motilities were full, the left eye was unable to follow the target properly. He could not recall if the eyelid was ptotic at the time. Interestingly enough, the patient denied appreciating any diplopia. The episode of left eye immobility reportedly lasted about an hour.

Upon further questioning, the patient denied any recent headaches, weight or appetite loss, fevers or chills. She was unable to give a clear answer when asked about jaw pain with chewing (jaw claudication).

The patient had a past ophthalmic history of cataracts and glaucoma, managed with nightly latanoprost in both eyes. Her past medical history was significant for coronary artery disease, diabetes mellitus, hypertension and renal failure.

Her entering visual acuities were 20/350 OD and 20/50 OS. She had a dense cataract in the right eye, greater than the left eye, which we felt explained the reduction in visual acuity. Her intraocular pressures were 14mm Hg OD and 12mm Hg OS. Her pupils were round and reactive to light without an afferent pupillary defect, and confrontation visual fields were full to finger counting. Extraocular movements and color vision were full bilaterally. Slit lamp examination revealed mild blepharitis and the aforementioned cataracts. She had moderate optic nerve cupping consistent with glaucoma, but there was no appreciable optic nerve pallor or edema. The remainder of the posterior segment was unremarkable.



Axial MRI images show acute lacunar infarct (circled in green) of the right cerebellar hemisphere. Areas that are bright on diffusion weighted images and dark on apparent diffusion coefficient are consistent with acute infarct.[8] A hyperintense area can be seen on fluid attenuated inversion recovery imaging due to the presence of vasogenic edema within hours after an acute stroke.[9]

Take it With a Grain of Salt?

As optometrists, we have a unique ability to visualize most pathology in its natural state. From macular degeneration or retinal detachments to cataracts and corneal ulcers, most of what we need to see lies right in front of our eyes. It is one of the features that likely draws many of us into this field in the first place. But in a case such as this one, in which the patient's symptoms have resolved by the time they present to us, the clinician is faced with a difficult task. We are forced to rely solely on the patient's narrative, which can prove challenging when we are not accustomed to doing so.

In this case, the patient provided as great of detail as she was able, but her story was tough to follow. For example, she initially said the colored lines she saw were in the left eye. Later, she reported that she had seen them in both eyes. As we all know, the laterality of such visual distortions can make a significant impact on our list of differential diagnoses. Additionally, though the patient had denied double vision, her son-in-law had been with her at the time and was able to provide a recount of her abnormal ophthalmic motilities.

So, now for our decision: do we tell the patient that everything looks good on our end, or do we pursue a further explanation for the transient, and seemingly disparate, symptoms?

About Dr. Bozung **Dr. Bozung** works in the Ophthalmic Emergency Department of the Bascom Palmer Eye Institute (BPEI) in Miami and serves as the clinical site director of the Optometric Student Externship Program as well as the associate director of the Optometric Residence Program at BPEI. She has no financial interests to disclose.

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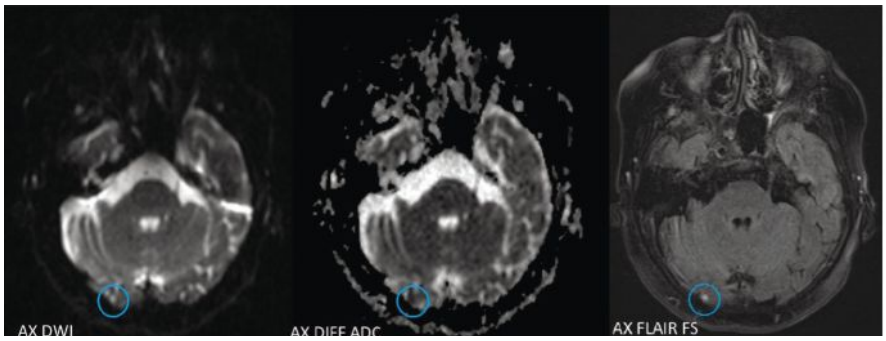


Axial MRI images show acute lacunar infarct (circled in blue) of the inferior medial aspect of the lingual gyrus of the right occipital lobe.

During the examination, a very intentional discussion regarding her reported symptoms occurred. Despite our relatively proficient clinical Spanish, a translator was used to clearly communicate all pertinent details of the story. Ultimately, we felt her symptoms were repeatable enough to likely be reliable and true.

Okay, You Convinced Me

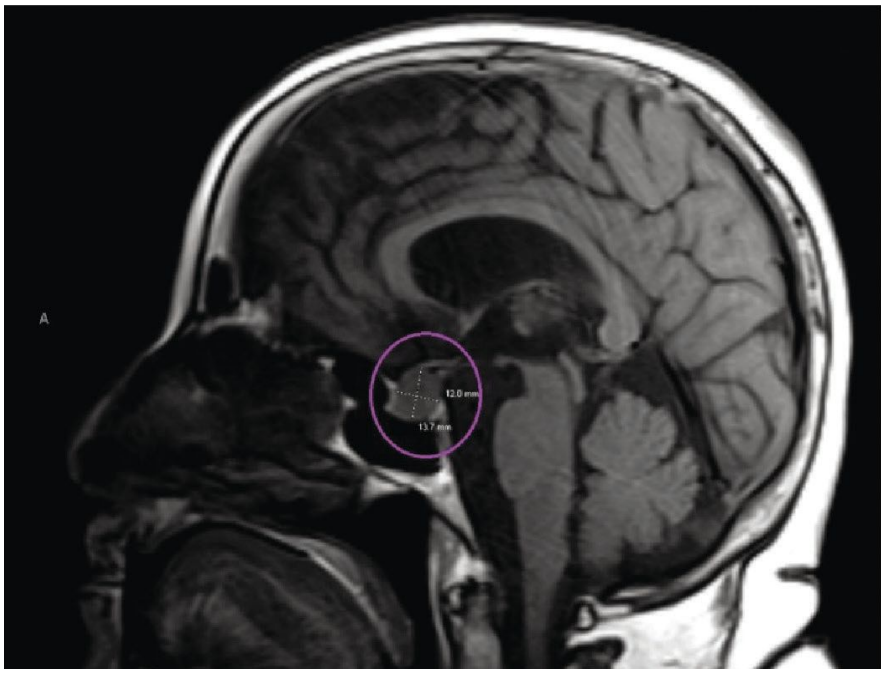
Given our concern, emergent laboratory studies were ordered. The erythrocyte sedimentation rate, C-reactive protein and complete blood count with differential (including platelets) returned grossly unremarkable, essentially eliminating giant cell arteritis. MRI of the brain and orbit without contrast was also obtained. Gadolinium contrast was not administered due to the patient's known history of renal failure and the risk of acute kidney injury or toxicity. Neuroimaging revealed a solid-appearing sellar/suprasellar mass, likely a pituitary macroadenoma, abutting the prechiasmatic segments of both optic nerves. Additionally, acute lacunar infarcts were visualized in both the lingual gyrus of the right occipital lobe and the inferior right cerebellar hemisphere.

These findings were consistent with an acute stroke, and the patient was transferred to our affiliated hospital for a full stroke workup and neurology consultation. She was diagnosed with a stroke due to embolism of the right cerebellar artery. Although a definitive source of emboli was not detected, she was started on low-dose daily aspirin, and a heart monitor patch was placed for continued observation.

Doubling Down

Our patient was found to have multiple pathologies on neuroimaging that could affect the visual system. First, she had a pituitary macroadenoma. Though many pituitary adenomas are asymptomatic and found incidentally, pituitary macroadenomas (adenomas measuring >10mm) may cause loss of vision due to their size and proximity to the optic chiasm/nerves.[1] They can cause monocular or binocular vision changes, but the classic presentation is a bitemporal visual field defect due to chiasmal compression.[2] Headaches in the periorbital region are common, and patients may experience additional symptoms such as amenorrhea, loss of libido, galactorrhea and infertility due to excessive hormonal release in functional adenomas.[3] Patients may also develop diplopia due to compression of one (or more) cranial nerves controlling ocular motilities, but motility deficits from compression would most likely persist rather than be transient as in our patient.[4,5]

Our patient also had evidence of acute stroke affecting the cerebellar and occipital regions. Stroke symptoms are inherently based on their extent and location. Cerebellar strokes often present with nausea, dizziness, gait ataxia, loss of balance and vomiting.[6] She denied any of these symptoms, but the cerebellum is also involved in oculomotor control, and therefore, patients with cerebellar lesions may present with a skew deviation (causing vertical diplopia), disconjugate pursuits or nystagmus.[7] Although our patient presented to the emergency department with full ocular motilities, remember her son-in-law's description of the eyes not tracking a target together. It is impossible to



Sagittal MRI reveals a pituitary lesion, most likely a macroadenoma (circled in purple). It was described to be abutting both optic nerves anterior to the chiasm.

know what her motilities looked like at the time of the episode, but it was a very intriguing description.

Finally, strokes involving the visual cortex within the occipital lobe are well known to cause contralateral visual phenomena or vision loss. One study of patients with acute stroke revealed self-limiting visual hallucinations may be relatively frequent in those with occipital lesions. Recall that our patient experienced temporary visual disturbances on the left side. If her vision changes were, in fact, caused by the ischemic lesion in the right occipital lobe, we could surmise that they were likely affecting the left hemisphere bilaterally.

Takeaways

Regardless of the exact etiology behind our patient's fleeting visual system symptoms, one important point remains: it is of the utmost importance to truly listen to our patients and do our best to understand what they are telling us. It is easy to "write off" symptoms at times, especially when they are transient or not visible to us. However, we must remain vigilant to spot warning signs of disease so we can continue to positively impact the health and well-being of our patients. ■

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A Reason to SMILE?

The presence of dry eye complicates a patient's prospects for refractive surgery. Which procedure is the better option?

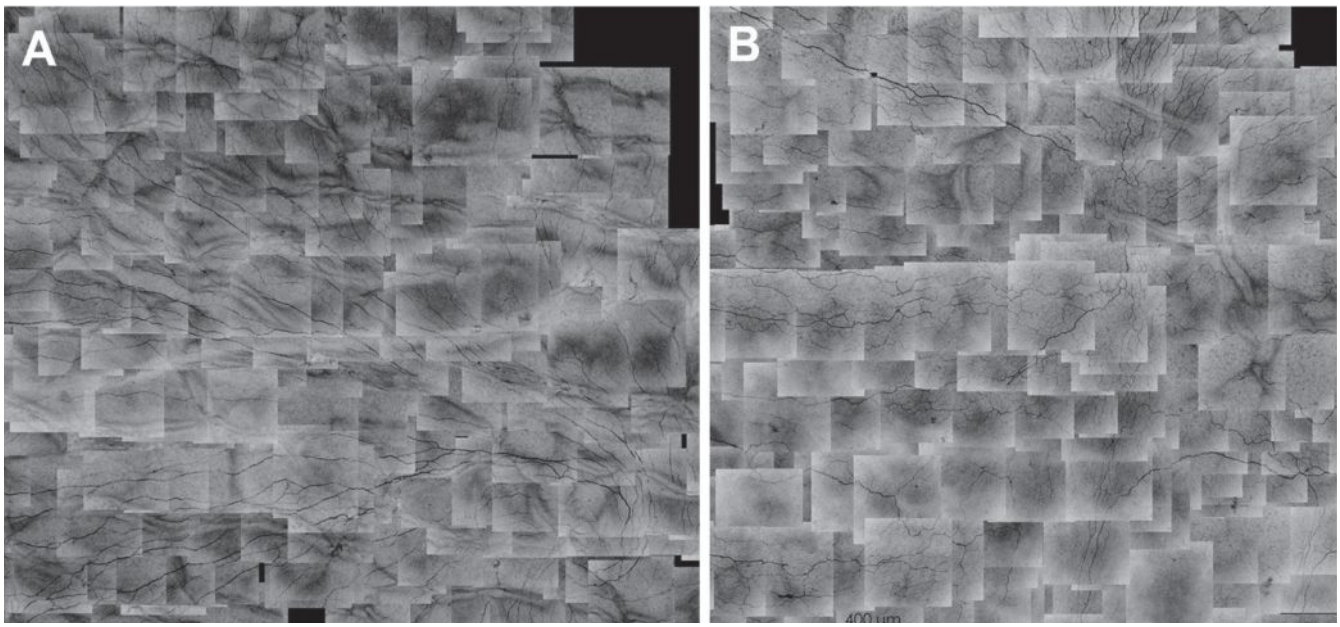
Q I have a patient interested in refractive surgery but they have borderline dry eye (decreased tear breakup time and some symptomatic complaints but no evidence of staining). What's the best course of action? Would this be a patient more suited for SMILE rather than LASIK? Either way, what are some best practices for follow-up care?

A "As a first step, it is highly recommended that dry eye should be treated using standard treatment, such as artificial tears and/or anti-inflammatory agents before any refractive surgery," says Cecilia Chao, BOptom, PhD, of UNSW Sydney's School of Optometry and Vision Science. She explains that improvement in dry eye signs and symptoms, such as dryness, grittiness or tearing, as well as their severity, should be recorded. This can be done with a validated questionnaire, like the Ocular Surface Disease Index, Ocular Pain Assessment Survey or Ocular Comfort Index. In addition, for mild dry eye, carefully review the patient's systemic health, medication use, previous eye and other surgeries and even mental health, as all these components are needed to clear the potential risk factors that may be associated with development of post-LASIK neuropathic dry eye.

"If dry eye symptoms cannot be relieved with typical treatment and the patient may have normal tear function with minimal to no corneal/conjunctival staining, mild neuropathic pain should be considered. The persistent symptoms can be confirmed using the validated questionnaires," Dr. Chao continues. If *in vivo* confocal microscopy (IVCM) is accessible in your clinic, it can further confirm the health of the corneal nerves and immune response through examination of corneal nerve integrity, the presence of corneal immune cells and morphology. It is the surgeon's or optometrist's responsibility to explain to patients the potential risk or further damage that can occur to the corneal nerves after surgery, as well as outline the potential to suffer from greater and persistent ocular neuropathic pain, she adds.

Surgery Effects

Regarding whether SMILE is superior to femtosecond-LASIK (FS-LASIK), there is still limited evidence. It's been shown that there are fewer short-term incidents of postoperative dry eye after



Corneal subbasal nerve map of emmetropic right eye of a normal subject (A) and right eye of another subject seven years after LASIK (B). Shown at 400mm of the scale bar.

About Dr. Shovlin

Dr. Shovlin, a senior optometrist at Northeastern Eye Institute in Scranton, PA, is a fellow and past president of the American Academy of Optometry and a clinical editor of *Review of Optometry* and *Review of Cornea & Contact Lenses*. He consults for Kala, Aerie, AbbVie, Novartis, Hubble and Bausch + Lomb and is on the medical advisory panel for Lentechs.

SMILE compared with LASIK, but long-term effect is still undetermined, Dr. Chao says. In addition, SMILE presents a more technical challenge due to the complexity of the procedure. This results in more intraoperative discomfort and greater visual symptoms after surgery when compared with LASIK. Therefore, it is all dependent on the expectation of the patient and the experience of the surgeons.

"Based on my experience, for follow-up after FS-LASIK, we use 0.5% levofloxacin four times a day for three days and 0.1% fluorometholone four times a day for one week," Dr. Chao notes. "The fluorometholone is tapered weekly and stopped by week four post-op."

It is vital not to drop the fluorometholone from four times a day to none for any type of refractive surgery, she warns. In addition, all refractive surgery patients should be instructed to use nonpreservative artificial tears at least four times a day and for at least six months, since corneal sensitivity will not return to normal anywhere from six to 12 months. Apart from a slit-lamp test, it is highly recommended to do sensitivity and IVCM testing every visit, along with monitoring the symptoms using a validated questionnaire, starting from one month after operation, she adds.

Dr. Chao highlights that usually, if patients suffer from neuropathic dry eye more than one year after the surgery, there may be minimal slit lamp and abnormal tear film function findings for an optometrist to observe. To combat this issue, hyperosmolar saline may be used as a diagnostic test before the surgery. "A more recent study in FS-LASIK outlines corneal dendritic cells (immune cells) and corneal nerves' interaction," Dr. Chao notes. "If IVCM is accessible, I would suggest looking at the cell morphology, since it is related to the ocular immune response." She also explains that there may be more migratory cells after LASIK, and this may be associated with post-LASIK neurogenic inflammation," explains Dr. Chao. In the absence of IVCM, the patient can be referred to an institute where IVCM can be conducted.

Takeaways

Remember that patients should always try to be treated for dry eye before any refractive surgery, regardless of what type will be used. Risk of developing post-op dry eye with neuropathic factors can be aided by IVCM. "While SMILE is an advanced refractive surgical technique, limited evidence indicates SMILE is superior to FS-LASIK in safety and performance. Therefore, patients should consult with their surgeon and optometrist about what the best option is," Dr. Chao concludes. ■

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PRODUCT REVIEW

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▸ EYE DROPS

First PF Latanoprost Eye Drop Hits the Market

Preservative-free eye drops are increasingly being preferred by patients and clinicians due to their low side effect profile and minimized impact on the ocular surface. Now, glaucoma patients on drop therapy can also reap the benefits of a preservative-free formula following the recent FDA approval of a new 0.005% latanoprost solution by Thea Pharma. The drop, called Iyuzeh, is made without any of the common ophthalmic solution preservatives, including benzalkonium chloride. It was approved to reduce elevated IOP in patients with open-angle glaucoma or ocular hypertension.

The company noted in its press release that Iyuzeh showed consistent IOP-lowering ability and good patient tolerability in clinical trials. In patients with open-angle glaucoma or ocular hypertension, treatment with Iyuzeh vs. a BAK-preserved formula (Xalatan) resulted in comparable IOP reduction (3mm Hg to 8mm Hg vs. 4mm Hg to 8mm Hg). The most common side effects observed in the two company-led clinical trials were conjunctival hyperemia (34%) and eye irritation (19%). For comparison, among patients who took Xalatan, 37% experienced conjunctival hyperemia and 31% experienced ocular irritation.

The drop is recommended by developers to be administered once daily in the evening. IOP reduction occurs approximately three to four hours after drop administration, and the maximum effect is reached after eight to 12 hours, Thea explains. It adds that the IOP-lowering effect persists for a minimum of 24 hours.

Biotrue Hydration Boost Drops for Soft, GP Lenses

Eye dryness is a frequent side effect of contact lens wear. To help alleviate discomfort and increase ocular surface hydration in these patients, clinicians often prescribe a preservative-free eye drop that's safe to use with contact lenses. Joining this market is a multi-dose drop by Bausch + Lomb for soft and rigid GP lenses, called Biotrue Hydration Boost Contact Lens Rehydrating Drops.

Like other products in B+L's Biotrue line, the new hydration boost drops have no preservatives and contain only natural ingredients informed by the Tear Film and Ocular Surface Society's DEWS II report, the company says. These include glycerin (the active ingredient), hyaluronan (a moisturizer naturally found in the eye), an electrolyte and an antioxidant. B+L also notes in its press release that the pH of the solution matches that of healthy tears to optimize lubrication and comfort. The drops are intended to keep eyes moisturized for eight hours.

▸ PRACTICE EQUIPMENT

Light it Up: Two-in-one Desk Lamp and Flashlight

Adequate lighting throughout your practice is imperative to ensure the accuracy of clinical tasks, from performing eye exams to reviewing countless patient files. In situations or spaces where you or your staff could benefit from more illumination, a portable light source—such as Eschenbach's new two-in-one desk lamp and flashlight—can be a handy solution. This dual-purpose tool—called the Magno travel lamp—features three color temperatures and can be dimmed from 100% to 10% light with the click of a button, the company says.

To use as a desk lamp, the flashlight head can be set on a table face-down—surrounded by a rubber grip for extra stability—while the lamp head is folded out from the handle. The lamp features three light settings clinicians can choose from: a warm yellow 3200K, a neutral white 4200K and a cool white 6000K. Eschenbach also notes in the press release that the color rendition index is greater than 90.

When using the device as a flashlight, simply fold the lamp head back into the handle. Unlike the lamp head, the flashlight head features only one lighting option. The rechargeable battery lasts for eight hours when the light is used as a desk lamp and five hours when used as a flashlight, according to the company.

A convenient perk of the Magno, Eschenbach says, is that it can easily be moved from room to room or taken on the road, weighing in at 13 ounces and measuring 10.5 inches in length (or 18 inches when the lamp head is extended). The lamp comes with a USB cable for charging and features a red light to indicate when the battery is below full capacity. ■

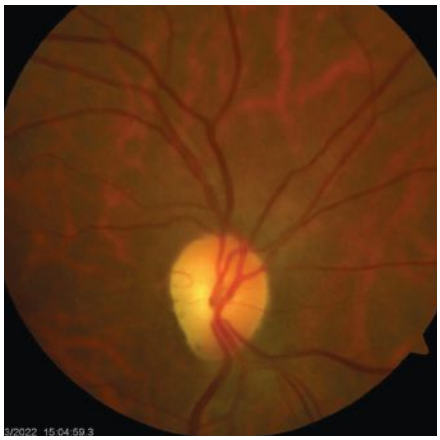
Practice For Sale

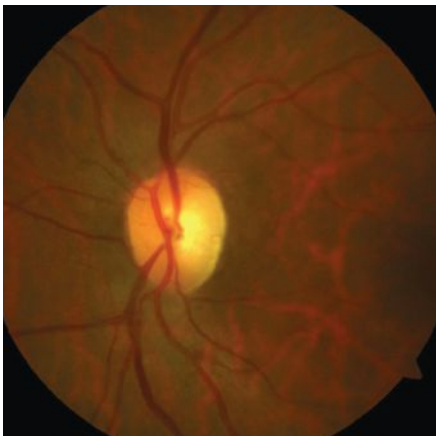
BY ANDREW S. GURWOOD, OD

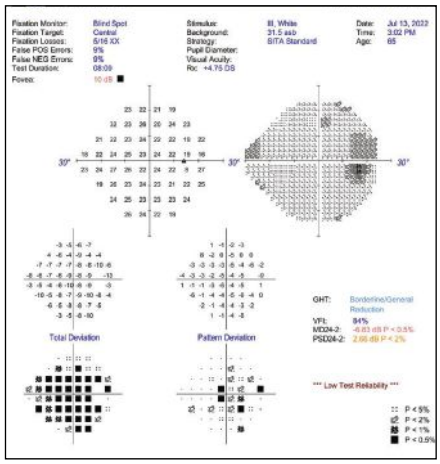
Disc Jockey

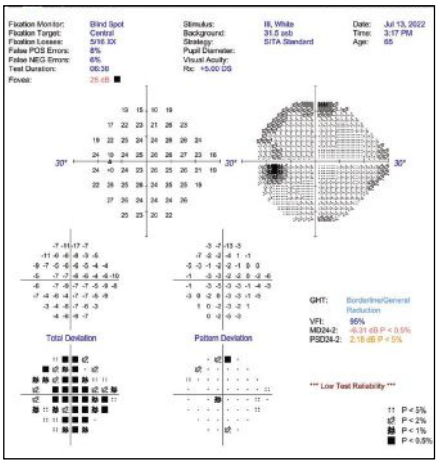
This patient's optic nerve head appearance was concerning. What could create this presentation, and what does it represent?

A 65-year-old Black female presented for a routine eye examination with a chief complaint of blurred vision, worse in the right eye. She noticed her vision had been declining for at least the past eight months. Her ocular history was remarkable for cataracts, OU. Her systemic history found no hypertension, diabetes or other illness. She denied any past ocular trauma and allergies to medications or other things.









Clinical Findings

Her best uncorrected entering visual acuities were 20/30 OD and 20/25 OS at distance and near with no improvement upon pinhole or refraction. Her external examination was normal and there was no afferent pupillary defect (APD).

Biomicroscopy uncovered normal tissues and structures OU with Grade II nuclear sclerotic cataracts. Her intraocular pressures measured 16mm Hg by Goldmann applanation tonometry. The pertinent posterior segment findings are demonstrated in the photographs.

Additional testing included color photodocumentation, OCT of both nerves, automated perimetry, red cap color testing, brightness testing and visual fields.

Your Diagnosis

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

Dr. Gurwood thanks Sam Kim, OD, for contributing this case.

Do you notice any correspondence between the fundus photos and the visual field results? If so, what does it signify?

About Dr. Gurwood Dr. Gurwood is a professor of clinical sciences at The Eye Institute of the Pennsylvania College of Optometry at Salus University. He is a co-chief of Primary Care Suite 3. He is attending medical staff in the department of ophthalmology at Albert Einstein Medical Center, Philadelphia. He has no financial interests to disclose.

NEXT MONTH IN THE MAG

In February, we present our annual issue devoted to diagnostic skills and techniques. Articles will include:

- Tips for Taking a Better Case History
- Controversies and Questions in the Approach to Keratoconus
- 27 Diagnostic Tips and Tricks: Are You Using These Technqiues to Their Fullest?
- Pupil Testing Methods and What They Indicate
- Yes, You Can—and Should—Order Labs and Neuroimaging, *Part 1 of 2*

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CooperVision®

We're willing to bet most eye care professionals don't realize just how prevalent *Demodex* blepharitis is.[1]

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References: 1. Data on file, Tarsus Pharmaceuticals, Inc. June 2022. **2.** O'Dell L et al. *Clin Ophthalmol.* 2022;16:2979-2987.

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